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Objective Sleep-wake Characteristics are Associated with Diabetes Symptoms in Young Adults with Type 1 Diabetes

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Abstract

Purpose: The primary purpose of this descriptive cross-sectional study was to examine the associations between sleep-wake characteristics (total sleep time, sleep variability, sleep onset latency, and sleep efficiency), distress symptoms (general and diabetes), and diabetes physical symptoms in young adults ages 18–30 years with T1D. The secondary purpose was to determine whether biological sex, body mass index (BMI), and T1D duration (covariates) influence the relationships among the study variables.

Methods: Forty-six young adults with T1D, recruited from diabetes clinics from December 2018 to February 2020, wore a wrist actigraph and continuous glucose monitor concurrently for 6–14 days and completed the PROMIS Emotional Distress Scale, Diabetes Distress Scale, and Diabetes Symptom Checklist-Revised.

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Data: Data are available upon reasonable request.

Results.—Shorter total sleep time and poorer sleep efficiency were associated with higher diabetes emotional distress symptoms. Higher sleep variability was associated with higher neurological pain symptoms. A longer sleep onset latency was associated with higher symptoms of diabetes distress, psychological, cognitive, hyperglycemia, and a higher total symptom burden. Associations remained statistically significant after adjusting for biological sex and BMI, with the exception of sleep onset latency and total symptom burden.

Conclusions.—Poorer objective sleep-wake characteristics were associated with higher diabetes symptoms even after considering biological sex and BMI among young adults with T1D.

Keywords

Sleep; diabetes; symptom management; health behavior; young adult

Despite the growing recognition that short sleep (duration <6.5 hours) and sleep variability (day-to-day changes in sleep duration) impacts general health and well-being, it remains unclear how objective sleep-wake characteristics relate to general and type 1 diabetes (T1D) symptom-specific emotional distress and physical symptoms. The risk of short and variable sleep among those with T1D is highest in young adulthood (18–30 years). Biological (later chronotype and delayed melatonin secretion onset) and environmental factors (early school/work start times, light exposure, social interactions, transition into adult diabetes care) impact diabetes self-management (e.g., diet, exercise, insulin).[1] Short sleep and not achieving glycemic targets (only 14–30%) is associated with a higher risk for premature vascular complications relative to peers.[2] Young adults with T1D experience high symptom burden leading to greater healthcare use and costs during the college or career transition.[2]

Symptoms associated with T1D may be directly related to hyperglycemia (e.g., polyuria, polydipsia, etc.), complications associated with diabetes (e.g., loss of sensation in the extremities, cognitive dysfunction), or hypoglycemia (e.g., irritability and mood dysregulation).[3] Short sleep magnifies the severity of these symptoms. Fourteen young adults in an experimental total sleep deprivation condition experienced more persistent hypoglycemia symptoms and prolonged cognitive dysfunction during the recovery period than the normal sleep time control condition.[4]

Objective and self-report indicators of sleep-wake characteristics are associated with diabetes emotional distress, fatigue, and daytime sleepiness in young adults with T1D[4] and general and diabetes emotional distress in adolescents with T1D.[5] Self-reported sleep-wake characteristics are linked to fatigue, a higher self-management burden, higher daytime sleepiness, depressive and anxiety symptoms.[6,7] Self-reported sleep is subject to self-report bias as participants mostly report time in bed vs. time asleep.[7] Also, data are collected at a single time point with the inability to capture sleep variability or habituality.

Therefore, the primary purpose of this descriptive cross-sectional study was to examine the associations between sleep-wake characteristics (total sleep time, sleep variability, sleep onset latency, and sleep efficiency), distress symptoms (general and diabetes), and diabetes physical symptoms in young adults ages 18–30 years with T1D. The secondary purpose was to determine whether biological sex, body mass index (BMI), and T1D duration

(covariates) influence the relationships among the study variables. Adjusting for symptom-related covariates will allow for more precision in determining relationships. Identifying key modifiable sleep-wake behavioral characteristics may serve as novel behavioral targets to improve the emotional and diabetes-related symptom burden in young adults with T1D, who are at a high risk of premature micro and macrovascular complications.

