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A Composite Measure of Sleep Health is Associated with Glycemic Target Achievement in Young Adults with Type 1 Diabetes

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Abstract

We investigated whether sleep health (each individual dimension and a composite measure) was associated with better glycemia among a cohort of young adults with type 1 diabetes (T1D) (mean age 21.5 years, mean BMI 24.55 kg/m²). Multiple validated self-report questionnaires were completed, and raw continuous glucose monitor data were shared. One self-reported sleep characteristic for each of the five sleep health dimensions was selected. A composite score was calculated by summing the number of “good” sleep health dimensions. We evaluated the associations between sleep health and glycemia and whether covariates, including age, T1D duration, and sleep apnea risk, influenced the relationships among the study variables using multivariable linear regression. Individual dimensions of sleep satisfaction ($\beta = 0.380, p = .019$; $\beta = -0.414, p = .010$), timing ($\beta = 0.392, p = .015$; $\beta = -0.393, p = .015$), and sleep efficiency ($\beta = 0.428, p = .007$) were associated with higher achievement of glycemic targets (J-index and time in range) however, these associations did not persist after considering covariates. A better sleep health composite score was associated with higher achievement of glycemic targets even after considering covariates. Using a multidimensional framework can guide future research on causal pathways between sleep and diabetes health, interventions to target sleep health profiles, and may improve sleep screening in routine diabetes care.

Keywords

sleep disturbance; glycemia; glucose regulation; gluoregulation; continuous glucose monitor

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Introduction

Sleep health is a broader multidimensional concept characterized across several dimensions (satisfaction, alertness, timing, efficiency, duration; SATED) (Buysse, 2014). Multiple dimensions of sleep health, including satisfaction, alertness, timing, efficiency, and duration, directly or indirectly influence glucose metabolism (i.e., glucoregulation) (Leproult et al., 2014; Spiegel et al., 1999; Van Cauter et al., 2007). Laboratory studies of partial or total sleep deprivation (i.e., duration) and forced desynchrony (i.e., timing) perturb metabolic pathways with an increase in insulin resistance and decrease in glucose tolerance in adults without chronic conditions (eg, healthy) (Leproult et al., 2014; Leproult & Van Cauter, 2010; Spiegel et al., 1999; Van Cauter et al., 2007) and insulin sensitivity adults with T1D (Donga et al., 2010). Specifically alterations to these two prominent sleep health dimensions, duration and timing, lead to an imbalance between glucose production by the liver and glucose utilization by the insulin-dependent tissues (eg muscle and adipose) and non-insulin-dependent tissues (eg brain) (Spiegel et al., 1999; Spiegel et al., 2004; Van Cauter et al., 2007). Impairments in both sleep health and glucoregulation are independent predictors of all-cause mortality (Buysse, 2014; Sondrup et al., 2022).

Type 1 diabetes (T1D) is an autoimmune T cell-mediated condition characterized by destruction of beta cells, absolute insulin deficiency, and a lifelong dependency on exogenous sources (e.g., insulin and glucose) that requires regular self-monitoring to achieve glycemic targets (Burrack et al., 2017). Glycated hemoglobin (HbA1C) is the gold standard long term (chronic) glycemic target measurement for individuals with T1D. Suboptimal achievement (HbA1C <7%) is a major predictor of long-term vascular complications (Nathan & DCCT EDIC Research Group, 2014). Short term glycemic target achievement is often monitored day-to-day during wake and sleep through continuous glucose monitors (i.e., target time-in-range 70 mg –180 mg/dL >80% and glucose management indicator, <7%). Self-monitoring of glucose is a unique challenge for young adults with T1D as they transition away from their childhood homes and providers to adult care and responsibilities (Morrissey et al., 2021).

