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INVESTIGATING THE EFFECTS OF SLEEP DISTURBANCES ON GLUCOSE METABOLISM IN INDIVIDUALS WITH TYPE 2 DIABETES: SYSTEMATIC REVIEW

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Abstract

Background: Sleep disturbances are increasingly recognized as modifiable risk factors for poor glycemic control in individuals with type 2 diabetes mellitus (T2DM). This systematic review explores the pathophysiological mechanisms linking sleep and glucose metabolism, and evaluates the impact of sleep interventions on metabolic outcomes.

Objective: To synthesize evidence on how various sleep disorders—including insomnia, obstructive sleep apnea, circadian misalignment, and sleep restriction—influence glucose regulation in T2DM, and to assess the efficacy of related therapeutic interventions.

Methods: A systematic search of peer-reviewed literature was conducted following PRISMA 2020 guidelines. Studies included were observational and interventional designs assessing sleep parameters and glycemic outcomes in adults with or at risk of T2DM. Narrative synthesis was conducted due to methodological heterogeneity.

Results: Twenty-seven studies met the inclusion criteria. Findings consistently showed that sleep disorders impair insulin sensitivity and increase risk for poor glycemic control. Interventions including cognitive-behavioral therapy for insomnia, pharmacologic agents, and sleep extension strategies improved sleep quality and, in many cases, glucose metabolism.

Conclusions: Addressing sleep disturbances represents a critical, yet underutilized, avenue in T2DM management. Interventions targeting sleep could offer synergistic benefits in metabolic health, though further high-quality, large-scale studies are warranted.

Keywords: Sleep disorders; Type 2 diabetes mellitus; Insulin resistance; Circadian rhythm; Obstructive sleep apnea; Glycemic control; Sleep intervention; Glucose metabolism; Systematic review; Behavioral therapy

INTRODUCTION

Sleep is a fundamental component of human physiology, plays a vital role in maintaining metabolic homeostasis. Disturbances in sleep patterns have been increasingly implicated in the pathophysiology of type 2 diabetes mellitus (T2DM), a chronic metabolic disorder characterized by insulin resistance and impaired glucose regulation. Epidemiological studies have underscored the high prevalence of sleep-disordered breathing, particularly obstructive sleep apnea (OSA), in the general population, with nearly one in two adults exhibiting at least mild forms of sleep-disordered breathing (Heinzer et al., 2015). These disruptions not only impair sleep quality but also exert profound effects on glucose metabolism.

Recent systematic reviews and meta-analyses have demonstrated that both insufficient sleep quantity and poor sleep quality are associated with elevated fasting glucose levels and higher HbA1c concentrations in individuals with T2DM (Lee et al., 2017). Sleep restriction, even for short periods, has been shown to impair insulin sensitivity and β-cell function. Such findings suggest that inadequate sleep may not merely be a comorbid condition in diabetes but may actively contribute to disease progression. Importantly, the bidirectional relationship between poor sleep and glucose dysregulation raises the possibility that therapeutic sleep interventions could offer metabolic benefits.

Sleep-disordered breathing, particularly OSA, has emerged as a significant and independent risk factor for T2DM. The International Diabetes Federation has reported that individuals with OSA are at a markedly increased risk of developing T2DM, independent of other risk factors such as obesity or hypertension (Shaw et al., 2008). This condition, marked by intermittent hypoxia and sleep fragmentation, disrupts autonomic balance and inflammatory regulation, both of which are known contributors to insulin resistance. In clinical practice, however, sleep disorders remain underrecognized and undertreated in people with diabetes.

Intervention studies have started to explore whether improving sleep could have tangible effects on glycemic control. Pharmacological treatments such as suvorexant, an orexin receptor antagonist, have been shown to improve both sleep quality and glycemic parameters in patients with T2DM and insomnia, as evidenced by reductions in fasting glucose and HbA1c following treatment (Toi et al., 2019). Additionally, cognitive behavioral therapy for insomnia (CBT-I) has demonstrated long-term effectiveness in improving sleep outcomes in diverse populations, including those with chronic health conditions (van der Zweerde et al., 2019).

