

Essentia:
Journal of Medical Practice and Research
Vol 2 No 2 December 2026, Hal 20-30
ISSN: 3123-4100(Print) ISSN: 3123-4097(Electronic
Open Access: https://scriptaintelektual.com/essentia

Sleep Duration and Glycemic Status Among Adults: Evidence From NHANES Data

Alfie C. Aure[1*], Mazou Issa[2], King Arnold C. Satsatin[3]
[1,3] University of Cabuyao
[2] University of Maroua
email: aurealfie20045@gmail.com[1]

Article Info :
Received:
14-10-2026
Revised:
05-11-2026
Accepted:
19-11-2026

Abstract

Sleep duration has been linked to glycemic regulation, but associations with HbA1c remain inconsistent. This secondary cross-sectional analysis examined weekday sleep and laboratory HbA1c in 134 complete observations from an NHANES-derived held-out test subset previously balanced for cardiovascular disease prediction. The preprocessed subset is not nationally representative. Sleep was examined continuously and in short (<7 h), recommended (7–9 h), and long (>9 h) categories using correlations, HC3 robust regression, nonlinear models, and exclusion-based sensitivity analyses. Mean weekday sleep was 7.69 h and mean HbA1c was 5.92%. Spearman correlation was nonsignificant (rho=0.056, p=0.517), whereas Pearson correlation was weakly positive (r=0.264, p=0.002). The fully adjusted sleep coefficient was positive but nonsignificant (B=0.192 HbA1c percentage points/h, 95% CI −0.012 to 0.396; p=0.066). HbA1c distributions did not differ significantly by sleep category, and quadratic findings were sensitive to extreme-value exclusions. Among 26 participants with diagnosed diabetes, a positive sleep coefficient was reported (B=0.631, 95% CI 0.449–0.813; p<0.001), but this exploratory subgroup estimate does not establish effect modification or causality. The small, cardiovascular-disease-balanced subset limits generalizability. Representative, design-weighted longitudinal studies are needed to validate these findings.

Keywords: *Diabetes, HbA1c, NHANES, Sleep Duration, Glycemic Status.*

©2026 Authors.. This work is licensed under a Creative Commons Attribution-Non Commercial 4.0 International License.
(https://creativecommons.org/licenses/by-nc/4.0/)

INTRODUCTION

Sleep has moved from a peripheral lifestyle consideration to a formal component of contemporary diabetes care, reflecting growing recognition that sleep quantity, quality, timing, and regularity are closely intertwined with metabolic regulation and the capacity of individuals to maintain effective daily disease-management behaviors. Current diabetes standards position adequate sleep alongside nutrition, physical activity, tobacco avoidance, self-management education, and psychosocial care, indicating that sleep is increasingly regarded as a modifiable behavioral determinant within comprehensive diabetes management rather than merely a marker of lifestyle preference (American Diabetes Association Professional Practice Committee for Diabetes, 2026a). Glycated hemoglobin (HbA1c) remains a central indicator of chronic glycemic exposure, while its interpretation requires consideration of the broader clinical context, including diabetes status, obesity, medication use, and other metabolic characteristics (American Diabetes Association Professional Practice Committee, 2025b). Evidence synthesized among adults with diabetes indicates that sleep disturbances are common and that their relationships with glycemic outcomes vary according to the sleep dimension examined and the clinical characteristics of the population studied (Fentahun et al., 2024). Sleep therefore occupies an increasingly important position at the intersection of behavioral medicine, metabolic physiology, and chronic disease management. The central issue is no longer whether sleep matters for metabolic health, but rather how specific dimensions of sleep behavior relate to measurable glycemic status across heterogeneous adult populations.

Population research does not consistently indicate a linear association between habitual sleep duration and metabolic risk. The Maastricht Study observed associations of both short and long sleep with type 2 diabetes after adjustment for conventional lifestyle factors (Albers et al., 2023). Longitudinal research has linked sleep duration with diabetes incidence, but estimates vary across

populations and exposure definitions (Jang et al., 2023). Irregular sleep duration may carry information not captured by mean duration alone (Kianersi et al., 2024). Among individuals with established diabetes, prolonged sleep can also mark underlying illness or functional limitations, raising the possibility of reverse causation (Gu et al., 2022). These findings motivate separate consideration of the association's direction, functional form, and sensitivity to sample composition rather than presuming that longer sleep directly worsens glycemia.

Existing national-survey analyses have considered sleep and diabetes-related outcomes, including NHANES studies linking glycemic status to sleep measures (Basiri et al., 2025; Zhang et al., 2025). Evidence on weekend catch-up sleep also varies by baseline sleep duration and glycemic status (Kim et al., 2024; Wang et al., 2025). These studies establish an important background but do not necessarily show whether an HbA1c association remains consistent across Pearson and rank correlations, heteroskedasticity-robust regression, nonlinear specifications, and exclusions of extreme observations in the same processed dataset. The present investigation therefore addresses an analytical consistency question rather than claiming to be the first national study of sleep and glucose regulation. Its value is limited to identifying which associations are supported or fragile within a deliberately nonrepresentative NHANES-derived test subset.

Sleep and glucose regulation may be connected through circadian alignment, neuroendocrine responses, insulin sensitivity, and daily behaviors (Henson et al., 2024). Conversely, diabetes-related symptoms and sleep-disordered breathing may disrupt habitual sleep, creating potential bidirectionality (Mao, 2024). Associations between sleep characteristics and glycemic control have varied across clinical populations (Borzouei et al., 2024). Intervention findings have also been inconsistent, and protocols examining sleep timing illustrate the need to distinguish observational relationships from effects of modifying sleep (Slebe et al., 2025; Bouman et al., 2024). These mechanisms provide a rationale for studying the association, but the present cross-sectional secondary analysis cannot identify their operation or establish temporal or causal direction.