Promoting modifiable dimensions of sleep health including regularity, timing, efficiency, and duration improves health outcomes among adolescents and young adults without chronic conditions. However less is known about the impact of modifying sleep health dimensions on diabetes health outcomes in individuals with T1D (Beebe et al., 2008; Beebe et al., 2014; Beebe et al., 2015; Van Dyk et al., 2017). In prior research of adolescents or young adults, only one sleep health dimension at a time, predominantly sleep duration or sleep efficiency has been examined with little attention given to other domains (Farabi et al., 2017; Martyn-Nemeth et al., 2018; Patel et al., 2018; Perfect et al., 2012). A combination of sleep health dimensions are likely associated with better diabetes health outcomes. Therefore, there is a need to examine the unique and cumulative contributions of sleep health dimensions on glycemic target achievement in this at-risk population (e.g., multidimensional sleep health studies). The aim of this study was to examine the association between sleep health and glycemia. Each sleep health dimension and a composite measure were the independent variables of interest. We hypothesized that better sleep health (individual dimensions and

composite) would be associated with better achievement of glycemic targets (less glucose variability and more time in range).

MATERIALS AND METHODS

Design

We followed the World Medical Association Declaration of Helsinki for research involving human subjects (World Medical Association, 2014) and received approval from the Case Western Reserve University Institutional Review Board (STUDY20201829). Participants were recruited over a 12-month period (February 2021 to April 2022) through social media, ResearchMatch, and newsletters (College Diabetes Network and a University in Cleveland Ohio). A national convenience sample of English-speaking young adults ages 18–25 years, with type 1 diabetes for at least 6-months, who did not work night shift, were not currently pregnant, or did not have a previous history of obstructive sleep apnea, severe mental illness (e.g., bipolar disorder, schizophrenia), or another major complex chronic condition were included.

Outcome

Glycemic target achievement (glucose management indicator, J index, coefficient of variation [CV], and time-in-range) was determined from the objective raw CGM data that were shared from each participant's existing CGM to capture glucose patterns. J index was calculated as $0.001 \times (\text{mean} + \text{SD})^2$ and time in range was calculated as % time spent in target range 70–180 mg/dL. CGM systems provide real-time, dynamic glucose information every five minutes — up to 288 readings in a 24-hour period (Danne et al., 2017). CGMs are accurate across a wide range of test-retest reliability levels ranging from 0.77 – 0.95 (Danne et al., 2017). Glucose variability indices were calculated across the days of monitoring (Danne et al., 2017).

Sleep Health Characteristics and Dimensions

Obstructive sleep apnea risk was assessed with the validated Berlin questionnaire and categorized as low versus high risk (Netzer et al., 1999).

Sleep satisfaction—Sleep satisfaction was measured with the 8-item PROMIS Sleep Disturbance scale (Cronbach's $\alpha = 0.95$) (Yu et al., 2012). Each item is ranked using a 5-point Likert scale (not at all to very much) (Yu et al., 2012). Scores range from 8–40, with higher scores indicating greater severity of sleep disturbance (Yu et al., 2012). The raw scores on the 8 items are summed to obtain a total raw score. The raw scores are then transformed into a T-score. The Cronbach's α for the PROMIS Sleep Disturbance in the current study was 0.875.

Alertness—Alertness was measured with the 8-item Epworth Sleepiness Scale (ESS) (Cronbach's $\alpha = 0.88$) (Johns, 1992). Scores range from 0–24, with higher scores indicating higher sleepiness (Johns, 1992). The scores on the 8 items are summed to obtain a total score. The Cronbach's α for the Epworth Sleepiness Scale in the current study was 0.771.

Timing—Timing was measured with the 19-item Morningness Eveningness Questionnaire (MEQ) (Cronbach's $\alpha = 0.88$) (Horne & Ostberg, 1976). The scores on the 19-items are summed to obtain a total score. Scores range from 16–86; scores < 41 indicate evening or later types; scores > 59 indicate morning types, and scores 42–58 indicate intermediate types. The Cronbach's alpha for the MEQ in the current study was 0.809.

Sleep efficiency—Sleep efficiency was derived from 3-items on the Pittsburgh Sleep Quality Index (PSQI) (sleep duration, getting up time, and bedtime) (Buysse et al., 1989). We calculated sleep efficiency as (sleep duration / time in bed) \times 100 (Buysse et al., 1989).

Sleep duration—Sleep duration was measured with one item on the PSQI (“*during the past month, how many hours of actual sleep did you get at night? This may be different than the number of hours you spend in bed*”) (Buysse et al., 1989).