Methods

Design, Setting, and Sample

The primary aim of this cross-sectional study was to examine the relationships between objective sleep-wake characteristics over 6–14 days and symptoms among young adults aged 18–30 years in the Northeastern United States. This study was reviewed and approved by both the Case Western Reserve University (#20200650) and the Yale University Human Investigation Committee (#1507016174). From December 2018 to February 2020, before the United States officially declared the COVID-19 pandemic, young adults ages 18–30 years (1) diagnosed with T1D for at least six months; (2) with no other major health problems (e.g., chronic medical conditions or severe psychiatric illness); (3) not participating in any intervention studies; and (4) understood English who were affiliated with a diabetes clinic were invited to participate. Those with a previous OSA diagnosis, night shift workers, and current pregnancy were not eligible to participate. The Berlin Questionnaire was used to screen participants for inclusion in the study.[8] Participants considered to be at high risk for sleep apnea were referred for treatment and not included in the study. It was reported previously that better sleep and circadian characteristics were associated with better glycemia across 6–14 days of monitoring between persons and that poorer sleep (lower efficiency, longer wake after sleep onset) predicted higher next day glucose variability and vice versa within-person.[9]

Measurements

Objective sleep-wake characteristics—Participants were instructed to wear the Spectrum Plus for 7–14-days. The Spectrum Plus is a non-dominant wrist worn device that is worn for the monitoring period 24/7. The Spectrum Plus collects activity data in 30-second epochs with a standard spectrum of light and off wrist detection. Five days or longer of monitoring reduces inherent measurement errors and increases reliability.[10] The PI computed sleep characteristics using Actiware 6.0.9 software from Spectrum Plus data including: total sleep time, sleep efficiency (%), wake after sleep onset, and sleep onset latency. Sleep variability was calculated using the Mean Square of Successive Differences (MSSD) across the 14 nights. These approaches for analyzing sleep variability have been documented in prior research.

Self-reported sleep-wake characteristics—Global sleep quality was assessed using the 19-item Pittsburgh Sleep Quality Index (PSQI) (Cronbach's $\alpha = 0.87$; diagnostic sensitivity 89.6%; specificity 86.5%).[11] PSQI component scores are summed and range from 0–21 with higher scores indicating poorer sleep quality.[12] The total scale score was used to describe the sample. Scores ≥ 5 meet the threshold for poor sleep quality.[12] The Cronbach's alpha for the PSQI in the current study was 0.750.

Emotional Distress Symptoms—General emotional distress was measured with the 8-item PROMIS v1.0 (emotional distress-depression) (Cronbach's alpha = 0.95).[13] Each item is ranked using a 5-point Likert scale (ranging from never to always).[13] Scores range from 8–40, with higher scores indicating more emotional distress-depressive symptoms.[13] The Cronbach's alpha in the current study was 0.882.

Diabetes emotional distress was measured with the 17-item Diabetes Distress Scale (Cronbach's alpha = 0.88 to 0.93).[14] The Diabetes Distress Scale measures diabetes-related emotional distress, and each item was answered using a 6-point Likert scale (1= not a problem to 6 = a very serious problem) reflecting the degree to which the item is perceived as a problem.[14] Scores range from 17–102, with higher scores indicating higher diabetes-related emotional distress. The Cronbach's alpha in the current study was 0.937.

Diabetes symptoms—Diabetes symptoms were measured with the 34-item Diabetes Symptom Checklist-Revised (Cronbach's alpha = 0.69–0.87).[3] The items of the Diabetes Symptom Checklist-Revised are grouped into eight symptom clusters or domains, each measuring a different aspect of diabetes symptomatology – hyperglycemia, hypoglycemia, psychological-cognitive, psychological-fatigue, cardiovascular, neurological-pain, neurological-sensory, and ophthalmologic.[3] For each item, participants were asked if they had experienced the symptom in the past four weeks, and if yes, how troublesome the symptom was. Items were summed to form domain scores, and all items were then summed to form the total symptom burden score. Higher scores indicate a higher symptom burden. The Cronbach's alphas in the current study ranged from 0.715 to 0.894 for the subscales and were 0.944 for the 34-item total symptom burden scale.