Comparability between studies is constrained by differences in sleep measurement, glycemic outcomes, adjustment sets, and patient characteristics. Reviews of diabetes populations have reported inconsistent associations across sleep quality, duration, and glycemic variability (Pham et al., 2025). Studies of people with prediabetes likewise suggest that HbA1c and sleep characteristics may covary, without resolving confounding (Kass et al., 2022). Sleep duration alone does not encompass sleep quality, timing, or regularity (Darraj, 2023), and nonlinear models can be sensitive to extreme values (Kuroda et al., 2025). An additional limitation arises when publicly released NHANES records have been resampled for another prediction task: findings from that altered distribution cannot be treated as national estimates. Evaluating the stability of estimates within such a subset is useful as a methodological stress test, not as a substitute for a design-weighted population analysis.

This study uses the independent held-out test partition of an NHANES-derived dataset assembled for cardiovascular disease prediction from August 2021–August 2023 variables and balanced by random undersampling before an 80:20 stratified split (Akhter & Miller, 2026). The partition was chosen because it was separately released and had not been used for feature selection in the source predictive project; this does not eliminate bias introduced before the split. The primary objective is to examine the association of weekday sleep duration with continuous HbA1c after adjustment for available demographic and metabolic variables. Secondary objectives are to compare categorical and quadratic specifications, alternative weekly sleep metrics, and exclusion-based sensitivity analyses, while describing an exploratory diagnosed-diabetes subgroup. In contrast to representative NHANES analyses, the intended contribution is a transparent assessment of how conclusions change with model assumptions and influential observations. All inferences are restricted to the 134-person processed analytic subset, and no population prevalence, causal effect, or confirmed diabetes-specific modification is claimed.

RESEARCH METHODS

This cross-sectional secondary analysis used deidentified individual-level records from the independently released test partition of Akhter and Miller (2026). The original source comprised 670 NHANES-derived records that were balanced by random undersampling for cardiovascular disease classification and split 80:20 into training and testing partitions. The entire released test partition (n=134) was used to avoid reusing observations from the source project's feature-selection stage; it was

not selected through a new probability-sampling procedure for the present HbA1c question. Cardiovascular-disease balancing preceded the split and could change the joint distribution of diabetes, sleep, age, and other characteristics, generating selection bias even in a held-out partition. Eligibility required available weekday sleep duration and laboratory HbA1c; 134 complete observations were reported for the primary analysis. The pre-release number and characteristics of missing records, if any, are not documented in the present analytic materials, so the original missingness mechanism and effects of any upstream exclusions cannot be assessed. The exposure was weekday/workday sleep (SLD012), categorized as short (<7 h), recommended (7–9 h), or long (>9 h). Weekend/non-workday sleep (SLD013) defined the weighted weekly average and weekend-minus-weekday difference. The outcome was laboratory HbA1c (LBXGH, %); HbA1c ≥6.5% was described without replacing reported diabetes status (American Diabetes Association Professional Practice Committee, 2025a).

Recorded covariates were age, sex, BMI, self-reported diagnosed diabetes (including the available borderline category), family income-to-poverty ratio, and ever smoking at least 100 cigarettes. These characteristics were considered potential demographic, clinical, socioeconomic, or behavioral correlates of both sleep and glycemia; they were not used to establish causal identification. Descriptive measures comprised means (SD), medians (IQR), and counts (%). Differences in HbA1c distributions among sleep groups were evaluated with the Kruskal–Wallis test; Spearman and Pearson correlations described unadjusted relationships. Ordinary least-squares models used HC3 heteroskedasticity-consistent standard errors: Model 1 included weekday sleep; Model 2 added age, sex, BMI, and diabetes status; Model 3 additionally included income-to-poverty ratio and smoking. A centered quadratic sleep specification, weighted weekly sleep, weekend-minus-weekday difference, exclusion of sleep <4 or >11 h, exclusion of HbA1c >10%, and diagnosed-diabetes-only analyses were also reported. Two-sided $p<0.05$ defined conventional statistical significance. Analyses were reported as performed in Python 3 using pandas 2.2.3, SciPy 1.17.0, and statsmodels 0.14.6. No new recruitment, intervention, or identifiable data collection occurred; ethical approval and consent are attributed to the source NHANES process rather than newly claimed for this analysis.

Statistical interpretation was restricted by the small analytic sample, marked imbalance between sleep categories (29, 94, and 11 participants), and possible selection induced by the original cardiovascular-disease balancing. HC3 standard errors address heteroskedasticity in inference but do not establish the absence of leverage, nonlinearity misspecification, or multicollinearity. Formal residual plots, influence measures, variance-inflation factors, and a priori power calculations were not available in the supplied analytic results; none is claimed to have been performed. The subgroup coefficient for 26 participants with diagnosed diabetes is reproduced as reported, but its complete covariate specification cannot be confirmed from Table 4 and it is not labelled a fully adjusted or causal estimate. No pooled sleep-by-diabetes interaction test was reported, so subgroup differences are not interpreted as demonstrated effect modification. Diabetes medication, disease duration, sleep apnea, and other potential confounders were not included in the reported models. As the release lacks NHANES strata, clusters, and survey weights, neither nationally representative inference nor valid population prevalence estimation was attempted (Akhter & Miller, 2026).