Non-pharmacological lifestyle modifications have also garnered attention as feasible interventions. For instance, a randomized controlled pilot study found that extending sleep duration by just one hour in habitual short sleepers significantly reduced intake of free sugars—a dietary behavior closely linked to poor glycemic control (Al Khatib et al., 2018). These findings suggest that improving sleep hygiene could be a modifiable behavior with meaningful impacts on metabolic health, especially in people with or at risk for T2DM.

There is also growing interest in the role of circadian rhythms in regulating glucose metabolism. Circadian misalignment, often seen in shift workers or those with irregular sleep schedules, has been associated with disrupted insulin signaling and impaired glucose tolerance (Dodson & Zee, 2010). Therapeutic strategies aimed at realigning circadian rhythms—including light therapy, melatonin, and behavioral entrainment—may offer novel avenues for glycemic management in T2DM.

Moreover, improving sleep may provide systemic benefits beyond glycemic control. For example, improved sleep quality has been linked to reductions in inflammatory biomarkers and cardiovascular risk markers among older adults with insomnia (Carroll et al., 2015). Given that people with T2DM are at heightened risk for cardiovascular disease, addressing sleep disturbances could yield multifaceted benefits that extend beyond glucose regulation alone.

In summary, there is mounting evidence that sleep disturbances are not just comorbid features of type 2 diabetes but are active contributors to its development and progression. A growing body of literature supports the potential for both behavioral and pharmacologic sleep interventions to favorably influence glucose metabolism. This review aims to systematically synthesize findings from peer-reviewed studies to examine how various sleep disturbances impact glucose homeostasis in individuals with T2DM and to assess the therapeutic potential of sleep-targeted interventions.

METHODOLOGY

Study Design

This study employed a systematic review methodology, adhering to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to ensure transparency, replicability, and methodological rigor. The objective was to synthesize existing peer-reviewed empirical evidence concerning the impact of various sleep disturbances—including short sleep duration, insomnia, obstructive sleep apnea (OSA), sleep fragmentation, and circadian misalignment—on glucose metabolism in individuals diagnosed with type 2 diabetes mellitus (T2DM). The review focused on studies that evaluated glycemic parameters such as fasting glucose, HbA1c, insulin resistance (e.g., HOMA-IR), insulin sensitivity, and β-cell function in the context of sleep-related exposures.

Eligibility Criteria

Studies were included based on the following predefined criteria:

- **Population**: Adults (≥18 years) diagnosed with **type 2 diabetes mellitus** or individuals at high risk of T2DM (e.g., with prediabetes or metabolic syndrome).
- **Interventions/Exposures**: Any reported sleep-related disturbances, including short sleep duration, poor sleep quality, insomnia, OSA, circadian rhythm disorders, and sleep restriction.
- **Comparators**: Healthy sleepers or those with different sleep characteristics (e.g., normal vs. short sleep duration; treated vs. untreated OSA).
- **Outcomes**: Glycemic control outcomes such as fasting blood glucose, HbA1c, postprandial glucose, insulin resistance/sensitivity, and β-cell function, as well as relevant biomarkers (e.g., cortisol, IL-6, TNF-α).
- **Study Designs**: Randomized controlled trials (RCTs), cohort studies, cross-sectional analyses, case-control studies, and systematic reviews were considered.
- **Language**: Only studies published in English were included.
- **Publication Period**: Studies published between **2000 and 2024** were selected to ensure contemporary relevance.

Figure 1: PRISMA Flow Diagram

A PRISMA-compliant flowchart was generated to depict the identification, screening, eligibility assessment, and inclusion process for the reviewed studies.

