



International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
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ARTICLE TITLE	ROLE OF SLEEP DISORDERS IN TYPE 2 DIABETES MELLITUS - A REVIEW OF CURRENT LITERATURE
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DOI	https://doi.org/10.31435/ijitss.3(51).2026.6666
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RECEIVED	08 June 2026
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ACCEPTED	07 September 2026
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PUBLISHED	09 September 2026
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ROLE OF SLEEP DISORDERS IN TYPE 2 DIABETES MELLITUS - A REVIEW OF CURRENT LITERATURE

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) and sleep disturbances are highly prevalent conditions that increasingly coexist in clinical populations. Evidence suggests a complex, bidirectional relationship between sleep health and glucose metabolism, yet sleep assessment remains underintegrated into routine diabetes care.

Objective: This narrative review synthesizes current evidence on the relationship between sleep disorders and T2DM, covering prevalence, risk pathways, diabetic complications, and management strategies.

Methods: A literature search was conducted in PubMed and Google Scholar using keywords related to diabetes, sleep disorders, sleep duration, sleep quality, obstructive sleep apnea, insomnia, shift work, and circadian rhythm. Full-text English-language studies published between 2006 and 2026 involving human adult subjects were included.

Results: Sleep disorders affect more than half of all individuals with T2DM, with obstructive sleep apnea (55-86%), insomnia (39%), and restless legs syndrome (8-45%) being the most prevalent. Both short (<6 hours) and long (>9 hours) sleep duration are associated with increased T2DM incidence. Sleep disturbances contribute to both micro- and macrovascular complications through mechanisms including intermittent hypoxia, sympathetic activation, HPA axis dysregulation and chronic inflammation. Established diabetes worsens sleep through nocturia, neuropathic pain, and nocturnal glycemic fluctuations. Lifestyle interventions, continuous positive airway pressure for obstructive sleep apnea, and cognitive behavioral therapy for insomnia represent evidence-based management approaches, though their impact on long-term glycemic outcomes requires further study.

Conclusions: Sleep disorders are common, clinically significant, and modifiable comorbidities in T2DM. Routine sleep screening should be integrated into standard diabetes care, with phenotype-guided treatment of obstructive sleep apnea, insomnia, and restless legs syndrome. Large-scale, long-term randomized trials are needed to determine whether sustained sleep interventions reduce diabetic complications.

KEYWORDS

Type 2 Diabetes Mellitus, Sleep Disorders, Sleep Duration, Sleep Quality

CITATION

Bogdan-Groszyk, Z., Mikula, L., Otrębska, K., Lewandowska, D., Makulec, A., Józwik-Szymańska, A., Kostecka, N., Szredzka, A., & Kucharski, J. (2026). Role of sleep disorders in type 2 diabetes mellitus - a review of current literature. *International Journal of Innovative Technologies in Social Science*, 3(51). [https://doi.org/10.31435/ijitss.3\(51\).2026.6666](https://doi.org/10.31435/ijitss.3(51).2026.6666)

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Introduction

In recent decades, the population suffering from type 2 diabetes mellitus (T2DM) increased significantly. It is a major public health issue, which contributes greatly to worldwide disability and mortality [1,2]. Despite developing new diagnostic and therapeutic methods, preventing and managing diabetes mellitus type 2 remains a major challenge. Therefore, addressing lifestyle changes is becoming more important in clinical practice.

At the same time, we can observe a growing issue regarding sleep disturbances across the population. Sleep disorders are grouped into six major categories in the ICSD-3-TR: Insomnia Disorders, Sleep-Related Breathing Disorders, Central Disorders of Hypersomnolence, Circadian Rhythm Sleep-Wake Disorders, Parasomnias, Sleep-Related Movement Disorders [3].

Not only do sleep disorders negatively affect the quality of life of the patients, but they also have been associated with poor glycemic control, increased insulin resistance, and a higher risk of diabetes-related complications [4]. Reflecting this, the American Diabetes Association (ADA) in Standards of Care 2025 explicitly included considering the assessment of sleep pattern and duration in comprehensive diabetes evaluation [5].

It is worth highlighting how important the assessment of sleep behaviours should be as a part of routine checkups in populations at risk of developing diabetes mellitus type 2, or those who already are diagnosed. The objective of this narrative review is to summarize current evidence regarding the relationship between sleep disorders and diabetes mellitus type 2.