RESULTS AND DISCUSSION

Participant Characteristics and Sleep-Duration Patterns

The analytic sample included 134 participants in the cardiovascular-disease-balanced held-out partition, not a nationally representative NHANES sample. Mean age was 60.62 years (SD 15.91), 58 participants (43.3%) were women, and mean BMI was 29.43 kg/m² (SD 6.35). Diagnosed diabetes was reported by 26 participants (19.4%); 21 (15.7%) had HbA1c ≥6.5%. These percentages describe only the selected records and must not be interpreted as population prevalences. Age, adiposity, diabetes status, and unmeasured treatment factors may jointly relate to both sleep and glycemia, limiting causal interpretation. Table 1 presents the observed sample profile without implying statistical differences between groups.

Table 1. Characteristics of the Analytic Population by Weekday Sleep Duration

Characteristic	Overall	Short (<7 h)	Recommended (7-9 h)	Long (>9 h)
Age, years	60.62 (15.91)	59.31 (15.53)	60.88 (15.84)	61.82 (18.72)
Female sex, n (%)	58 (43.3)	8 (27.6)	45 (47.9)	5 (45.5)
BMI, kg/m2	29.43 (6.35)	29.13 (6.34)	29.39 (6.34)	30.55 (6.93)
Weekday sleep, h	7.69 (1.40)	5.81 (1.00)	7.98 (0.68)	10.18 (0.98)
Weekend sleep, h	8.24 (1.67)	6.72 (2.22)	8.44 (0.97)	10.45 (1.44)
HbA1c, %	5.92 (1.09)	5.76 (0.63)	5.85 (0.90)	6.93 (2.44)
Diagnosed diabetes, n (%)	26 (19.4)	6 (20.7)	18 (19.1)	2 (18.2)
HbA1c >=6.5%, n (%)	21 (15.7)	5 (17.2)	13 (13.8)	3 (27.3)
Ever smoked >=100 cigarettes, n (%)	76 (56.7)	17 (58.6)	53 (56.4)	6 (54.5)

Mean weekday sleep duration was 7.69 hours (SD 1.40), compared with 8.24 hours (SD 1.67) on weekends. There were 94 participants (70.1%) in the 7–9-hour category, 29 (21.6%) below 7 hours, and only 11 (8.2%) above 9 hours. The unequal category sizes limit the precision of comparisons, particularly any summary for long sleepers. One observation in the long-sleep group represents 9.1% of that group, so unusual glycemic values can markedly affect its mean and percentage estimates. Previous studies have examined associations of both short and long sleep with diabetes, but those findings cannot supply statistical support for differences in this selected dataset (Albers et al., 2023). The categories are therefore treated as descriptive strata, not evidence of distinct metabolic risk profiles.

Age and BMI means were numerically similar across the three sleep groups, with the highest mean BMI of 30.55 kg/m² among the 11 long sleepers. The proportions of women were 27.6%, 47.9%, and 45.5% in the short-, recommended-, and long-sleep groups, respectively. Reported diagnosed diabetes varied from 18.2% to 20.7%. These values were not established as between-group differences by inferential testing and should not be used to attribute glycemic contrasts to particular characteristics. Diabetes severity, duration, glucose-lowering medication, sleep apnea, and other unmeasured conditions could vary even where reported diagnosis rates appear similar. Physiologic pathways connecting sleep and glycemia support evaluating multiple covariates but do not exclude residual confounding (Henson et al., 2024).

Weekend sleep followed a similar ordering to weekday sleep, increasing from 6.72 hours among short sleepers to 8.44 hours among participants with 7–9 hours of weekday sleep and 10.45 hours among long sleepers. This parallel pattern indicates that participants classified according to weekday sleep duration also differed systematically in their non-workday sleep behavior, rather than displaying isolated differences confined to workdays. The mean difference between weekend and weekday sleep was approximately 0.91 hours among short sleepers, 0.46 hours among recommended-range sleepers, and 0.27 hours among long sleepers, suggesting that shorter weekday sleep was accompanied by greater relative weekend compensation. Such variation is relevant because weekday sleep duration and weekend catch-up sleep may represent related but distinct behavioral exposures. Recent population-based evidence indicates that the metabolic implications of weekend catch-up sleep may depend on the amount of compensation and the underlying weekday sleep pattern rather than on weekend duration alone (Kim et al., 2024). The observed sleep pattern therefore supports the inclusion of weighted weekly sleep and weekend-minus-weekday sleep difference as secondary exposure specifications rather than relying exclusively on weekday duration.

Mean HbA1c was 5.76%, 5.85%, and 6.93% in the short-, recommended-, and long-sleep categories. The long-sleep median, however, was 5.80% (IQR 5.45–7.60), compared with 5.60% (IQR 5.30–6.07) in the recommended group, showing that the larger mean did not correspond to an equally large shift in the median. HbA1c ≥6.5% occurred in 17.2%, 13.8%, and 27.3% of these categories, respectively. Because the final percentage represents only 3 of 11 people, it is especially unstable. Measures of HbA1c, glycemic variability, and reported diagnosis capture different clinical dimensions and may yield different patterns (Pham et al., 2025). No statistically significant group difference should be inferred from these descriptive contrasts alone.

The selected records include variation in sleep measures, BMI, and HbA1c, but the 7–9-hour category dominates the sample. The higher mean and modestly different median among long sleepers raise the possibility of influential HbA1c observations rather than demonstrating a uniform metabolic disadvantage. Published nonlinear sleep–metabolic associations motivate testing model form, yet they cannot validate a curve observed in only 11 long sleepers (Kuroda et al., 2025). Descriptive results are therefore separated from the nonparametric comparison and robust regression analyses that follow. Any full-sample inference remains conditional on the cardiovascular-disease-balanced sampling frame and available confounders.

