

Research Article

Association of Sleep Quality, Depression, and Quality of Life with Glycemic Control in Patients with Type 2 Diabetes Mellitus

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Abstract

Objectives: To evaluate the relationship between sleep quality, depressive symptoms, and health-related quality of life (HRQoL) in patients with type 2 diabetes mellitus (T2DM) and to identify independent predictors of poor sleep quality.

Methods: In this cross-sectional study, 100 adults with T2DM were assessed using the Pittsburgh Sleep Quality Index (PSQI), Beck Depression Inventory (BDI), and 36-Item Short Form Health Survey (SF-36). Poor sleep quality was defined as PSQI >5. Multivariable logistic and linear regression analyses were performed after adjustment for age, sex, body mass index, diabetes duration, glycated hemoglobin (HbA1c), and BDI score.

Results: Fifty-seven patients (57%) had poor sleep quality. Compared with good sleepers, they had higher HbA1c ($8.7 \pm 1.1\%$ vs. $8.2 \pm 0.8\%$, $p=0.03$), higher BDI scores (11 vs. 4, $p<0.001$), and lower scores across all SF-36 domains (all $p<0.05$). PSQI correlated positively with BDI ($p=0.58$, $p<0.001$) and negatively with SF-36 Vitality ($p=-0.71$, $p<0.001$). After adjustment, only BDI score independently predicted poor sleep quality (OR 1.19, 95% CI 1.09–1.31; $p<0.001$).

Conclusion: Poor sleep quality in T2DM was independently associated with depressive symptom severity rather than glycemic control and was accompanied by impaired HRQoL. Screening for depressive symptoms and sleep disturbance may improve comprehensive diabetes care.

Keywords: Depression, sleep quality, type 2 diabetes mellitus

Cite This Article: Korkmaz Yılmaz M, Hatipoğlu E. Association of Sleep Quality, Depression, and Quality of Life with Glycemic Control in Patients with Type 2 Diabetes Mellitus. EJMI 2026;10(4):583–589.

Type 2 diabetes mellitus (T2DM) is a chronic metabolic condition whose global prevalence has continued to rise in recent decades, affecting an estimated 537 million adults in 2021, with projections suggesting further increases in the coming decades.^[1] Beyond its well-established metabolic and cardiovascular consequences, T2DM is increasingly recognized as a condition with substantial

psychological and behavioral burden that extends well beyond glycemic control alone.

Sleep disturbance is highly prevalent among patients with T2DM, with reported rates of poor sleep quality ranging widely across populations, from approximately 18% to nearly 80% depending on the assessment method and population studied.^[2] The Pittsburgh Sleep Quality Index (PSQI),

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Submitted Date: July 30, 2026 **Accepted Date:** September 14, 2026

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developed by Buysse et al.^[3], remains the most widely used instrument for the subjective assessment of sleep quality and has been validated in numerous populations, including a Turkish adaptation.^[4] Importantly, poor sleep quality in T2DM has been associated with impaired glycemic control; prior work adjusting for age, sex, and body mass index (BMI) has demonstrated meaningfully higher glycated hemoglobin (HbA1c) levels among poor sleepers compared with good sleepers^[2], suggesting a bidirectional relationship in which disrupted sleep may both result from and contribute to metabolic dysregulation. Beyond its association with impaired quality of life, poor sleep has also been prospectively linked to an increased risk of developing T2DM itself, with meta-analytic evidence showing that both short sleep duration and difficulty maintaining sleep predict incident diabetes.^[5] Sleep disorders in T2DM have also been reviewed extensively in relation to associated health outcomes more broadly.^[6-8] Depression represents a major global health burden, affecting hundreds of millions of people worldwide and ranking among the leading causes of disability.^[9] It is another common yet frequently under-recognized comorbidity in T2DM. A large-scale meta-analysis of observational studies estimated a pooled global depression prevalence of approximately 28% among patients with T2DM, roughly double the rate observed in the general population.^[10] Depressive symptoms in this population have been linked to poorer self-management behaviors, worse glycemic outcomes, and increased risk of diabetes-related complications.^[10,11] The Beck Depression Inventory (BDI), originally developed by Beck et al.^[12] and validated in Turkish populations^[13], is among the most widely used self-report instruments for quantifying depressive symptom severity in clinical research. Health-related quality of life (HRQoL), commonly assessed using the 36-Item Short Form Health Survey (SF-36)^[14], provides a multidimensional measure of physical and mental well-being that captures the functional impact of chronic disease beyond biochemical markers. A systematic review examining the relationship between sleep and quality of life in T2DM reported a moderate negative association between PSQI global scores and SF-36 subdimensions, alongside a strong positive correlation between PSQI and depression scores.^[15] These findings indicate that sleep, mood, and quality of life are closely interrelated in this population. However, most existing studies have examined these relationships in isolation or in pairs. Few have simultaneously characterized how sleep quality, depressive symptoms, and multidimensional quality of life relate to one another and to glycemic control within a single T2DM cohort. Understanding these interrelationships may help identify modifiable factors associated with poor sleep quality and support more comprehensive patient management.