Derivation of the Sleep Health Composite: Derivation of the Sleep Health Composite was based on several considerations. We decided to dichotomize each dimension as “good” or “poor.” This approach is more feasible and deployable in clinical practice and has been documented in prior studies (Dong et al., 2019). The Sleep Health Composite was coded as 1 = good and 0 = poor, with scores ranging from 0–5 and higher scores indicating better sleep health (Dong et al., 2019). We chose the cut-off point for each dimension based on the extant literature and data from previously published studies in normative samples (Dong et al., 2019; Ohayon et al., 2017). The cut points for the five dimensions were: satisfaction (PROMIS <56.36, i.e., < 1 SD above the mean), alertness (ESS = 7.5), timing (MEQ 42–58), efficiency = 85%, duration 7–9 hours.

Non-sleep Risk Factors

Participants completed a sociodemographic and clinical form and other study instruments about their diabetes management via an online survey. They shared either their raw continuous glucose monitor data or a share code from Dexcom Clarity so that the research team could access and export the raw glucose data.

Statistical analysis

Prior to the analysis, we screened data for missing or out-of-range values and examined distributions of continuous variables. Data were managed using the REDCap site and exported into the Statistical Package for the Social Sciences (SPSS) version 28 for analysis. CGM data were calculated with Glyculator v. 3.0 software (Czerwoniuk et al., 2011).

A quantitative descriptive approach was used to characterize sleep health dimensions and glycemia. Glucose variability indices were calculated based on data across the days of monitoring. Descriptive statistics were used to summarize each of the variables, including the scores for multi-item scales. Self-report A1c was used to determine chronic glycemia and CGM data were used to calculate short-term day-to-day glycemia (Suh & Kim, 2015). A series of *t*-tests and correlations were conducted to determine differences in sleep health dimensions and glucose variables by covariates, including age, sex, T1D duration, race, and BMI.

Bivariate and multivariable linear regression models were used to examine the relationships between sleep health dimensions and glycemia. A series of linear regression models were conducted for each individual sleep health dimension and for the Sleep Health Composite score to evaluate the explanatory contributions of sleep health dimensions to glycemia. Covariates were included in multivariable models if they were significantly associated with sleep health (composite measure or individual dimension) or glycemia (glucose management indicator, J-index, coefficient of variation, or time in range) ($p < .05$). We also controlled for sleep apnea risk due to *a priori* knowledge of an independent effect of sleep apnea on glycemia (Priou et al., 2012). Statistical significance was set at $p < .05$.

Results

Participant characteristics

Seventy-five participants (74.7% female sex assigned at birth, 9.3% gender minority) between the ages of 18–25 years (mean 21.47, SD = 2.06) participated in the study (64.9% on an insulin pump). Participants reported a mean T1D duration of 9.4 years (SD = 5.8 years) and mean HbA1c of 6.8 % (SD = 1.04%). Mean GMI was 7.01 (SD = 0.63%) and mean time in range was 70% (SD = 17.2%) from raw CGM data. We present demographic, clinical profile, and sleep characteristics in Table 1.

Individual sleep health dimensions

In the first set of models, we examined the associations between each individual sleep health dimension and glycemia as measured by the Glucose Management Indicator (GMI). Higher satisfaction and lower sleep efficiency were significantly associated with a lower GMI (better achievement of target), accounting for 18.6% and 14.9% of the variance, respectively. Satisfaction and efficiency had a medium effect on GMI ($f^2 = 0.23$ and $f^2 = 0.18$), and timing had a small effect on GMI ($f^2 = 0.04$). Alertness and duration did not have an effect on GMI. However, the associations were no longer significant after considering covariates (age, T1D duration, race, insulin mode of delivery, and sleep apnea risk) (Table 2).

In the second set of models, we examined the associations between each individual sleep health dimension and glycemia as measured by J-index. In these models, lower satisfaction, later timing, and lower sleep efficiency were significantly associated with lower achievement of glycemic targets as measured by J-index accounting for 14.5%, 15.3%, and 10.3% respectively; however, these relationships were no longer significant when adjusting for covariates (age, T1D duration, insulin mode of delivery, race, and sleep apnea risk) (Table 3). Satisfaction and timing had a medium effect on J-index ($f^2 = 0.17$ and $f^2 = 0.18$) and efficiency had a small effect on J-index ($f^2 = 0.11$). In the next set of models (Table 4) we examined the associations between individual sleep health dimensions and glycemia as measured by Coefficient of Variation and none of the associations were statistically significant.