Methods

We conducted a search of publications using the PubMed database, as well as the Google Scholar database. Keywords such as “diabetes”, “type 2 diabetes mellitus”, “prediabetes”, “insulin resistance” and “sleep”, “sleep duration”, “sleeping”, “sleep quality”, “sleep disorders”, “insomnia”, “OSA”, “shift work” were used to find relevant papers. A representative PubMed search block was: (“type 2 diabetes” OR “T2DM” OR “prediabetes” OR “insulin resistance”) AND (“sleep” OR “insomnia” OR “sleep quality” OR “sleep duration” OR “obstructive sleep apnea” OR circadian OR “shift work” OR “restless legs syndrome”). Reference lists of key reviews and guidelines were also screened for additional relevant articles.

We included full-text English-language studies that were published between 2006 and 2026. Selected articles involved human adult subjects. Priority was given to systematic reviews, meta-analyses, randomized controlled trials, and prospective cohorts. Cross-sectional studies were also included. Case reports and pediatric-only studies were excluded.

Titles and abstracts were assessed in order to select relevant publications. Then, full texts were read and analyzed, which allowed us to include eligible papers. Publications which assess the relationship between sleep disturbances and diabetes mellitus type 2 were chosen as they fulfill the intended objective of the analysis. Findings were synthesized narratively by topic (prevalence, incident risk, bidirectional pathways, complications, and management). A comparative synthesis framework was used in order to contrast consistency of direction and magnitude across study designs rather than presenting isolated studies sequentially. Because this is a narrative review, no *de novo* meta-analysis was performed and no single risk-of-bias instrument was applied across all included designs.

Results

Prevalence of sleep disorders in type 2 diabetics

Available data highly suggests that sleep disorders occur more commonly in the population suffering from type 2 diabetes mellitus than in the general population. A review by Schipper et al. found that the most prevalent disorders include insomnia, obstructive sleep apnea (OSA), restless legs syndrome (RLS), as well as excessive daytime sleepiness and circadian rhythm disturbances. Prevalence of insomnia, defined as difficulty initiating and maintaining sleep or by waking up earlier than desired despite adequate opportunity to sleep, was 39% in people with type 2 diabetes mellitus, which is four times higher than the general population. OSA - a sleep disorder characterized by repeated episodes of complete (apnea) or partial (hypopnea) collapse of the upper airway, causing oxygen desaturation or sleep arousal, leading to fragmented, nonrestorative sleep, was present in 55 to 86% of patients. The percentage varied depending on sex and body mass - men and obese individuals were at higher risk of being diagnosed with obstructive sleep apnea. Restless legs syndrome is characterised by the urge to move in response to uncomfortable and unpleasant sensations in the legs during periods of rest or inactivity, thus interfering with sleep. The prevalence of RLS varies from 8 to 45%, however the authors highlight the risk of overrepresentation due to inability to differentiate some cases of RLS from peripheral neuropathy [4].

A systematic review and meta-analysis conducted by Khalil et al., which included 41 studies, estimated the overall pooled prevalence of diagnosed sleep disorders in adults with diabetes at 52%. Sleep apnea was the most frequent disorder (69% for unspecified sleep apnea and 60% for OSA), followed by restless legs syndrome (27%) [6]. Cross-sectional studies from different regions similarly report a high burden of poor sleep quality complaints in T2DM populations [7,8].

Overall, current evidence indicates that sleep disorders affect more than half of all individuals with T2DM, placing them among the most common diabetes-related comorbidities [5-8]. However, estimates vary because of differences in case definitions, objective vs subjective measurement, and referral bias in sleep clinic-based cohorts. Obstructive sleep apnea, insomnia, and restless legs syndrome account for the majority of diagnosed disorders, while poor sleep quality is even more widespread.

Sleep disorders as a risk factor for developing type 2 diabetes mellitus

Although obesity, the lack of physical activity, poor dietary habits and genetic predisposition remain the classic primary risk factors, a number of prospective cohort studies, systematic reviews, and meta-analyses indicate that both quantitative and qualitative aspects of sleep influence glucose metabolism and insulin sensitivity. This poses sleep habits as an important, potentially modifiable risk factor.