Therefore, the present study aimed to evaluate the relationship between sleep quality, depressive symptoms, and HRQoL in patients with T2DM and to determine the independent clinical and psychological predictors of poor sleep quality after adjusting for demographic and metabolic parameters.

Material and Methods

Study Design and Participants

This single-center, cross-sectional observational study was conducted at the outpatient clinic of the Department of Endocrinology and Metabolic Diseases, Basaksehir Cam and Sakura City Hospital. Consecutive adult patients with an established diagnosis of T2DM who had attended routine outpatient follow-up visits at this clinic between July 2025 and June 2026 were screened for eligibility using retrospective medical records. Following ethics committee approval, eligible patients were prospectively invited to complete the study questionnaires (PSQI, BDI, and SF-36) between June and July 2026. The study protocol was approved by the Basaksehir Cam and Sakura City Hospital Scientific Research 2nd Ethics Committee (no: 315, date: 03.06.2026) and was conducted in accordance with the ethical principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to study enrollment.

Eligible participants were required to be ≥ 18 years of age, have a confirmed diagnosis of T2DM, possess sufficient cognitive ability to understand and complete the study questionnaires, provide written informed consent, and have an available HbA1c measurement obtained within the previous three months. Patients with type 1 diabetes mellitus, pregnancy, active malignancy, severe psychiatric disease or cognitive impairment interfering with questionnaire completion, chronic systemic glucocorticoid treatment, or incomplete clinical or laboratory data were excluded.

Sample size estimation was based on the primary hypothesis of a correlation between sleep quality and HbA1c. Assuming a moderate correlation coefficient ($r=0.30$), a two-sided α level of 0.05, and 80% statistical power, a minimum sample size of 84 participants was required. To compensate for potential missing data and incomplete questionnaires, the planned recruitment target was set at 100 participants.

Data Collection

Demographic and clinical characteristics, including age, sex, diabetes duration, height, weight, BMI, current antidiabetic treatment, and accompanying comorbidities, were obtained from the electronic medical records and hospital

information system. Laboratory variables, including HbA1c and fasting plasma glucose, were retrieved from routine biochemical measurements performed during standard outpatient follow-up. No additional laboratory investigations were performed specifically for the study.

Assessment of Sleep Quality

Sleep quality was evaluated using the PSQI, a validated 19-item self-administered questionnaire that assesses subjective sleep quality during the preceding month across seven domains, including sleep duration, sleep latency, sleep efficiency, sleep disturbances, use of sleep medication, daytime dysfunction, and subjective sleep quality.^[3] The global PSQI score ranges from 0 to 21, with higher scores indicating poorer sleep quality. Consistent with the original validation study, participants with a global PSQI score >5 were classified as poor sleepers.^[3] The validated Turkish version of the PSQI^[4] was administered to all participants during their outpatient visit.

Assessment of Depressive Symptoms

Depressive symptoms were assessed using the BDI, a validated 21-item self-report questionnaire designed to evaluate the severity of depressive symptoms.^[12] Each item is scored from 0 to 3, yielding a total score ranging from 0 to 63, with higher scores indicating more severe depressive symptomatology. The validated Turkish version of the BDI^[13] was used throughout the study.