In the next set of models, we examined the associations between each individual sleep health dimension and achievement of glycemic targets as defined by % time in glucose range (70–180 mg/dL). Higher satisfaction, earlier timing, and higher sleep efficiency were

significantly associated with more time in range (better target achievement), accounting for 17.1%, 15.4% and 18.3% of the variance, respectively. Satisfaction, timing, and efficiency had a medium effect on time in range ($f^2 = .021$, $f^2 = 0.18$, and $f^2 = 0.22$ respectively). However, the associations were no longer significant after considering covariates (age, T1D duration, insulin mode of delivery, race, and sleep apnea risk) (Table 5).

Sleep Health Composite Score

In the last set of models, we examined the associations between the Sleep Health Composite and all glycemia indices (GMI, J index, CV, and time in range) (Table 6). Better sleep health was significantly associated with better achievement of glycemic targets even after considering covariates (age, T1D duration, insulin mode of delivery, race, and sleep apnea risk) across all glycemic measures GMI ($\beta = -.337$, $p = .031$), J index ($\beta = -.308$, $p = .045$) and time in range ($\beta = .434$, $p = .013$) except for CV ($p = .793$) accounting for 41.3%, 42.7% and 43.4% of the variance respectively. Also, the sleep health composite had a medium effect on all glycemic metrics (GMI $f^2 = 0.21$, J-index, $f^2 = 0.20$, and $f^2 = 0.24$ respectively) except for CV (Table 6). We present the scatterplots of sleep satisfaction, timing, and sleep efficiency and percentage (%) time in range (70–180 mg/dL) in Figure 1.

Discussion

In a sample of community-dwelling young adults with type 1 diabetes, we found that better sleep health (individual sleep health dimensions and a composite measure of sleep health dimensions) was associated with higher achievement of glycemic targets. Specifically, a better sleep health composite score was associated with higher achievement of glycemic targets as measured by continuous glucose monitoring over 7 days even after considering covariates. Only three individual sleep health dimensions, including satisfaction, timing, and efficiency, were significantly associated with achieving glycemic targets in the unadjusted models; however, these were not statistically significant after controlling for covariates. Most of the individual sleep health dimensions had a small or medium effect on each of the glucose variability indices. The composite had a medium effect size on all glucose variability indices except for coefficient of variation. To our knowledge, this is the first study where a composite measure of sleep health dimensions and glycemic targets were investigated in a sample of young adults with T1D. Although in previous studies, poorer glycemia was associated with individual dimensions of sleep health such as short duration and variability in sleep duration (Borel et al., 2009; Chontong et al., 2016; Griggs, Grey, et al., 2021; Griggs et al., 2020; Larcher et al., 2016; Patel et al., 2018), we suggest based on our findings that these sleep health dimensions have positive additive effects. Examining multidimensional sleep health profiles may advance our understanding of the relationships between sleep and glycemia in young adults with T1D and in other populations with and at risk for cardiometabolic disease.

A better sleep health composite score was associated with better glycemic target achievement across most glycemia measures supporting our hypothesis. Other studies where the association between a sleep health composite and glycemia were not available for comparison. Associations between individual sleep health dimensions and glycemia have

varied across other comparison studies (Borel et al., 2009; Chontong et al., 2016; Griggs et al., 2022; Griggs, Grey, et al., 2021; Griggs, Hickman, et al., 2021; Griggs et al., 2020; Griggs, Strohl, et al., 2021; Perfect et al., 2012; Rechenberg et al., 2020; Reutrakul et al., 2016). In these studies poorer individual dimensions of sleep health (e.g., lower satisfaction, lower daytime alertness, variability in timing, lower efficiency, and or shorter duration) were associated with lower achievement of glycemic targets (Borel et al., 2009; Chontong et al., 2016; Griggs et al., 2022; Griggs, Grey, et al., 2021; Griggs, Hickman, et al., 2021; Griggs et al., 2020; Griggs, Strohl, et al., 2021; Perfect et al., 2012; Rechenberg et al., 2020; Reutrakul et al., 2016).