Regarding the duration of sleep, there have been studies that describe an U-shaped association between the length of sleep and incidence of type 2 diabetes mellitus. A landmark meta analysis by Cappuccio et al. reported that individuals who slept fewer than 5-6 hours per night had a significantly higher risk of developing T2DM compared with those sleeping 7-8 hours. Long sleep duration (>8-9 hours) was also associated with increased diabetes risk [9]. While short sleep is thought to increase metabolic risk through several mechanisms, such as decreased insulin sensitivity, increased sympathetic nervous system activity, elevated evening cortisol concentrations, and alterations in appetite-regulating hormones [9, 10], long sleep duration is more difficult to interpret [9, 15]. Individuals who report prolonged sleep also present with depressive symptoms, low socioeconomic status, unemployment, a low level of physical activity, undiagnosed health conditions, and poor general health. Those factors might confound the association [9].

Sleep quality encompasses aspects such as subjective satisfaction, sleep latency, efficiency and nocturnal awakenings. Subjective poor sleep quality and fragmented sleep have been associated with impaired glucose tolerance, reduced insulin sensitivity, and increased inflammatory activity even when total sleep duration remains within the recommended range [11]. Objective sleep assessments further suggest that reduced sleep efficiency independently predicts future disturbances in glucose metabolism [12]. Frequent nocturnal awakenings disrupt slow-wave sleep, a critical stage for maintaining physiological endocrine and metabolic function [12].

One of the most investigated sleep disorders associated with higher T2DM risk is obstructive sleep apnea (OSA). Epidemiological studies consistently demonstrate that individuals with OSA exhibit significantly higher rates of insulin resistance, impaired glucose tolerance, metabolic syndrome, and T2DM compared with the general population [13]. Importantly, these associations persist even after adjustment for body mass index, indicating that OSA contributes independently to metabolic dysfunction [4]. Large observational studies indicate that increasing OSA severity is associated with progressively greater diabetes risk. Individuals with severe OSA frequently demonstrate elevated fasting glucose concentrations, increased glycated hemoglobin (HbA1c), and impaired β -cell function [14]. However, confounding aspects such as age and obesity should be taken into account [14].

Insomnia symptoms, especially when persistent and accompanied by short objective sleep, increase the risk of incident T2DM [10, 13]. Insomnia contributes to sustained activation of the hypothalamic-pituitary-adrenal axis, resulting in elevated cortisol secretion and impaired insulin action. Sleep deprivation and fragmentation also increases sympathetic nervous system activity, promotes systemic inflammation, and alters glucose homeostasis [15].

An important group of patients worth mentioning and distinguishing are shift workers. Long-term rotating night-shift exposure is associated with increased T2DM incidence in large cohorts, including the Nurses' Health Study cohorts (Pan et al.) [16]. Meta-analyses estimate that shift workers experience approximately a 10-40% higher risk of diabetes. The direction of evidence is consistent with circadian-disruption mechanisms, but effect sizes vary by occupation, cumulative exposure, and lifestyle covariates [17,18].

Overall, associations between sleep pathology and T2DM are strongest and most replicated for sleep-duration extremes and OSA, while causal interpretation remains constrained because intervention studies targeting incident diabetes prevention are limited.

A bidirectional relationship

The relationship between sleep disorders and T2DM is bidirectional. On one side, sleep disruption may worsen insulin sensitivity, appetite control, autonomic balance, and inflammatory tone. It is also worth highlighting that sleep pathology may affect diabetes by impairing daytime decision-making, increasing the likelihood of unhealthy dietary choices, reduced physical activity, and poorer adherence to self-management behaviors.

On the other side, established diabetes may worsen sleep via nocturia, neuropathic pain, nocturnal hypoglycemia concern, depression, and polypharmacy effects [4,5,14]. This creates a vicious cycle, therefore clinical pathways should evaluate both aspects rather than treating insufficient sleep only as a secondary complaint.

Sleep disorders and diabetes complications

Sleep disorders have been associated with both microvascular and macrovascular diabetic complications [4]. Considered mechanisms include intermittent hypoxia, oxidative stress, systemic inflammation, endothelial dysfunction, sympathetic nervous system activation, and persistent hyperglycaemia.