Assessment of Health-Related Quality Of Life

HRQoL was assessed using the SF-36, a widely used generic instrument for evaluating physical and mental health status.^[14] The SF-36 comprises eight domains: physical functioning, role physical, bodily pain, general health, vitality, social functioning, role emotional, and mental health. Scores for each domain range from 0 to 100, with higher scores indicating better perceived HRQoL. The validated Turkish version of the SF-36 was administered to all participants.^[16]

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Shapiro–Wilk test. Continuous variables are presented as mean±standard deviation (SD) or median with interquartile range (IQR), as appropriate according to data distribution, whereas categorical variables are expressed as frequencies and percentages.

Comparisons between patients with good sleep quality (PSQI ≤5) and poor sleep quality (PSQI >5) were performed

using the independent-samples t test or the Mann–Whitney U test for continuous variables and the chi-square test or Fisher's exact test for categorical variables, as appropriate.

Associations between PSQI total score and continuous clinical, metabolic, psychological, and health-related quality-of-life variables were evaluated using Spearman's rank correlation coefficient (ρ).

Multivariable logistic regression analysis was performed to identify independent predictors of poor sleep quality (PSQI >5). Variables considered clinically relevant or associated with sleep quality in univariable analyses were entered into the multivariable model, including age, sex, BMI, diabetes duration, HbA1c, and BDI score. Odds ratios (ORs) and corresponding 95% confidence intervals (CIs) were calculated.

In addition, multivariable linear regression analysis was performed to determine independent factors associated with the continuous PSQI total score using the same covariates. HbA1c was selected a priori as the primary marker of long-term glycemic control; therefore, fasting plasma glucose was not included in multivariable analyses. All statistical tests were two-sided, and a $p < 0.05$ was considered statistically significant.

Results

A total of 100 patients with T2DM were enrolled in this cross-sectional study. The mean age was 56.3 ± 9.8 years, and 58% of participants were female. The mean BMI was 31.4 ± 5.2 kg/m², mean diabetes duration was 9.4 ± 6.3 years, and mean HbA1c was $8.5 \pm 1.0\%$. Insulin was used by 36% of patients, and 62% had a diagnosis of hypertension. The mean PSQI score was 6.7 ± 3.4 , and 57 patients (57%) were classified as poor sleepers (PSQI >5). The median BDI score was 8 (IQR 4–13). Baseline characteristics are summarized in Table 1.

When patients were stratified by sleep quality, no significant differences were observed between good and poor sleepers in age, sex, BMI, diabetes duration, insulin use, or hypertension (all $p > 0.05$), indicating that the two groups were well balanced at baseline. Poor sleepers had significantly higher HbA1c levels than good sleepers ($8.7 \pm 1.1\%$ vs. $8.2 \pm 0.8\%$, $p = 0.03$). BDI scores were markedly higher among poor sleepers (median 11 [IQR 7–16] vs. 4 [IQR 2–7], $p < 0.001$). All eight assessed SF-36 subscales were significantly lower in the poor sleep group (all $p < 0.05$; Table 2), with the largest differences observed for Vitality (55 vs. 77, $p < 0.001$), Mental Health (63 vs. 81, $p < 0.001$), Bodily Pain (62 vs. 76, $p = 0.002$), and Social Function (68 vs. 84, $p < 0.001$). These findings suggest that poor sleep quality was more closely associated with depressive symptoms and impaired HRQoL than with glycemic control.

Table 1. Baseline demographic and clinical characteristics of the study population

Variable	Total (n=100)
Age, years	56.3±9.8
Female sex, n (%)	58 (58.0)
BMI, kg/m ²	31.4±5.2
Diabetes duration, years	9.4±6.3
HbA1c, %	8.5±1.0
Fasting glucose, mg/dL	142±41
Insulin use, n (%)	36 (36.0)
Hypertension, n (%)	62 (62.0)
PSQI total score	6.7±3.4
Poor sleep quality (PSQI > 5), n (%)	57 (57.0)
BDI score, median (IQR)	8 (4–13)
SF-36 Physical Function	79±15
SF-36 Physical Role	74±24
SF-36 Bodily Pain	68±20
SF-36 General Health	66±17
SF-36 Vitality	65±18
SF-36 Social Function	76±18
SF-36 Emotional Role	73±23
SF-36 Mental Health	71±17

Data are presented as mean±SD, n (%), or median (IQR), as appropriate. BDI: Beck Depression Inventory; BMI: Body mass index; HbA1c: Glycated hemoglobin; PSQI: Pittsburgh Sleep Quality Index; SF-36: 36-Item Short Form Health Survey.