In the current study, poor sleep satisfaction (specifically sleep disturbance) was significantly associated with lower achievement of glycemic targets. This finding is consistent with several cross-sectional studies of small clinical samples and epidemiological studies (Matejko et al., 2015; Perfect et al., 2012; Reutrakul et al., 2016; von Schnurbein et al., 2018). In other studies of adolescents with T1D, sleep satisfaction was not associated with glycemia (Patel et al., 2018; Perfect et al., 2012). However, measures of sleep satisfaction differ among these studies. Satisfaction in the current study was measured with the PROMIS sleep disturbance scale, which measures perceptions of sleep quality, sleep depth, and restoration associated with sleep over the past 7-days. The Pittsburgh Sleep Quality Index global score or an individual sleep quality item was used in the other studies (Patel et al., 2018; Reutrakul et al., 2016).

Of the individual sleep health dimensions, sleep timing had the strongest association with glycemia. This finding is consistent with some previous studies (Griggs, Grey, et al., 2021; Reutrakul et al., 2016), but not others (Perfect et al., 2012; Siwasaranond et al., 2016). Consistent with our findings in the current study, the association between better or more stable timing and lower glycemia in individuals with T1D has been highlighted in numerous studies (Chontong et al., 2016; Griggs et al., 2022; Griggs, Grey, et al., 2021; Griggs, Hickman, et al., 2021; Griggs et al., 2020; Griggs, Strohl, et al., 2021; Patel et al., 2018; Perfect et al., 2012; Rechenberg et al., 2020; Reutrakul et al., 2016; Reutrakul & Van Cauter, 2014). Beyond cross sectional studies, in a recent clinical trial, the chronic sleep restriction with recurrent circadian disruption condition had significantly elevated postprandial plasma glucose levels whereas the condition with chronic sleep restriction and minimized circadian disruption had no adverse glycemic effects after 3 weeks of exposure among adults without chronic conditions (Yuan et al., 2021).

Neither sleep duration nor daytime alertness as individual dimensions were significantly associated with any of the glycemic target measures in the current study. This finding was not expected given the evidence of impaired glucose metabolism in adults without chronic conditions undergoing experimental sleep deprivation (Donga et al., 2010; Knutson & Van Cauter, 2008; Spiegel et al., 1999; Van Cauter et al., 2007). In previous comparison studies of adults with T1D, this association has been mixed with some researchers reporting an association between a shorter sleep duration and poorer glycemia (Borel et al., 2013; Denic-Roberts et al., 2016; Reutrakul et al., 2016; von Schnurbein et al., 2018) or between a lower daytime alertness and poorer glycemia (Griggs, Hickman, et al., 2021; Perfect et al., 2012; Zhu et al., 2021). In other studies, the associations between either sleep duration or

daytime alertness and glycemia have not been significant (Griggs, Grey, et al., 2021; Griggs, Hickman, et al., 2021; Griggs et al., 2020; Patel et al., 2018; Rechenberg et al., 2020).

The current cross-sectional study and analyses have several limitations to consider when interpreting the results. First, the sample was not representative of community dwelling young adults with T1D as a majority were Non-Hispanic White (87%) and female (75%). Therefore, results cannot be generalized to the general T1D population, to other age groups (adolescents, middle or older adults), or to clinical populations with sleep disorders. However, the current sample did achieve glycemic targets at comparable rates than the T1D Exchange, a national comparison sample (mean GMI 7.0% vs. 7.3%) (Foster et al., 2019). Also, neither sex- nor race-based differences could be determined. Despite these limitations, the current study design is an important first step to gauge the utility of a sleep health composite score in young adults with T1D. Larger population-based studies are needed to evaluate the psychometric properties of a sleep health composite score in young adults with T1D as well as other clinical and non-clinical samples. Second, though we constructed items in equal weights based on clinically significant setpoints in the extant literature, different dimensions may warrant different weights especially in this unique sample of young adults with T1D. Third, while the MEQ measures self-reported timing preference, it is likely distinct from sleep variability. Although they both assess timing of sleep, the impact of a later chronotype (especially consistent late sleep timing) and sleep variability (where sleep timing varies from day to day) on achievement of glycemic targets requires further investigation. Those with a later chronotype may experience higher sleep variability consistently. In the future, researchers should incorporate objective measures of sleep health dimensions and glycemia such as concurrent actigraphy and continuous glucose monitors as well as lab drawn, or chart extracted A1c. Nevertheless, we used well validated and reliable scales to determine sleep health, and this approach is more feasible and deployable in clinical settings.