Poor sleep quality and reduced sleep efficiency have been linked with elevated HbA1c, which is the main driver of microvascular damage [19]. In the context of diabetic retinopathy, OSA is particularly detrimental. The chronic intermittent hypoxemia associated with OSA increases the production of vascular endothelial growth factor (VEGF), resulting in proliferative retinopathy, and reactive oxygen species. Longitudinal data indicate that OSA is an independent risk factor for the development and rapid progression to sight-threatening proliferative retinopathy, a risk that may be partially mitigated by early intervention with continuous positive airway pressure (CPAP) [20]. Furthermore, diabetic peripheral neuropathy shows a strong independent association with OSA. Studies demonstrate that patients with comorbid OSA exhibit elevated levels of circulating nitrotyrosine and lipid peroxides, markers of oxidative stress that correlate with the severity of nocturnal hypoxemia and nerve damage [21]. Patients with neuropathy frequently report insomnia and reduced sleep quality due to neuropathic pain [22], while sleep disruption itself may further exacerbate neural injury through inflammatory and metabolic mechanisms. The kidneys are also highly susceptible to the ischemic and inflammatory stress induced by sleep disturbances. A pathological triad consisting of OSA, systemic hypertension, and chronic kidney disease is frequently observed in diabetic cohorts, where the nocturnal blood pressure non-dipping pattern typical of OSA and chronic insomnia accelerates the decline of the estimated glomerular filtration rate [23, 24, 25].

At the macrovascular level, the relationship between sleep disturbances and cardiovascular health is multifaceted. Both short sleep durations (≤ 5 hours) and long sleep durations (≥ 9 -10 hours) were independently associated with a marked increase in the incidence of major adverse cardiovascular events (MACE) and CVD-related mortality in individuals with T2DM [26]. Large-scale meta-analyses confirm that deviation from the optimal 7-8 hour sleep window significantly elevates the risk of ischemic stroke, coronary heart disease, and heart failure, likely mediated by endothelial dysfunction and chronic low-grade systemic inflammation [27]. Circadian misalignment and rotating night shift work are particularly insidious, as they disrupt the timing of glucose metabolism and lipid regulation, leading to a state of metabolic inflexibility that promotes atherosclerosis [16]. Insomnia also contributes to macroangiopathy through the activation of the hypothalamic-pituitary-adrenal axis [15], leading to sustained hypertension and increased arterial stiffness. OSA acts as an independent risk factor for resistant hypertension and atrial fibrillation, triggering massive sympathetic nervous system surges during apneic events that lead to left ventricular remodeling [28].

Current evidence supports sleep disorders as clinically meaningful risk markers and potential contributors to diabetes complications. However, most evidence remains observational and should be interpreted as association rather than proof of direct causation.

Management of sleep disorders in type 2 diabetes mellitus

Routine screening for sleep disorders should be incorporated into diabetes care, particularly in patients with obesity, resistant hypertension, excessive daytime sleepiness, cardiovascular disease, or poor glycaemic control. The ADA Professional Practice Committee recommends evaluating sleep quality during routine diabetes assessment because sleep disturbances significantly influence metabolic outcomes [5]. Practical tools to consider include Pittsburgh Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), STOP-Bang questionnaire, and Insomnia Severity Index (ISI), with confirmatory testing for suspected OSA via polysomnography or home sleep apnea testing when appropriate [13]. When it comes to lifestyle interventions, they remain the baseline for managing sleep disorders in T2DM patients [5, 29]. For example, weight reduction in people with obesity and OSA was associated with improvements in OSA severity as assessed by AHI (Apnea-Hypopnea Index). The degree of AHI improvement was associated with the magnitude of weight reduction [30]. Regular physical activity contributes to improved sleep quality, reduced sleep latency, enhanced insulin sensitivity, decreased inflammation and lower cardiovascular risk [31-33]. Sleep hygiene education is equally important. Patients should be encouraged to maintain consistent sleep schedules, avoid caffeine and alcohol before bedtime, reduce evening exposure to electronic devices, and optimize the sleeping environment [5, 29].

Optimization of diabetes pharmacotherapy contributes indirectly to improved sleep by reducing nocturia, neuropathic pain, obesity, and glucose variability. Modern obesity-pharmacotherapy pathways (GLP-1 receptor agonists and dual GIP/GLP-1 agonists) may reduce OSA severity largely through weight loss and

cardiometabolic improvement [34, 35]. SGLT2 inhibitors are also being studied for possible favorable OSA-related effects through fluid and cardiometabolic mechanisms, but evidence remains early and heterogeneous [36]. These approaches are promising adjuncts, not replacements for established sleep-disorder treatment pathways.