PSQI total score showed a weak but significant positive correlation with HbA1c ($p=0.27$, $p=0.007$), while correlations with age ($p=0.08$, $p=0.41$), BMI ($p=0.09$, $p=0.35$), and diabetes duration ($p=0.12$, $p=0.21$) were not statistically significant. In contrast, PSQI score was strongly and positively correlated with BDI score ($p=0.58$, $p<0.001$) and showed strong inverse correlations with SF-36 Vitality ($p=-0.71$, $p<0.001$) and Mental Health ($p=-0.69$, $p<0.001$), as well as moderate-to-strong inverse correlations with all remaining SF-36 subscales, including Social Function ($p=-0.56$), General Health ($p=-0.52$), Bodily Pain ($p=-0.43$), Physical Function ($p=-0.39$), Emotional Role ($p=-0.48$), and Physical Role ($p=-0.36$) (all $p<0.001$; Table 3). These findings indicate that depressive symptom burden and impaired HRQoL — rather than metabolic or anthropometric variables — are the factors most closely linked to poor sleep quality in this population.

In multivariable logistic regression analysis, BDI score was the only independent predictor of poor sleep quality (OR 1.19, 95% CI 1.09–1.31, $p<0.001$), corresponding to a 19% increase in the odds of poor sleep for each one-point in-

Table 2. Comparison of clinical, psychological, and quality-of-life parameters between good and poor sleepers

Variable	Good sleep (n=43)	Poor sleep (n=57)	p
Age, years	55.7±9.5	56.8±10.0	0.58
Female sex	23 (53%)	35 (61%)	0.42
BMI, kg/m ²	30.9±4.8	31.8±5.5	0.36
Diabetes duration, years	8.9±6.0	9.8±6.5	0.47
Insulin use	14 (33%)	22 (39%)	0.54
Hypertension	25 (58%)	37 (65%)	0.49
HbA1c, %	8.2±0.8	8.7±1.1	0.03
BDI	4 (2–7)	11 (7–16)	<0.001
SF-36 Physical Function	83±12	75±16	0.004
SF-36 Physical Role	81±20	69±24	0.01
SF-36 Bodily Pain	76±17	62±20	0.002
SF-36 General Health	73±14	61±16	<0.001
SF-36 Vitality	77±15	55±17	<0.001
SF-36 Social Function	84±14	68±18	<0.001
SF-36 Emotional Role	81±19	67±24	0.006
SF-36 Mental Health	81±13	63±17	<0.001

Good sleep quality was defined as PSQI ≤ 5 and poor sleep quality as PSQI > 5. p-values were derived from independent-samples t-test or Mann-Whitney U test, as appropriate, based on the distribution of each variable. BDI: Beck Depression Inventory; HbA1c: Glycated hemoglobin; PSQI: Pittsburgh Sleep Quality Index; SF-36: 36-Item Short Form Health Survey.

Table 3. Correlation of PSQI total score with clinical, psychological, and quality-of-life parameters

Variable	Spearman's ρ	p
Age	0.08	0.41
BMI	0.09	0.35
Diabetes duration	0.12	0.21
HbA1c	0.27	0.007
BDI	0.58	<0.001
SF-36 Physical Function	-0.39	<0.001
SF-36 Physical Role	-0.36	<0.001
SF-36 Bodily Pain	-0.43	<0.001
SF-36 General Health	-0.52	<0.001
SF-36 Vitality	-0.71	<0.001
SF-36 Social Function	-0.56	<0.001
SF-36 Emotional Role	-0.48	<0.001
SF-36 Mental Health	-0.69	<0.001

Correlation coefficients (Spearman's ρ) represent the rank correlation between PSQI total score and each listed variable. BDI: Beck Depression Inventory; BMI: Body mass index; HbA1c: Glycated hemoglobin; PSQI: Pittsburgh Sleep Quality Index; SF-36: 36-Item Short Form Health Survey.