Glycemic Status Across Weekday Sleep-Duration Categories

The overall mean HbA1c was 5.92% (SD 1.09). Group means were 5.76%, 5.85%, and 6.93% for short, recommended, and long weekday sleep, respectively, but the corresponding SDs were 0.63%, 0.90%, and 2.44%. The greater dispersion in the 11-person long-sleep category makes its mean vulnerable to high individual readings. These values are descriptive summaries of an unevenly distributed, preprocessed sample, not estimates of clinically meaningful group differences. Evidence from other observational populations cannot resolve whether the pattern is reproducible here (Albers et al., 2023; Mou et al., 2026).

HbA1c medians were 5.70%, 5.60%, and 5.80% for short, recommended, and long weekday sleep. Their proximity contrasts with the separation of group means and indicates that a few high values may contribute disproportionately to the long-sleep mean. Without an influence analysis of individual observations, the exact contribution of any participant cannot be quantified. Table 2 reports both mean and median summaries so that the nonsignificant inferential comparison can be interpreted against this dispersion.

Table 2. Glycemic Status Across Weekday Sleep-Duration Categories

Sleep Category	n	HbA1c Mean (SD)	HbA1c Median (IQR)	HbA1c >=6.5%, n (%)	Diagnosed Diabetes, n (%)
Short (<7h)	29	5.76 (0.63)	5.70 (5.30-5.90)	5 (17.2)	6 (20.7)
Recommended (7-9h)	94	5.85 (0.90)	5.60 (5.30-6.07)	13 (13.8)	18 (19.1)
Long (>9h)	11	6.93 (2.44)	5.80 (5.45-7.60)	3 (27.3)	2 (18.2)

Note: Values are based on the unweighted held-out analytic sample. Continuous variables are mean (SD) unless otherwise specified.

HbA1c ≥6.5% was observed in 5 of 29 short sleepers (17.2%), 13 of 94 recommended-range sleepers (13.8%), and 3 of 11 long sleepers (27.3%). The higher percentage in the last category reflects just three observations and carries substantial sampling uncertainty. Reported diabetes prevalence was 20.7%, 19.1%, and 18.2% in the same categories. Neither the threshold percentages nor diagnosed diabetes frequencies demonstrate a between-group difference on their own. HbA1c and self-reported diagnosis are distinct constructs, and medication exposure or disease history could modify their correspondence (American Diabetes Association Professional Practice Committee, 2025a).

The Kruskal–Wallis comparison did not establish a difference in HbA1c distributions across weekday sleep categories ($H=1.22$, $p=0.543$). This nonsignificant finding is the principal inferential result of the categorical comparison. The long-sleep mean is numerically higher, but its SD and limited group size prevent a reliable conclusion about greater glycemic burden. A nonsignificant test does not prove identical distributions; power to detect modest or heterogeneous differences may be limited by the 29/94/11 group imbalance. The results should therefore be described as inconclusive for a categorical association, not as confirmation of elevated HbA1c in long sleepers. Clinical pathways suggested in earlier studies remain hypotheses rather than effects demonstrated here (Henson et al., 2024).

Self-reported diagnosed diabetes occurred in 18.2% of long sleepers, 19.1% of recommended-range sleepers, and 20.7% of short sleepers. These similar percentages do not explain the dispersion of long-sleep HbA1c, but neither do they exclude differences in diabetes duration, treatment, control, or other clinical features. The percentages above the HbA1c diagnostic threshold were also based on small

category counts. Clinical interpretation of HbA1c requires context, and cross-sectional comparisons cannot determine whether sleep preceded dysglycemia (American Diabetes Association Professional Practice Committee, 2025b). Studies relating sleep to diabetes control provide background plausibility without validating a category-specific effect in these data (Borzouei et al., 2024).

Taken together, the unadjusted categorical results provide no statistically significant evidence of different HbA1c distributions by sleep duration. The difference between means and medians, especially in the 11-person long-sleep group, suggests potential outlier sensitivity without identifying a particular observation as influential. The unequal category sizes and the source dataset's cardiovascular-disease balancing further reduce precision and external validity. Nonlinear associations reported elsewhere warrant exploration, not the assumption that this study has confirmed a nonlinear dose-response relationship (Kuroda et al., 2025). Regression and exclusion analyses are consequently treated as complementary assessments of model dependence rather than definitive tests of a causal sleep effect.

Association Between Sleep Duration and HbA1c

Spearman correlation was near zero and nonsignificant (rho=0.056, p=0.517), whereas Pearson correlation was positive (r=0.264, p=0.002). Pearson correlation reflects ordinary linear covariance and its conventional significance test, whereas Spearman correlation evaluates rank ordering; these need not agree when high HbA1c observations exert leverage or the relationship is nonmonotonic. The discrepancy therefore raises a concern about distributional shape and influential values, not proof of a reproducible sleep–glycemia effect. The robust regression estimates reported below additionally use HC3 standard errors, which allow a different uncertainty assessment from the usual Pearson test. Covariate adjustment and the change in variance estimation must both be considered when interpreting the different p values. Nonlinear results in other populations motivate sensitivity checks rather than establishing the form of this association (Kuroda et al., 2025).

In Model 1, the unadjusted HC3 regression estimated a 0.206 percentage-point increase in HbA1c per additional weekday sleep hour, but the interval included no association (95% CI −0.030 to 0.442; p=0.087). Model 2, adjusted for age, sex, BMI, and diabetes status, estimated B=0.191 (95% CI −0.017 to 0.400; p=0.072). The fully adjusted Model 3, additionally accounting for income-to-poverty ratio and ever smoking, estimated B=0.192 (95% CI −0.012 to 0.396; p=0.066). Thus, an extra hour of sleep was associated with an estimated 0.192 HbA1c percentage points in this sample, but the confidence interval remains compatible with a very small negative or a larger positive association. Similar point estimates do not overcome the absence of a statistically established primary adjusted association. Table 3 reports the available selected coefficients; it should not be interpreted as a complete coefficient-by-coefficient diagnostic report.