Glycemia—Achievement of glycemic targets was determined by the most recent glycated hemoglobin (A1C), which is routinely measured at quarterly clinic visits using the Siemens Vantage Glucose Analyzer ® (range = 2.5 – 14%).[15] Glucose variability was determined from the CGM data that were downloaded directly from each participant's existing or the provided blinded Dexcom G4 CGM to capture glucose patterns. All participants wore a CGM for the study period as this was a requirement of the study. CGM systems provide real-time, dynamic glucose information every five minutes — up to 288 readings in a 24-hour period.[16] Participants used an automatic inserter to insert a small sensor wire just under their skin (Wagner et al., 2012).[16] CGMs are accurate across a wide range of test-retest reliability levels ranging from 0.77 – 0.95.[17] Glucose variability indices were calculated from CGM as mean ± SD across the days of monitoring.[18] Both glycemia and glucose variability were used to describe the sample.

Demographic and clinical characteristics—Clinical and demographic data were extracted from the electronic medical record (EMR), including age, BMI (kg/m^2), duration of diabetes, most recent A1C, and medical history. Ethnicity, education, primary caregiver, employment status, full-time student status, work hours, marital status, residence, household count, income, cigarette smoking, alcohol or other substance use, insulin therapy regimen, CGM device brand (if applicable), and last menstrual period for females' data were collected via self-report survey. When appropriate, self-report data were also cross validated with data found in the EMR.

Statistical analyses

Data were managed using the REDCap site and exported into the Statistical Package for the Social Sciences version 27 and SAS 9.4 for analysis. Actigraphy data generated from the Spectrum Plus were scored with Actiware v. 6.0.9 software. CGM data were calculated with Glyculator v. 2.0 software.[19] Descriptive statistics were used to summarize each of the variables, including scores for multi-item scales. A quantitative descriptive approach was used to characterize sleep-wake and symptoms among the 46 young adults with T1D over the 6 – 14 days to capture weekend and weekday differences. A1C was used for glycemic target and CGM data to calculate mean glucose across the days of monitoring.[18] Objective sleep-wake characteristics were summarized across the days of collection.

Bivariate correlations and linear regression models were used to examine the relationships among objective sleep-wake characteristics and symptoms. To evaluate explanatory contributions of objective sleep-wake characteristics to symptoms, a series of linear regression models were conducted for each of the sleep-wake variables that were significant in the unadjusted associations. Statistical significance was set at $p < .05$.

Results

Sample characteristics

Forty-six young adults in the study had a mean age of 22.3 (± 3.2) years, mean BMI of 27.0 (± 4.4) kg/m^2 were 67.4% female, 84.8% non-Hispanic White, and 93.5% reported the ability to meet their monthly expenses. The mean diabetes duration was 10.3 (± 6.0) years, mean HbA1c was 7.2 % (± 1.1%, 55 mmol/mol), and most used an insulin pump (80.4%) and CGM (87%) for treatment and monitoring. The mean glucose was 163.0 (± 30.2) mg/dL measured via CGM across the 7–14 days. Actigraphy data were available for all participants ($N = 46$, mean = 8.7 ± 2.6 days/nights) with 97.8% wearing it the requested number of days. There were no missing self-reported data.

Sleep-wake characteristics

The majority of the current sample slept less than 7 hours on average (54.3%, $n = 25$), ranging from 5 hours 24 minutes to 9 hours 25 minutes, across the days of monitoring. Young adults reported a mean PSQI global sleep quality score of 5.91 (± 3.5) (poor sleep quality ≥ 5). Self-report sleep was associated with objectively measured total sleep time ($r = 0.46$, $P = .001$).