Table 4. Multivariable logistic regression analysis for predictors of poor sleep quality (PSQI > 5)

Variable	OR	95% CI	p
Age	1.02	0.98–1.06	0.29
Female sex	1.11	0.51–2.41	0.78
BMI	1.01	0.94–1.09	0.82
Diabetes duration	1.03	0.98–1.08	0.18
HbA1c	1.29	0.88–1.92	0.16
BDI score	1.19	1.09–1.31	<0.001

Dependent variable: poor sleep quality (PSQI > 5, reference: PSQI ≤ 5). BDI: Beck Depression Inventory; OR: Odds ratio; CI: Confidence interval; BMI: Body mass index; HbA1c: Glycated hemoglobin; PSQI: Pittsburgh Sleep Quality Index.

crease in depressive symptom score. Age (OR 1.02, 95% CI 0.98–1.06, $p=0.29$), female sex (OR 1.11, 95% CI 0.51–2.41, $p=0.78$), BMI (OR 1.01, 95% CI 0.94–1.09, $p=0.82$), diabetes duration (OR 1.03, 95% CI 0.98–1.08, $p=0.18$), and HbA1c (OR 1.29, 95% CI 0.88–1.92, $p=0.16$) were not independently associated with poor sleep quality after adjustment, suggesting that the univariable association between HbA1c and poor sleep quality may be at least partly explained by depressive symptom burden (Table 4).

Fasting glucose was included in the baseline characterization (Table 1) but was not entered into subsequent correlation or regression analyses due to its collinearity with HbA1c, which was used as the primary glycemic control parameter.

Discussion

In this cross-sectional study of 100 patients with T2DM, depressive symptom severity was the only variable independently associated with poor sleep quality after adjustment for age, sex, BMI, diabetes duration, and HbA1c. Poor sleep quality was also associated with substantially reduced quality of life across all SF-36 domains.

In the present sample, 57% of patients met criteria for poor sleep quality, a figure within the upper range of estimates reported for T2DM populations, which vary from approximately 18% to 80% depending on setting and diagnostic threshold.^[2,15] A systematic review of 24 studies categorized predictors of poor sleep quality in T2DM into socio-demographic, disease-related, psychological, and physical domains.^[2] Our findings align most closely with the psychological domain, as depressive symptoms outweighed all clinical and demographic variables examined. Diabetes duration and insulin use, previously identified as relevant in other cohorts, did not reach significance in this sample, which may reflect differences in sample size or population characteristics relative to the broader evidence base.

Poor sleepers in this cohort had higher HbA1c than good sleepers in univariable analysis (8.7% vs. 8.2%, $p=0.03$); however, this association was attenuated after adjustment for depressive symptoms in the multivariable model ($p=0.16$). This finding diverges from at least one prior study reporting that sleep quality remained independently associated with HbA1c even after adjustment for age, sex, BMI, and diabetes-related complications.^[2] A direct, depression-independent mechanism linking sleep and glycemic control is nonetheless well supported experimentally: short-term sleep restriction has been shown to impair glucose tolerance and insulin sensitivity^[17], and meta-analytic evidence from prospective cohorts indicates that poor sleep quality predicts incident T2DM.^[5] The present data do not contradict this mechanism but suggest that, within this cohort, depressive symptom burden accounted for a substantial portion of the variance otherwise attributable to HbA1c. This raises the possibility that studies not accounting for depressive symptoms may overestimate the independent contribution of glycemic control to sleep quality.