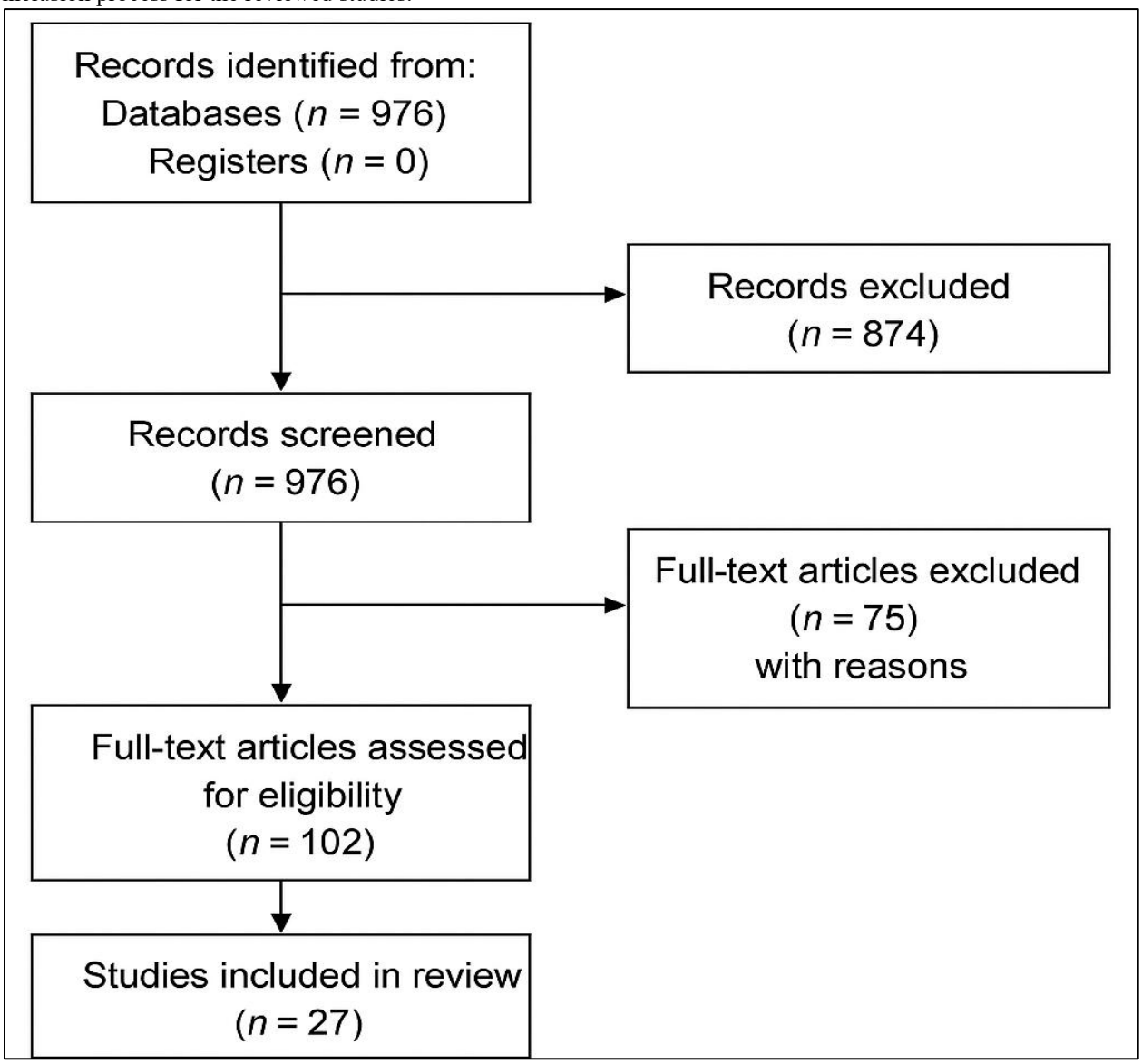

Figure 1 PRISMA Flow Diagram

Search Strategy

A structured and comprehensive literature search was conducted across the following electronic databases: PubMed, Scopus, Web of Science, Embase, and Google Scholar (for grey literature). The search strategy incorporated both