There is a paucity of studies focused on determining whether modifying sleep has an effect on glycemia in young adults with T1D. Promoting sleep duration had a positive impact on glucose targets in two pilot studies of adolescents with T1D in the short-term (3 months) (Jaser et al., 2020; Perfect et al., 2016; Perfect et al., 2018). Specifically, in a randomized controlled trial, Perfect and colleagues established that a behavioral intervention aimed at increasing sleep duration by 30 minutes per day led to an improvement in time in range after 3 months (Perfect, 2018). In a small experimental hyper insulinemic euglycemic clamp study of 7 middle-aged adults with T1D (mean age 44, SD 7 years), sleep restriction reduced the glucose disposal rate reflecting decreased peripheral sensitivity (Donga et al., 2010). It is unknown if extending sleep duration over time is sustainable, nor what the long-term impact is on clinical outcomes specifically in young adults with T1D who have unique developmental and social needs.

The current study provides support of the utility of a sleep health composite score. The results can help to establish validity in the context of chronic illness and may translate to other related populations such as those with type 2 diabetes, hypertension, or obesity. Future researchers may consider a similar approach in other populations and contexts. Linear relationships were examined in the current study, and in the future, it may be warranted

to examine both linear and nonlinear associations between sleep health dimensions and glycemia. The current study was cross sectional therefore the mechanistic pathways between sleep health and glucose variability could not be determined. Future longitudinal and experimental studies can provide further insight into the findings presented here and whether poor sleep precedes greater glucose variability, or vice versa, or acts bidirectionally. In addition, age effects on multidimensional sleep health and whether relationships are consistent in broader age ranges, such as adolescents and middle to older aged adults with T1D should be examined. Promoting healthy sleep through a multidimensional framework addresses the entire diabetes population rather than only those with sleep disorders.

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SG, PI on the grant (R00NR018886), secured the funding, designed the study, collected, analyzed, and interpreted the data and wrote the manuscript. GP interpreted the data and cowrote the manuscript. Senior author RLH contributed to the study design, interpreted the findings, and co-wrote the manuscript. All authors have seen and approved the final version of this manuscript.

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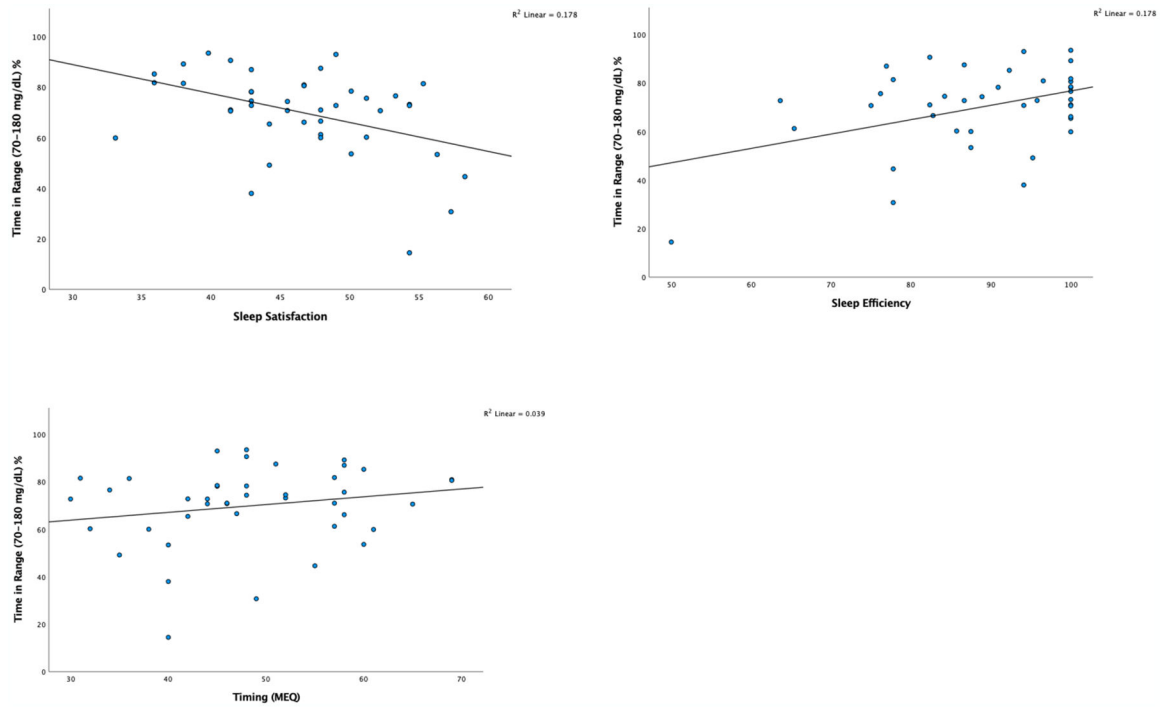