PSQI and BDI scores were strongly correlated ($p=0.58$, $p<0.001$), consistent with a systematic review reporting a comparably strong association between these constructs across multiple T2DM studies.^[15] This finding is consistent with broader epidemiological data indicating that depression affects approximately 28% of patients with T2DM globally, approximately twice the prevalence observed in the general population^[10], within the context of depression being among the leading causes of disability worldwide.^[9] The present study extends this literature by providing a multivariable-adjusted estimate: each one-point increase in BDI score was associated with a 19% increase in the odds of poor sleep quality, independent of all other covariates. Given the well-established psychometric properties of the BDI across multiple populations^[18], this finding is unlikely to reflect measurement artifact.

All eight SF-36 subscales were significantly lower among poor sleepers, with the largest differences observed for Vitality and Mental Health, the same two domains most strongly correlated with PSQI score. This pattern is consistent with a systematic review reporting a moderate negative association between PSQI and SF-36 subdimensions, alongside a strong positive association between PSQI and depression scores, across multiple T2DM studies.^[15] That review further described individual studies in which the co-occurrence of poor sleep quality and psychological distress was associated with a disproportionately greater reduction in quality of life than either factor alone. Although anxiety was not assessed in the present study, the finding that the psychological variable examined accounted for more of the sleep-quality-of-life association than the metabolic variable is consistent with this broader pattern in the literature.

Two non-mutually exclusive mechanisms may underlie these findings. The first involves behavioral and inflammatory pathways, whereby depression is thought to worsen diabetes-related outcomes through chronic low-grade inflammation, neuroendocrine dysregulation, and reduced treatment adherence.^[19] The second involves a more direct physiological pathway, whereby sleep loss independently disrupts cortisol secretion, sympathetic activity, and appetite-regulating hormones such as leptin and ghrelin.^[17,20] Both mechanisms are biologically plausible; however, only the depression-related pathway remained significant after adjustment in the present analysis, which does not exclude a contribution from the second mechanism but suggests it was comparatively less influential in this cohort. Given the frequent co-occurrence of depressive symptoms and disrupted sleep architecture, a bidirectional relationship, in which impaired mood and impaired sleep mutually reinforce one another, remains a plausible interpretation, although the cross-sectional design precludes any determination of temporal precedence.

The relationships between sleep quality and glycemic control, and separately between sleep quality and quality of life, have each been investigated extensively, typically in isolation. Comparatively few studies have examined sleep quality, depressive symptoms, and multidimensional quality of life concurrently within a single, multivariable-adjusted analysis in a T2DM cohort. The present study addresses this gap and indicates that, within this cohort, depressive symptoms rather than glycemic control or demographic factors were the principal correlate of poor sleep quality.

The strengths of this study include the use of validated Turkish versions of all three instruments, assessment of the complete SF-36 subscale panel rather than a subset, and the use of multivariable rather than univariable analysis to identify independent predictors. Several limitations should nonetheless be considered. The cross-sectional design precludes causal inference. All three primary outcome measures relied on self-report, without corroborating objective sleep assessments such as actigraphy or polysomnography. This was a single-center study, which may limit generalizability. Fasting glucose was excluded from multivariable analyses due to collinearity with HbA1c, and other potentially relevant variables, including diabetes-related complications, specific antidiabetic medication regimens, anxiety symptoms, and diabetes-specific distress, were not assessed. Finally, although the sample size was adequate for the primary correlation analysis, statistical power to detect smaller effects among covariates in the multivariable models may have been limited.

Conclusion

Poor sleep quality was common in this cohort of patients with T2DM and was independently associated with depressive symptom severity rather than glycemic control after adjustment for demographic and metabolic factors. Poor sleep quality was also associated with substantially impaired HRQoL across all SF-36 domains. These findings suggest that depressive symptoms may represent a more clinically actionable target than glycemic control alone for improving sleep quality in this population and support the routine screening for depressive symptoms and sleep disturbance in the clinical management of T2DM.

Disclosures

Ethics Committee Approval: The study was approved by the Basaksehir Cam and Sakura City Hospital Scientific Research 2nd Ethics Committee (no: 315, date: 03.06.2026).

Informed Consent: Written informed consent were obtained from all participants prior to study enrollment.

Conflicts of Interest: The authors declared no conflict of interest.

Funding: The authors declared that this research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Use of AI for Writing Assistance: The authors declared that artificial intelligence-assisted language editing was used to improve grammar and readability. The authors reviewed and approved the final manuscript and take full responsibility for its content.