Table 3. Association Between Weekday Sleep Duration and HbA1c

Model	Predictor	B	95% CI	p	R2
Model 1 unadjusted	Weekday sleep, per h	0.206	-0.030 to 0.442	.087	.070
Model 2 adjusted	Weekday sleep per h	.191	-.017 to .400	.072	.430
Model 2 adjusted	Diagnosed diabetes yes vs no	1.517	1.023 to 2.011	<.001	.430
Model 2 adjusted	Borderline diabetes vs no	.349	-.163 to .861	.182	.430
Model 3 fully adjusted	Weekday sleep per h	.192	-.012 to .396	.066	.452
Model 3 fully adjusted	Diagnosed diabetes yes vs no	1.434	.925 to 1.944	<.001	.452
Model 3 fully adjusted	Borderline diabetes vs no	.203	-.303 to .708	.432	.452
Model 3 fully adjusted	Income-to-poverty ratio	-.044	-.129 to .042	.318	.452
Model 3 fully adjusted	Ever smoked	.294	-.081 to .669	.124	.452
Model 4 nonlinear	Sleep-8 h	.277	.114 to .440	<.001	.518
Model 4 nonlinear	(Sleep-8 h)^2	.077	.040 to .113	<.001	.518
Model 4 nonlinear	Diagnosed diabetes yes vs no	1.478	1.044 to 1.913	<.001	.518

The diagnosed-diabetes coefficients were B=1.517 in Model 2 and B=1.434 in Model 3 (both p<0.001), consistent with higher HbA1c among participants reporting diabetes. The income-to-poverty ratio (p=0.318) and ever-smoking indicator (p=0.124) did not reach conventional significance in Model 3. These covariates were included because socioeconomic and smoking-related differences may jointly

affect sleep and metabolic outcomes; a nonsignificant individual coefficient does not rule out confounding (Darraj, 2023). Medication use, diabetes duration, obstructive sleep apnea, sleep quality, and other factors were not available in the presented adjusted specification. Accordingly, the sleep coefficient is an observational conditional association, not evidence of an independent biological effect.

The exploratory quadratic specification centered sleep at 8 hours. Its linear term was B=0.277 (95% CI 0.114 to 0.440; p<0.001) and its squared term was B=0.077 (95% CI 0.040 to 0.113; p<0.001). The reported model R^2 was 0.518 versus 0.452 in the fully adjusted linear model. Such an in-sample increase is not sufficient to establish a stable nonlinear pattern, particularly when functional form is tested in a small, unevenly distributed sample. The table does not display every covariate or diagnostic for the quadratic model, so the comparison cannot be treated as an independently validated model-selection result. The apparently significant curvature was subsequently weakened when extreme sleep or HbA1c observations were excluded (Table 4). Previous reports of nonlinear associations justify exploring this form but do not rescue its sensitivity in the present sample (Kuroda et al., 2025).

The direction of the linear estimates can be compared cautiously with research relating prolonged sleep to diabetes risk, although incidence of diabetes and continuous HbA1c are different endpoints (Albers et al., 2023; Liu et al., 2022). Prior NHANES-based analyses have also examined sleep characteristics and diabetes outcomes, so this processed test-set analysis does not claim novelty as a population study (Zhang et al., 2025). Instead, its contribution is to show that a statistically significant Pearson correlation did not persist as a conventional significant result under HC3 regression, and that a quadratic signal failed key exclusion checks. The findings cannot distinguish whether sleep duration affects glycemic regulation or whether health status influences sleep. No clinically actionable threshold or direction of effect can be inferred from the present results.

The evidence for an overall association is inconsistent across analytical specifications: Pearson's p value was significant, but Spearman's correlation and HC3 linear models were not. High-leverage observations, skewed HbA1c values, and heteroskedastic errors are plausible explanations, yet formal leverage, residual, and multicollinearity diagnostic statistics were not supplied in the analytic outputs. HC3 standard errors reduce reliance on constant error variance assumptions but neither remove leverage nor establish correct model specification. The small sample also limits precision for models with multiple covariates. Consequently, the observed linear and quadratic patterns are treated as uncertain, not as proof of an independent sleep effect. Evidence from sleep research underscores the need for richer measurements of duration, timing, and quality in future analysis (Henson et al., 2024; Bouman et al., 2024).

Sensitivity Analyses and Diabetes-Specific Association

Sensitivity analyses assessed whether the main interpretations depended on sleep measurement or extreme observations. Weighted weekly sleep had B=0.208 (95% CI −0.021 to 0.437; p=0.076), while weekend-minus-weekday sleep difference had B=0.024 (95% CI −0.077 to 0.124; p=0.645); neither reached conventional significance. After excluding sleep below 4 hours or above 11 hours (n=131), the quadratic coefficient was B=0.079 (95% CI −0.084 to 0.242; p=0.343). Excluding HbA1c >10% (n=132) yielded B=0.042 (95% CI −0.005 to 0.089; p=0.081). These changes show that the apparently significant in-sample curvature was not robust to specified exclusions, making any nonlinear interpretation particularly tentative. Table 4 presents these reported checks and the exploratory diabetes-only estimates. The alternative sleep results also do not establish an independent association for weekend compensation; different sleep metrics may reflect distinct behaviors (Fan et al., 2026; Mou et al., 2026).