Figure 1. Scatterplots of Sleep Satisfaction, Timing, Sleep Efficiency (%) and Time in Range 70–180 mg/dL %

Table 1

Demographic, clinical, and sleep profile

Demographic profile		
	Mean or N	SD or (%)
Age in years	21.47	2.056
BMI (kg/m ²)	24.55	4.65
Non-Hispanic White (%)	65	86.7
Female (%)	56	74.7
Gender minority (%)	7	9.3
Clinical profile		
T1D duration in years	9.37	5.74
HbA1c (%)	6.83	1.04
Glucose management indicator	7.01	0.63
Glucose, mean	154.64	26.22
J Index	44.84	17.84
Coefficient of Variation (CV)	34.05	5.30
Time in range (% 70–180 mg/dL)	69.93	17.17
Time above range (% > 180 mg/dL)	27.93	16.98
Time above range (% > 250 mg/dL)	8.13	10.66
Time below range (% < 70 mg/dL)	2.14	2.40
Time below range (% < 54mg/dL)	0.43	1.01
Insulin pump (n) (%)	48	(64.9)
Sleep profile		
Satisfaction ¹	48.69	7.70
Alertness ²	7.64	4.26
Timing ³ total	48.33	9.24
Early (n) (%)	10	(13.3)
Intermediate (n) (%)	49	(65.3)
Late (n) (%)	16	(21.3)
Efficiency ⁴	86.13	13.01
Duration (PSQI) ⁴	7.25	1.29
Sleep Health Composite	3.04	1.26

Note:

¹PROMIS Sleep Disturbance;²ESS Epworth Sleepiness Scale;³MEQ, Morningness Eveningness Questionnaire;⁴PSQI, Pittsburgh Sleep Quality Index.

Contribution of individual sleep health dimensions to glucose management indicator (linear regression models)

Table 2

Predictors	Model 1 Unadjusted				Model 2 Adjusted for Covariates						
	B	SE	β	R ²	P value	F ²	B	SE	β	R ²	P value
Satisfaction PROMIS	0.55	.018	.431	.186	.004	0.23	.026	.020	.230	.328	.214
Alertness	.007	.025	.042	.002	.792	0.00	.038	.022	.267	.356	.096
Timing	-.011	.010	-.184	.034	.244	0.04	.010	.010	.172	.316	.314
Efficiency	-.020	.008	-.385	.149	.014	0.18	-.005	.009	-.101	.319	.536
Duration	.007	.103	.011	.001	.945	0.00	-.049	.100	-.078	.315	.630

Note: Model 1 is unadjusted. Model 2 is adjusted for covariates: age, type 1 diabetes duration, race, mode of insulin delivery, and sleep apnea risk. B is the unstandardized coefficient regression coefficient. SE standard error. β is the standardized regression coefficient. R² = coefficient of determination shown for each model.