Authorship Contributions: Concept – MKY, EH; Design – MKY, EH; Supervision – EH; Materials – MKY; Data Collection or Processing: MKY; Analysis or Interpretation: MKY; Literature Search: MKY; Writing: MKY; Critical Review: EH.

Peer-review: Externally peer-reviewed.

References

1. International Diabetes Federation. IDF Diabetes Atlas, 10th ed. Brussels: International Diabetes Federation; 2021.
2. Kurnia AD, Thato R, Tsai HT. Predicting factors of sleep quality among adults with type 2 diabetes mellitus: A systematic review. *Diabetes Res Clin Pract* 2025;227:112388. [\[CrossRef\]](#)
3. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989;28(2):193–213. [\[CrossRef\]](#)
4. Ağargün MY, Kara H, Anlar Ö. The validity and reliability of the Pittsburgh Sleep Quality Index. [Article in Turkish]. *Türk Psikiyatri Derg* 1996;7:107–15.
5. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. *Diabetes Care* 2010;33(2):414–20. [\[CrossRef\]](#)

6. Schipper SBJ, Van Veen MM, Elders PJM, van Straten A, Van Der Werf YD, Knutson KL, et al. Sleep disorders in people with type 2 diabetes and associated health outcomes: a review of the literature. *Diabetologia* 2021;64(11):2367–77. [CrossRef]
7. Khandelwal D, Dutta D, Chittawar S, Kalra S. Sleep disorders in type 2 diabetes. *Indian J Endocrinol Metab* 2017;21(5):758–61. [CrossRef]
8. Darraj A. The link between sleeping and type 2 diabetes: a systematic review. *Cureus* 2023;15(11):e48228. [CrossRef]
9. World Health Organization. Depressive disorder (depression). Geneva: WHO; 2023. Available at: <https://www.who.int/news-room/fact-sheets/detail/depression>. Accessed October 12, 2026.
10. Khaledi M, Haghighatdoost F, Feizi A, Aminorroaya A. The prevalence of comorbid depression in patients with type 2 diabetes: an updated systematic review and meta-analysis on huge number of observational studies. *Acta Diabetol* 2019;56(6):631–50. [CrossRef]
11. Alzahrani MS, Alabbasi A, Alzahrani Z, Alsafrani T. Prevalence and patterns of psychiatric comorbidities in type 2 diabetes mellitus: a systematic review and meta-analysis. *Front Public Health* 2026;14:1697079. [CrossRef]
12. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch Gen Psychiatry* 1961;4:561–71. [CrossRef]
13. Hisli N. A study on the validity of Beck Depression Inventory. [Article in Turkish]. *Psikoloji Derg* 1988;6(22):118–22.
14. Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med Care* 1992;30(6):473–83. [CrossRef]
15. Laverty B, Puthethath Jayanandan S, Smyth S. Understanding the relationship between sleep and quality of life in type 2 diabetes: A systematic review of the literature. *J Health Psychol* 2023;28(8):693–710. [CrossRef]
16. Koçyiğit H, Aydemir Ö, Fişek G, Ölmez N, Memiş A. Kısa form-36 (KF36)'nın Türkçe versiyonunun güvenilirliği ve geçerliliği. *İlaç ve Tedavi Derg* 1999;12(2):102–6.
17. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet* 1999;354(9188):1435–9. [CrossRef]
18. Beck AT, Steer RA, Garbin MC. Psychometric properties of the Beck Depression Inventory. Twenty-five years of evaluation. *Clin Psychol Rev* 1988;8(1):77–100. [CrossRef]
19. Liu Y, Huang SY, Liu DL, Zeng XX, Pan XR, Peng J. Bidirectional relationship between diabetes mellitus and depression: Mechanisms and epidemiology. *World J Psychiatry* 2024;14(10):1429–36. [CrossRef]
20. Knutson KL, Spiegel K, Penev P, Van Cauter E. The metabolic consequences of sleep deprivation. *Sleep Med Rev* 2007;11(3):163–78. [CrossRef]