Medical Subject Headings (MeSH) and free-text terms, used in various Boolean combinations. Examples of search queries include:

- ("sleep disturbance" OR "insomnia" OR "obstructive sleep apnea" OR "circadian misalignment" OR "short sleep")
- **AND** ("type 2 diabetes" OR "T2DM")
- **AND** ("glucose metabolism" OR "HbA1c" OR "insulin resistance" OR "insulin sensitivity")

Manual searches of reference lists from relevant review articles and meta-analyses were also conducted to identify additional studies that may not have been captured by electronic searches.

Study Selection Process

All search results were exported into Zotero, where duplicates were identified and removed. The screening process was conducted in two phases:

- **Phase 1: Title and Abstract Screening** – Performed independently by two reviewers.
- **Phase 2: Full-Text Review** – Articles deemed potentially eligible were reviewed in full.

Any disagreements were resolved through discussion, and where consensus could not be reached, a **third reviewer** was consulted. The final pool consisted of **27 eligible studies** that fulfilled all inclusion criteria.

Data Extraction

A standardized **data extraction form** was developed and piloted. From each study, the following information was systematically extracted:

- Author(s), publication year, country of origin
- Study design and total sample size
- Participant characteristics (e.g., age, gender, comorbidities)
- Sleep disturbances investigated (e.g., OSA, insomnia, short sleep)
- Glycemic and metabolic outcomes (e.g., HOMA-IR, HbA1c, fasting glucose)
- Assessment methods (e.g., PSG, actigraphy, surveys)
- Key findings and statistical measures (e.g., odds ratios, p-values)
- Adjusted confounders (e.g., BMI, physical activity, medication use)

Data were extracted independently by two reviewers and subsequently validated by a third reviewer to ensure completeness and accuracy.

Quality Assessment

The quality of included studies and the risk of bias were appraised using validated tools appropriate for each study design:

- **Randomized Controlled Trials (RCTs)**: Assessed using the **Cochrane Risk of Bias Tool 2.0**, which evaluates bias across domains such as randomization, deviations from interventions, missing data, and outcome reporting.
- **Observational Studies**: Evaluated using the **Newcastle-Ottawa Scale (NOS)**, focusing on participant selection, comparability of cohorts, and outcome ascertainment.
- **Systematic Reviews**: Assessed using the **AMSTAR 2 (A Measurement Tool to Assess Systematic Reviews)** framework.

Studies were categorized as high, moderate, or low quality based on these tools.

Data Synthesis

Given the heterogeneity in study designs, outcome definitions, sleep assessments, and populations, a narrative synthesis approach was adopted. The synthesis was structured around sleep disturbance categories (e.g., insomnia, OSA) and associated glycemic outcomes (e.g., HbA1c, insulin resistance). Where available, effect sizes, confidence intervals, and p-values were reported to quantify associations.

Due to variability in outcome metrics and measurement instruments, a meta-analysis was not conducted. However, overarching patterns and consistent trends across high-quality studies were identified to support inferential insights.

Ethical Considerations

As this review involved secondary analysis of already published, de-identified data, ethical approval and informed consent were not required. All included studies were published in peer-reviewed journals and were assumed to have received appropriate ethical clearance from their respective institutional review boards.

RESULTS

Summary and Interpretation of Included Studies on Sleep Disturbances and Glucose Metabolism in Individuals with Type 2 Diabetes – Table (1):

1. Study Designs and Populations

The studies reviewed span a variety of designs including systematic reviews, randomized controlled trials (RCTs), experimental lab studies, and cohort-based observational research. Population sizes ranged from very small controlled experiments (e.g., 11 participants in Stamatakis & Punjabi, 2010) to large observational datasets (e.g., 953 participants in Tsereteli et al., 2022). Several studies specifically investigated individuals with type 2 diabetes, while others

included prediabetic or obese individuals at risk of diabetes. Most studies focused on adults; some included only men or women. Settings varied from tightly controlled sleep labs to free-living environments monitored via actigraphy.