Continuous positive airway pressure (CPAP) is the first-line treatment for moderate-to-severe obstructive sleep apnoea, with well-established efficacy in reducing daytime sleepiness, blood pressure, and cardiovascular risk [37]. However, its impact on glycaemic control in patients with comorbid type 2 diabetes mellitus (T2DM) remains inconclusive. The evidence base is heterogeneous, and conclusions depend on study design, population characteristics, CPAP adherence, and the glycaemic endpoint assessed. Some meta-analyses indicate that CPAP produces modest reductions in HbA1c, alongside improvements in insulin resistance and inflammatory biomarkers [38]. On the other side, several meta-analyses and large randomised controlled trials have reported null effects on HbA1c [39, 40]. A growing body of evidence suggests that the metabolic consequences of OSA may be more closely reflected in glycaemic variability than in mean HbA1c levels [41].

When it comes to insomnia, clinical guidelines recommended cognitive behavioural therapy for insomnia (CBT-I) as first-line treatment [42]. CBT-I addresses dysfunctional sleep behaviours through stimulus control, sleep restriction, cognitive restructuring, relaxation techniques and sleep hygiene education. Some randomized trials have demonstrated improvements in sleep quality accompanied by modest improvements in glycaemic control following CBT-I [43]. However, they have several limitations, such as small control groups and short observation periods. Pharmacological treatment should be reserved for selected patients when behavioural interventions prove insufficient. Short-term use of non-benzodiazepine hypnotics, melatonin receptor agonists, or low-dose doxepin may be considered, while long-term benzodiazepine use is generally discouraged because of dependence, falls, cognitive impairment, and worsening sleep architecture [42].

Restless legs syndrome (RLS) requires careful differential diagnosis from diabetic neuropathy. Clinicians should also address exacerbating factors such as alcohol, caffeine, antihistaminergic, serotonergic, antidopaminergic medications, and untreated obstructive sleep apnea. Management includes correction of iron deficiency when indicated and use of $\alpha 2\delta$ calcium channel ligands (gabapentin or pregabalin) or dopamine agonists in appropriately selected patients [44].

Discussion

Analysis of the available evidence confirms that sleep disorders are common across the population suffering from type 2 diabetes mellitus. They are consistently associated with adverse metabolic and clinical results. The strongest and most reproducible data is available for OSA and insomnia. Evidence certainty is highest for symptom and sleep-quality improvement after phenotype-targeted treatment, moderate for short-term metabolic improvements in selected groups, and lower for hard long-term diabetes outcomes such as complication prevention.

While this study provides strong evidence for the importance of managing sleep pathology in T2DM, several limitations must be acknowledged. Firstly, the mostly observational nature of the data limits our ability to definitively establish causality. Secondly, the reliance on self-reported sleep quality in a portion of the cohorts may introduce recall bias. Sleep measurement tools are not uniform across studies. Future research should leverage objective measures, and large-scale, long-term randomized controlled trials are urgently needed to determine whether sustained sleep interventions can reduce macrovascular and microvascular diabetes complications over time.

In practical terms, this review supports routine sleep screening in diabetes care and phenotype-guided treatment of likely OSA, chronic insomnia, and RLS at present, rather than waiting for definitive causal proof. In contrast, the magnitude of long-term HbA1c improvement, complication prevention, and cardiometabolic risk reduction from sleep-targeted therapy remains hypothesis-generating and should be framed cautiously. The same caution applies to emerging pharmacologic approaches: they are promising adjuncts, but current evidence is insufficient to treat them as established sleep-disorder therapies. The central clinical message is that sleep assessment is warranted now, while hard-outcome benefit still requires stronger long-term trials.

Conclusions

Sleep disorders are a key risk factor both for developing and poor control of diabetes mellitus type 2. More importantly, it is modifiable. Lately the field has been shifting towards actionable management - from routine screening and lifestyle interventions (sleep hygiene, CBT-I) to specific treatments, for example for OSA. Optimal treatment requires collaboration among endocrinologists, sleep physicians, primary care physicians, psychologists, dietitians, physiotherapists, and diabetes educators. Future clinical practice should systematically integrate sleep assessment into standard diabetes care pathways and apply phenotype-specific treatment strategies (for example CPAP for OSA and CBT-I for chronic insomnia). The evidence base for complication prevention and sustained glycemic benefit is promising but still incomplete, and future long-term randomized studies are needed to define which sleep interventions provide the greatest metabolic and cardiovascular benefit in specific T2DM subgroups.

Conflicts of Interest Statement: No conflicts of interest to declare.

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