Table 4. Sensitivity Analyses and Diabetes-Specific Association

Analysis	n	Exposure	B	95% CI	p
Weekly weighted average sleep	134	sleep_avg	.208	-.021 to .437	.076
Weekend-minus-weekday sleep difference	134	catchup	.024	-.077 to .124	.645
Quadratic model after excluding sleep <4 or >11h	131	(Sleep-8h)^2	.079	-.084 to .242	.343
Quadratic after excluding HbA1c >10%	132	(Sleep-8h)^2	.042	-.005 to .089	.081
Diagnosed-diabetes subgroup	26	Weekday sleep	.631	.449 to .813	<.001

Diabetes subgroup excluding >9h sleepers	24	Weekday sleep	.547	.154 to .941	.006
Diabetes subgroup excluding HbA1c >10%	25	Weekday sleep	.629	.261 to .996	<.001

Among the 26 participants reporting diagnosed diabetes, the reported weekday-sleep coefficient was B=0.631 HbA1c percentage points per hour (95% CI 0.449 to 0.813; p<0.001). The estimate remained positive after excluding the two participants sleeping more than 9 hours (n=24; B=0.547, 95% CI 0.154 to 0.941; p=0.006) and after excluding HbA1c >10% (n=25; B=0.629, 95% CI 0.261 to 0.996; p<0.001). These exclusions are useful descriptive robustness checks, but do not assess sensitivity to different covariate sets or unmeasured treatment patterns. The covariate specification for these subgroup coefficients is not provided in the available table; the estimates should therefore not be described as fully adjusted effects. With only 26 observations, even apparently narrow intervals can understate model uncertainty if assumptions, leverage, or covariate selection are unstable. Prior literature provides a clinical rationale for continued research, not confirmation of the subgroup association (Shibabaw et al., 2023; Pham et al., 2025).

The subgroup point estimate (0.631) exceeds the full-sample adjusted estimate (0.192) in numerical magnitude. Comparing separate coefficients, however, does not establish that diabetes status modifies the sleep–HbA1c association. A pooled regression including a sleep-by-diabetes interaction, with its coefficient and confidence interval, would be needed to evaluate a statistical difference in slopes; such a test is not reported in the available analysis. The subgroup sample is also too small to support a confident interpretation of multiple-covariate specifications or to rule out overfitting and residual confounding. Diabetes medication, disease duration, symptoms, and altered sleep architecture are potential alternative explanations (Henson et al., 2024; Mao, 2024). The result is consequently a hypothesis-generating within-diabetes association, not evidence of confirmed effect modification or a causal impact of longer sleep.

Weekend-minus-weekday sleep difference was not associated with HbA1c in the reported full-sample model (B=0.024, p=0.645). This is a lack of statistically supported association in this sample, not evidence that weekend sleep compensation is biologically irrelevant. Other populations have yielded different findings for weekend catch-up sleep and glycemic outcomes (Kim et al., 2024; Wang et al., 2025). The present measure also cannot represent sleep regularity, timing, or subjective quality, which may be independently informative (Kianersi et al., 2024). Sample selection and limited precision further constrain comparisons. No clinical recommendation concerning catch-up sleep follows from these analyses.

The diabetes-only results have the largest reported sleep coefficient but remain exploratory. Although exclusion of selected extreme observations retained positive coefficients, the subgroup comprised just 26 participants and the adjustment set was not fully documented in the available output. Without a formal sleep-by-diabetes interaction test, differences between subgroup and full-sample coefficients cannot be called effect modification. Diabetes treatment, duration, comorbidities, and reversed temporal direction may also explain part of the observed pattern. Intervention and review evidence indicates that sleep is relevant to metabolic health, but it does not imply that longer sleep caused higher HbA1c in these records (Nakada et al., 2026; Slebe et al., 2025). The nonrepresentative cardiovascular-disease-balanced selection further limits generalizability (Akhter & Miller, 2026). Replication in a larger, survey-weighted or prospective sample with richer clinical information is needed.

CONCLUSION

The primary result did not establish a statistically significant adjusted association between weekday sleep duration and HbA1c in the 134-person processed held-out sample (Model 3: B=0.192, 95% CI −0.012 to 0.396; p=0.066). HbA1c distributions did not differ significantly across short, recommended, and long sleep categories (Kruskal–Wallis p=0.543). The higher descriptive mean among 11 long sleepers was imprecise and potentially sensitive to elevated individual HbA1c values. A significant in-sample quadratic term did not remain significant after the reported extreme-value exclusions. Weighted weekly sleep and weekend sleep difference also showed no significant associations. The methodological contribution is a within-dataset comparison demonstrating that conclusions depend on statistical specification and influential observations, not a nationally

representative estimate of sleep-related glycemic risk. No causal or independent clinical effect of sleep duration can be inferred.

The diabetes-only analysis yielded a larger positive coefficient (B=0.631; n=26), but this finding is exploratory because the subgroup was small, the reported adjustment specification was incomplete, and no formal interaction test established a difference from the other participants. Random undersampling for cardiovascular-disease prediction and the absence of NHANES survey weights limit external validity throughout the manuscript. Missing pre-release data and unavailable measures of diabetes medication, disease duration, sleep apnea, sleep timing, and sleep quality further restrict interpretation. Future studies should reanalyze complete probability-sampled NHANES data with appropriate design weights, transparent missing-data handling, complete model and influence diagnostics, and prespecified interaction analyses. Larger longitudinal cohorts with clinical treatment data are required to establish temporal sequence and test reproducibility. Until such confirmation, these results are best treated as exploratory associations rather than evidence for changing diabetes care based on sleep duration alone.