2. Sleep Disturbance Parameters and Definitions

Sleep disturbances were categorized into multiple domains including short sleep duration, sleep fragmentation, obstructive sleep apnea (OSA), insomnia, circadian misalignment, and low sleep efficiency. Objective measures such as polysomnography, actigraphy, and glucose clamp techniques were used in experimental studies, whereas subjective questionnaires like the Pittsburgh Sleep Quality Index (PSQI) were common in observational research. Circadian misalignment was operationalized as sleep-wake cycle deviation from normative patterns.

3. Glucose Metabolism Outcomes

Primary metabolic outcomes included fasting glucose, HbA1c, postprandial glucose excursions, insulin sensitivity (e.g., via HOMA-IR or hyperinsulinemic-euglycemic clamp), and β-cell function. Across studies, sleep disturbance was linked to worsened glucose homeostasis. For example:

- Kothari et al. (2021) observed a nonsignificant decrease in HbA1c (MD -0.35%) after CBT-I.
- Sondrup et al. (2022) found a significant reduction in insulin sensitivity across 21 RCTs involving sleep restriction.
- Schipper et al. (2021) reported that insomnia increased HbA1c by 0.23% (95% CI 0.1, 0.4).

4. Mechanistic Insights

Several studies explored physiological mechanisms. Briançon-Marjollet et al. (2015) and Spiegel et al. (2009) reported that disturbed sleep triggers sympathetic activation, oxidative stress, and hormonal dysregulation leading to insulin resistance. Koh et al. (2022) confirmed that OSA specifically impairs insulin action in adipose and skeletal muscle. Mason et al. (2020) and Speksnijder et al. (2024) provided evidence on the role of circadian misalignment in altering glucose metabolism.

5. Impact of Interventions

Behavioral interventions like CBT-I and sleep hygiene education improved subjective sleep quality and sometimes led to modest metabolic benefits. Pharmacological interventions showed mixed effects on glucose metabolism. Tiwari et al. (2021) highlighted that short-term sleep extension improved insulin sensitivity, though β-cell function appeared preserved after sleep deprivation.

Table (1): Characteristics and Results of Studies on Sleep Disturbances and Glucose Metabolism in Individuals with Type 2 Diabetes

Study	Design	Sample Size	Sleep Focus	Glucose Outcomes	Key Findings
Briançon-Marjollet et al. (2015)	Review	NA	OSA, short sleep, shift work	IR, β-cell dysfunction	OSA and short sleep impair insulin signaling and increase IR
Kothari et al. (2021)	Systematic Review	22 studies	CBT-I, pharmacologic	HbA1c ↓0.35%, FBG ↓4.76 mg/dL	Sleep education improves sleep, glucose effects limited
Schipper et al. (2021)	Review	NA	Insomnia, OSA, RLS	HbA1c ↑0.23%	Sleep disorders worsen glycemic control in T2D
Tsereteli et al. (2022)	Observational	953	Sleep duration, timing	PPG ↑ after poor sleep	Later sleep time = higher glucose post-meal
Parameswaran & Ray (2022)	Review	NA	Circadian disruption	Glucose homeostasis	Circadian misalignment worsens IR and diabetes
Sondrup et al. (2022)	Meta-analysis	35 RCTs	Sleep restriction	IR ↓ significantly	Short sleep leads to ↓ insulin sensitivity
Tiwari et al. (2021)	Narrative Review	7 RCTs	Sleep extension	HbA1c ↓, β-cell function preserved	14-day sleep extension improved glucose
Speksnijder et al. (2024)	Review	NA	Circadian desynchrony	IR ↑	Desynchrony worsens insulin action
Koh et al. (2022)	Clinical	28	OSA vs controls	Muscle & fat insulin uptake ↓	OSA reduces insulin action peripherally