Contribution of individual sleep health dimensions to glucose variability J-index (linear regression models)

Table 3

Predictors	Model 1 Unadjusted				Model 2 Adjusted for Covariates						
	B	SE	β	R ²	P value	F ²	B	SE	β	R ²	P value
Satisfaction PROMIS	1.085	.440	.380	.145	.019	0.17	.369	.468	.138	.343	.437
Alertness	.179	.737	.041	.002	.809	0.00	.968	.623	.237	.380	.131
Timing	11.538	4.517	.392	.153	.015	0.18	.231	.271	.140	.345	.401
Efficiency	-433	.213	-.320	.103	.050	0.11	-.120	.248	-.077	.345	.633
Duration	.561	2.917	.032	.001	.849	0.00	-1.312	2.873	-.072	.345	.651

Note: Model 1 is unadjusted. Model 2 is adjusted for covariates: age, type 1 diabetes duration, race, mode of insulin delivery, and sleep apnea risk. B is the unstandardized coefficient regression coefficient. SE standard error. β is the standardized regression coefficient. R² = coefficient of determination shown for each model. F² = Cohen's f² effect size.

Contribution of individual sleep health dimensions to glucose variability – Coefficient of Variation (linear regression models)

Table 4

Predictors	Model 1 Unadjusted				Model 2 Adjusted for Covariates						
	B	SE	β	R ²	P value	F ²	B	SE	β	R ²	P value
Satisfaction PROMIS	.089	.136	.104	.011	.514	0.01	-.010	.138	-.013	.310	.945
Alertness	.183	.210	.136	.019	.389	0.01	.098	.188	.083	.316	.608
Timing	-.078	.082	-.148	.022	.350	0.02	-.034	.080	-.072	.213	.670
Efficiency	.069	.066	.167	.028	.302	0.03	.008	.074	.018	.308	.912
Duration	.904	.786	.183	.034	.257	0.04	5.00	4.44	.178	.308	.270

Note: Model 1 is unadjusted. Model 2 is adjusted for covariates: age, type 1 diabetes duration, race, mode of insulin delivery, and sleep apnea risk. B is the unstandardized coefficient regression coefficient. SE standard error. β is the standardized regression coefficient. R² = coefficient of determination shown for each model. F² = Cohen's f² effect size.

Contribution of individual sleep health dimensions to time in range (linear regression models)

Table 5

Predictors	Model 1 Unadjusted				Model 2 Adjusted for Covariates						
	Model 1 Unadjusted				Model 2 Adjusted for Covariates						
	B	SE	β	R ²	P value	F ²	B	SE	β	R ²	P value
Satisfaction PROMIS	-1.137	.417	-.414	.171	.010	0.21	-.547	.414	-.235	.327	.196
Alertness	-.071	.709	-.017	.000	.920	0.00	-.950	.557	-.266	.351	.099
Timing	-11.127	4.343	-.393	.154	.015	0.18	-.160	.245	-.111	.298	.518
Efficiency	.612	.216	.428	.183	.007	0.22	.207	.222	.151	.317	.359
Duration	.047	2.807	.003	.000	.987	0.00	1.63	2.59	.101	.306	.535

Note: Model 1 is unadjusted. Model 2 is adjusted for covariates: age, type 1 diabetes duration, race, mode of insulin delivery, and sleep apnea risk. B is the unstandardized coefficient regression coefficient. SE standard error. β is the standardized regression coefficient. R² = coefficient of determination shown for each model. F² = Cohen's f² effect size.

Table 6

Contribution of sleep health dimension composite score to glycemia (glucose management indicator, coefficient of variation, and time in range) (linear regression models)

Outcomes	Model 1 Unadjusted				Model 2 Adjusted for Covariates						
	B	SE	β	R ²	P value	F ²	B	SE	β	R ²	P value
Glucose Management indicator	-2.47	.088	-.414	.171	.008	0.21	-.175	.077	-.337	.413	.031
J index	-6.634	2.432	-.405	.164	.010	0.20	-4.705	2.25	-.308	.427	.045
CV	-.405	.747	-.088	.008	.591	0.01	-.188	.712	-.043	.309	.793
Time in range	6.937	2.304	.439	.193	.005	0.24	5.194	1.96	.387	.434	.013

Note: Model 1 is unadjusted. Model 2 is adjusted for covariates: age, type 1 diabetes duration, race, mode of insulin delivery, and sleep apnea risk. B is the unstandardized coefficient regression coefficient. SE standard error. β is the standardized regression coefficient. R² = coefficient of determination shown for each model. F² = Cohen's f² effect size.