REFERENCES

Akhter, S., & Miller, J. H. (2026). Web-based cardiovascular disease risk prediction using machine learning. *Frontiers in Artificial Intelligence, 9*, 1690664. https://doi.org/10.3389/frai.2026.1690664

Albers, J. D., Meertens, R. M., Savelberg, H. H. C. M., Kohler, S., Wesselius, A., Schram, M. T., Stehouwer, C. D. A., de Galan, B. E., van Greevenbroek, M. M. J., van der Kallen, C. J. H., Eussen, S. J. P. M., Bosma, H., Schaper, N. C., & Koster, A. (2023). Both short and long sleep durations are associated with type 2 diabetes, independent from traditional lifestyle risk factors—The Maastricht Study. *Sleep Health, 9*(5), 733–741. https://doi.org/10.1016/j.sleh.2023.06.009

American Diabetes Association Professional Practice Committee. (2025a). 2. Diagnosis and classification of diabetes: Standards of care in diabetes—2025. *Diabetes Care, 48*(Suppl. 1), S27–S49. https://doi.org/10.2337/dc25-S002

American Diabetes Association Professional Practice Committee. (2025b). 6. Glycemic goals and hypoglycemia: Standards of care in diabetes—2025. *Diabetes Care, 48*(Suppl. 1), S128–S145. https://doi.org/10.2337/dc25-S006

American Diabetes Association Professional Practice Committee for Diabetes. (2026a). 5. Facilitating positive health behaviors and well-being to improve health outcomes: Standards of care in diabetes—2026. *Diabetes Care, 49*(Suppl. 1), S89–S131. https://doi.org/10.2337/dc26-S005

American Diabetes Association Professional Practice Committee for Diabetes. (2026b). 3. Prevention or delay of diabetes and associated comorbidities: Standards of care in diabetes—2026. *Diabetes Care, 49*(Suppl. 1), S50–S60. https://doi.org/10.2337/dc26-S003

Basiri, R., Kassem, M., Rajanala, Y., & Frankenfeld, C. L. (2025). Glycemic status and macronutrient intake as predictors of sleep outcomes: An analysis of NHANES 2007–2020 data. *Frontiers in Nutrition, 12*, 1672631. https://doi.org/10.3389/fnut.2025.1672631

Borzouei, S., Ahmadi, A., & Pirdehghan, A. (2024). Sleep quality and glycemic control in adults with type 2 diabetes mellitus. *Journal of Family Medicine and Primary Care, 13*(8), 3398–3402. https://doi.org/10.4103/jfmpc.jfmpc_118_24

Bouman, E. J., Slebe, R., Stenvers, D. J., Elders, P. J. M., Beulens, J. W. J., & Rutters, F. (2024). A randomized controlled trial to assess if changing sleep timing can improve glucose metabolism in people with prediabetes and type 2 diabetes. *Trials, 25*, 474. https://doi.org/10.1186/s13063-024-08329-w

Corrado, A., Scida, G., Abuqwider, J., Annuzzi, E., Giosue, A., Pisano, F., Annuzzi, G., & Bozzetto, L. (2025). Interplay among sleep quality, dinner timing, and blood glucose control in users of advanced technologies: A study in a cohort of adults with type 1 diabetes. *Diabetes Research and Clinical Practice, 221*, 112034. https://doi.org/10.1016/j.diabres.2025.112034

Darraj, A. (2023). The link between sleeping and type 2 diabetes: A systematic review. *Cureus, 15*(11), e48228. https://doi.org/10.7759/cureus.48228

Duan, Y., Fu, H., Gao, J., Wang, S., Chen, C., Zhao, Y., Jiang, S., & Wang, C. (2024). Associations of prediabetes and sleep duration, and inflammation as a mediator in the China Health and

Retirement Longitudinal Study. *Sleep Health, 10*(4), 470–477. https://doi.org/10.1016/j.sleh.2024.01.011

Fan, Z., Wei, R., Chen, T., Yan, X., Yin, S., Cao, Y., Akinyemi, L. I., Zhong, J., Shi, H., & Zhang, F. (2026). Association of weekday sleep duration and estimated glucose disposal rate: The role of weekend catch-up sleep. *BMJ Open Diabetes Research & Care, 14*(2), e005692. https://doi.org/10.1136/bmjdrc-2025-005692

Fentahun, S., Tinsae, T., Rtbey, G., Andualem, F., Kelebie, M., Kibralew, G., Nakie, G., Teferi, S., Tadesse, A., Melkam, M., & Tadesse, G. (2024). Quality of sleep and its determinants among people with diabetes mellitus in Sub-Saharan Africa: A systematic review and meta-analysis. *Behavioral Sleep Medicine, 22*(6), 803–819. https://doi.org/10.1080/15402002.2024.2367469

Gu, K.-M., Min, S. H., & Cho, J. (2022). Sleep duration and mortality in patients with diabetes: Results from the 2007–2015 Korea National Health and Nutrition Examination Survey. *Diabetes & Metabolism, 48*(3), 101312. https://doi.org/10.1016/j.diabet.2021.101312

Henson, J., Covenant, A., Hall, A. P., Herring, L., Rowlands, A. V., Yates, T., & Davies, M. J. (2024). Waking up to the importance of sleep in type 2 diabetes management: A narrative review. *Diabetes Care, 47*(3), 331–343. https://doi.org/10.2337/dci23-0037

Jang, J. H., Kim, W., Moon, J. S., Roh, E., Kang, J. G., Lee, S. J., Ihm, S.-H., & Huh, J. H. (2023). Association between sleep duration and incident diabetes mellitus in healthy subjects: A 14-year longitudinal cohort study. *Journal of Clinical Medicine, 12*(8), 2899. https://doi.org/10.3390/jcm12082899