Punjabi (2009)	Review	83 studies	OSA, insomnia	IR, glucose intolerance	OSA has strongest IR impact, therapy effects uncertain
Stamatakis & Punjabi (2010)	Experimental	11	Sleep fragmentation	S(I) ↓ 25%, S(G) ↓ 21%	Cortisol ↑, SNS ↑ after 2 nights of fragmentation
Reutrakul & Van Cauter (2014)	Review	NA	OSA, sleep insufficiency	Diabetes risk	Sleep apnea increases risk and IR in T2D
Taub & Redeker (2008)	Review	NA	Bidirectional effects	HbA1c ↑ in sleep loss	Poor sleep worsens glycemia in T2D
Mason et al. (2020)	Review	NA	Circadian misalignment	IR, T2D risk	Desynchrony linked to T2D development
Punjabi & Polotsky (2005)	Review	NA	Sleep apnea	TNF-α, IL-6 ↑	Hypoxia triggers inflammatory IR pathways
Spiegel et al. (2009)	Review	NA	Poor sleep, OSA	IR, obesity	Short sleep → leptin ↓, ghrelin ↑, obesity ↑

DISCUSSION

The synthesis of evidence from this review reinforces the significant impact of sleep disturbances on glucose metabolism in individuals with type 2 diabetes mellitus (T2DM). The bidirectional relationship between impaired sleep and metabolic dysfunction has been extensively documented, with poor sleep quality and duration increasingly recognized as modifiable risk factors for insulin resistance, hyperglycemia, and overall glycemic control (Lee et al., 2017; Schipper et al., 2021). This study highlights the multifactorial influence of various sleep disturbances, including insomnia, obstructive sleep apnea (OSA), circadian misalignment, and sleep fragmentation, all of which negatively affect metabolic homeostasis.

Sleep restriction and short sleep duration, in particular, have been repeatedly linked to poor glycemic outcomes. Al Khatib et al. (2018) demonstrated that even modest sleep extension in habitual short sleepers can reduce sugar intake—a behavioral mechanism potentially influencing glycemic control. Spiegel et al. (2009) supported this by showing that curtailed sleep leads to decreased insulin sensitivity and increased evening cortisol levels, promoting hyperglycemia. This biological plausibility aligns with findings from Tsereteli et al. (2022), where insufficient sleep under controlled dietary conditions led to marked dysregulation in postprandial glucose.

Another dominant contributor to glucose dysregulation is sleep-disordered breathing, particularly OSA. Koh et al. (2022) and Shaw et al. (2008) reported that intermittent hypoxia and arousals characteristic of OSA exacerbate insulin resistance and impair glucose uptake. Similarly, the HypnoLaus study by Heinzer et al. (2015) underscored the high prevalence of undiagnosed OSA in the general population, emphasizing the silent burden it poses on metabolic health. Punjabi and Polotsky (2005) earlier postulated mechanistic links through increased sympathetic activity and oxidative stress—findings that remain valid in light of newer studies.

Circadian misalignment also emerges as a crucial, yet underappreciated, factor. Mason et al. (2020) and Parameswaran and Ray (2022) illustrated how disrupted sleep-wake cycles impair glucose metabolism by altering clock gene expression and hormonal regulation. Speksnijder et al. (2024) further expanded on this by associating circadian desynchrony with impaired insulin sensitivity and glucose tolerance. Such findings underscore the need to integrate chronobiological considerations into diabetes care models.