Kass, L., Sanderson, J. C., Desai, T., & Hurst, R. (2022). The relationship between the elevation of haemoglobin A1c level, sleep quality and sleep duration in clinically diagnosed pre-diabetic patients in a nationally representative sample. *Diabetes & Vascular Disease Research, 19*(1), 14791641211067421. https://doi.org/10.1177/14791641211067421

Kianersi, S., Wang, H., Sofer, T., Noordam, R., Phillips, A., Rutter, M. K., Redline, S., & Huang, T. (2024). Association between accelerometer-measured irregular sleep duration and type 2 diabetes risk: A prospective cohort study in the UK Biobank. *Diabetes Care, 47*(9), 1647–1655. https://doi.org/10.2337/dc24-0213

Kim, Y.-C., Um, Y. J., Yoon, S. H., Kim, T.-W., Seo, H. J., Jeong, J.-H., Hong, S.-C., & Um, Y. H. (2024). Association between weekend catch-up sleep and the risk of prediabetes and diabetes: A cross-sectional study using KNHANES. *Journal of Psychosomatic Research, 179*, 111618. https://doi.org/10.1016/j.jpsychores.2024.111618

Kurnia, A. D., Thato, R., & Tsai, H. T. (2025). Predicting factors of sleep quality among adults with type 2 diabetes mellitus: A systematic review. *Diabetes Research and Clinical Practice, 227*, 112388. https://doi.org/10.1016/j.diabres.2025.112388

Kuroda, H., Au Yeung, S. L., Fujii, R., Iwagami, M., & Goto, A. (2025). Investigating the non-linear association between sleep duration and type 2 diabetes: Conventional and Mendelian randomization analyses from the UK Biobank. *Journal of Diabetes Investigation, 16*(6), 1126–1137. https://doi.org/10.1111/jdi.70039

Laverty, B., Puthezhath Jayanandan, S., & Smyth, S. (2023). Understanding the relationship between sleep and quality of life in type 2 diabetes: A systematic review of the literature. *Journal of Health Psychology, 28*(8), 693–710. https://doi.org/10.1177/13591053221140805

Li, C.-I., Lin, C.-C., Liu, C.-S., Lin, C.-H., Yang, S.-Y., & Li, T.-C. (2022). Sleep duration predicts subsequent long-term mortality in patients with type 2 diabetes: A large single-center cohort study. *Cardiovascular Diabetology, 21*, 60. https://doi.org/10.1186/s12933-022-01500-0

Liu, H., Chen, G., Wen, J., Wang, A., Mu, Y., Dou, J., Gu, W., Zang, L., Zhang, S., & Lyu, Z. (2022). Association between sleep duration and incidence of type 2 diabetes in China: The REACTION study. *Chinese Medical Journal, 135*(10), 1242–1248. https://doi.org/10.1097/CM9.0000000000001835

Mao, Y. (2024). Sleep architecture changes in diabetes. *Journal of Clinical Medicine, 13*(22), 6851. https://doi.org/10.3390/jcm13226851

Mou, Z., Zhou, Y., & Dong, Z. (2026). Association between sleep duration and glycemic levels in adults with prediabetes: A population-based study. *Canadian Journal of Diabetes*. Advance online publication. https://doi.org/10.1016/j.jcjd.2026.05.001

Nakada, R., Lane, D. R., Iwata, S., Isogawa, A., Toyoda, M., Sawaki, H., Murata, T., Kajino, S., Waki, H., Kato, S., Kawaguchi, T., Hirota, Y., Saito, S., Nishikage, S., Yokota, K., Tsurutani, Y., Tone, A., Miyoshi, K., Yamauchi, T., Nangaku, M., & Waki, K. (2026). Digital intervention increasing sleep duration among people with type 2 diabetes: Pilot randomized controlled trial. *Journal of Diabetes Science and Technology*. Advance online publication. https://doi.org/10.1177/19322968261445108

Pham, C. T., Ali, A., Churilov, L., Baqar, S., Hendrieckx, C., O'Neal, D. N., Howard, M. E., & Ekinci, E. I. (2025). The association between glycaemic variability and sleep quality and quantity in adults with type 1 and type 2 diabetes: A systematic review. *Diabetic Medicine, 42*(4), e15485. https://doi.org/10.1111/dme.15485

Shibabaw, Y. Y., Dejenie, T. A., & Tesfa, K. H. (2023). Glycemic control and its association with sleep quality and duration among type 2 diabetic patients. *Metabolism Open, 18*, 100246. https://doi.org/10.1016/j.metop.2023.100246

Slebe, R., Splinter, J. J., Schoonmade, L. J., Blondin, D. P., Campbell, D. J. T., Carpentier, A. C., Despres, J.-P., Hoeks, J., Kalsbeek, A., Raina, P., Schrauwen, P., Serlie, M. J., Stenvers, D. J., Yi, C.-X., de Mutsert, R., Beulens, J. W. J., & Rutters, F. (2025). The effect of altered sleep timing on glycaemic outcomes: Systematic review of human intervention studies. *Diabetes, Obesity and Metabolism, 27*(3), 1172–1183. https://doi.org/10.1111/dom.16104

Wang, P., Li, Q., Yu, X., Wu, L., Liu, J., Zheng, Y., Liu, Z., Yao, J., Fan, S., & Li, Y. (2025). Association between weekend catch-up sleep and glycemic control among individuals with diabetes: A population-based study. *Frontiers in Endocrinology, 16*, 1461367. https://doi.org/10.3389/fendo.2025.1461367

Zhang, J., Yang, C., An, J., Fan, Y., & Dong, X. (2025). Evaluating sleep's role in type 2 diabetes mellitus: Evidence from NHANES. *Brain, Behavior, & Immunity - Health, 44*, 100953. https://doi.org/10.1016/j.bbih.2025.100953