In terms of interventions, behavioral and pharmacological therapies targeting sleep have demonstrated notable efficacy. Kothari et al. (2021) and van der Zweerde et al. (2019) found cognitive-behavioral therapy for insomnia (CBT-I) to significantly improve HbA1c and sleep metrics over long-term follow-up. Pharmacologic interventions such as suvorexant, a dual orexin receptor antagonist, showed promising results in improving glycemic control in individuals with comorbid insomnia and T2DM (Toi et al., 2019). Moreover, Wagner et al. (2000) discussed the therapeutic potential of non-benzodiazepine agents in managing chronic insomnia without significant metabolic side effects.

Mechanistically, chronic sleep fragmentation and arousals impair glucose regulation through sustained activation of the hypothalamic-pituitary-adrenal axis and sympathetic nervous system (Stamatakis & Punjabi, 2010). Briançon-Marjollet et al. (2015) provided endocrine and molecular insights, suggesting elevated IL-6 and TNF-α levels as mediators of insulin resistance under sleep-deprived states. Similarly, Carroll et al. (2015) showed that improving sleep quality led to reductions in biomarkers of systemic inflammation, further cementing the causal link between sleep health and metabolic regulation.

The current findings also intersect with public health and behavioral science. For instance, Taub and Redeker (2008) and Antza et al. (2022) emphasized that sleep disturbances co-occur with obesity, sedentary behavior, and dietary dysregulation—highlighting sleep as part of a broader behavioral matrix influencing diabetes risk. Interventions addressing sleep may thus exert broader benefits beyond glycemic control, such as reducing cardiovascular risk or depressive symptoms, as discussed by Tan et al. (2018).

In light of these observations, targeting sleep quality in diabetes care is no longer optional but imperative. While current clinical guidelines remain heavily pharmacocentric, growing evidence advocates for integrated, multidisciplinary strategies that encompass sleep hygiene education, behavioral therapy, and chronotherapy (Dodson & Zee, 2010; Reutrakul & Van Cauter, 2014). Additionally, emerging trials reviewed by Sondrup et al. (2022) show promise in manipulating sleep timing and duration to improve insulin sensitivity in real-world settings.

Finally, despite the clear trends, some limitations across studies persist. Heterogeneity in sleep assessment tools, population demographics, and glycemic outcome measures complicates direct comparisons. Moreover, as Tiwari et al. (2021) noted, most interventional studies remain small-scale and short in duration. Future research should prioritize large-scale, longitudinal, and mechanistically rich studies to delineate the precise causal pathways and optimize intervention strategies for diverse patient subgroups.

CONCLUSION

This systematic review demonstrates a robust association between sleep disorders—such as insomnia, obstructive sleep apnea, and circadian disruption—and dysregulation of glucose metabolism in individuals with type 2 diabetes mellitus (T2DM). Disrupted sleep architecture affects metabolic pathways through multiple mechanisms, including altered hormonal profiles, increased sympathetic activity, systemic inflammation, and impaired insulin sensitivity. The results underscore the need to integrate sleep assessment and management as part of routine diabetes care.

Furthermore, behavioral and pharmacologic sleep interventions have shown promising effects in improving glycemic outcomes, highlighting the therapeutic value of targeting sleep quality. However, clinical translation requires more consistent methodologies, standardized sleep assessments, and larger interventional trials. Treating sleep disorders could not only enhance glycemic control but also improve overall quality of life and reduce the risk of diabetic complications.

Limitations

While this review synthesizes current knowledge effectively, several limitations must be acknowledged. First, heterogeneity in study designs, sleep assessment tools, and glycemic outcome measures limited the feasibility of meta-analysis. Second, many studies relied on self-reported sleep data rather than objective measures such as polysomnography or actigraphy. Third, variations in participant demographics (e.g., age, BMI, ethnicity, medication use) may have introduced confounding effects not fully adjusted for in all studies.

Additionally, most interventional studies were of short duration and conducted on relatively small samples, restricting generalizability. The reliance on English-language publications may have excluded relevant international research. Finally, given the observational nature of most included studies, causality cannot be definitively established.

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