Emotional distress symptoms and diabetes symptoms

The mean emotional general distress score for the total sample was 49.39 (± 7.19) with 30.4% ($n = 14$), meeting the cutoff for moderate emotional distress.[13] The mean diabetes distress score for the total sample was 32.17 (± 14.07) with 41.3% ($n = 19$) meeting the threshold for moderate diabetes distress (mean item score ≥ 3).[14] Sex and BMI differences were noted for diabetes symptoms. Specifically, females reported higher hypoglycemia (M ± SD = 1.77 ± 0.99 vs. 0.73 ± 0.88, $P = .001$) and fatigue symptoms (2.15 ± 1.13 vs.1.13 ± 1.24, $P = .008$) than males respectively. Those with higher BMI reported greater psychological fatigue ($r = 0.32$, $P = .030$), neurological pain ($r = 0.41$, $P = .005$), and

cardiovascular symptoms ($r = 0.34$, $P = .033$). There was not a significant association between T1D duration and any of the symptom scores.

Associations among objective sleep-wake characteristics and symptoms

Shorter total sleep time was associated with higher diabetes emotional distress ($r = -0.32$, $P = .032$). A longer sleep onset latency and poorer sleep efficiency were associated with higher diabetes emotional distress ($r = 0.36$, $P = .014$ and $r = -0.35$, $P = .018$ respectively). The associations between the sleep-wake characteristics and general emotional distress symptoms were not significant.

Higher sleep variability was associated with higher symptoms of neurological pain ($r = 0.32$, $P = .028$). A longer sleep onset latency was associated with higher psychological cognitive symptoms ($r = 0.37$, $P = .012$), higher hyperglycemia symptoms ($r = 0.33$, $P = .024$), and a higher total symptom burden ($r = 0.30$, $P = .042$). Poorer sleep efficiency was associated with higher diabetes emotional distress ($r = -0.35$, $P = .018$). The associations between wake after sleep onset or the sleep fragmentation index and diabetes symptoms were not significant. The contribution of significant sleep-wake characteristics to symptoms while controlling for covariates in separate models are presented in Tables 1, 2, and 3.

In the first model, total sleep time was examined (Table 1). The association between total sleep time and diabetes emotional distress remained statistically significant ($P = .033$) after controlling for sex and BMI accounting for 16.5% of the variance. In the second model, we examined sleep efficiency (Table 1). The association between sleep efficiency and diabetes distress remained statistically significant ($P = .028$) after controlling for sex and BMI accounting for 17% of the variance.

In the next model, sleep variability was examined (Table 2). The association between sleep variability and neurological pain symptoms remained statistically significant ($P = .020$) after controlling for sex and BMI, and BMI was also significant ($P = .006$), accounting for 27% of the variance. The association between sleep variability and total symptom burden remained statistically significant ($P = .041$) after controlling for sex and BMI accounting for 19.8% of the variance.

In the final model, sleep onset latency was examined (Table 3). The association between sleep onset latency and diabetes distress remained statistically significant ($P = .027$) after controlling for sex and BMI accounting for 17.1% of the variance. The associations between sleep onset latency and psychological cognitive and hyperglycemia symptoms in separate models remained statistically significant after controlling for sex and BMI accounting for 19.6% and 15.4% of the variance, respectively. The association between sleep onset latency and total symptom burden was no longer significant when sex and BMI were added to the model.

Discussion

Among young adults with T1D, objective measures of poorer sleep-wake characteristics (shorter total sleep time, lower sleep efficiency, higher variability, and a longer sleep onset

latency) were associated with higher diabetes emotional and physical symptoms even after considering biological sex and BMI. Diabetes emotional distress symptoms, but not general distress symptoms, were associated with objective sleep characteristics (total sleep time, sleep efficiency, sleep variability, and sleep onset latency). This cohort of young adults with T1D also exhibited significant sleep-wake alterations, particularly short sleep duration captured both objectively and via self-report and clinically significant sleep disturbance. Together, our findings highlight the critical importance of sleep duration and variability as contributors to individuals' perception of their diabetes and emotional distress symptom severity. Our findings support the need for greater attention to sleep health as an evidence-based component of T1D care for young adults.

The current findings add to previous studies of T1D. A short and variable sleep duration have unique contributions to a higher symptom burden among young adults with T1D who are required to follow an intensive regimen involving glucose and diet management, insulin administration, and engagement in regular physical activity. The finding in the current study related to shorter sleep duration and higher neuropathic pain was consistent with another study of adults with T1D;[20] however, other diabetes symptoms (e.g., hypoglycemia, hyperglycemia, fatigue, etc.) were not measured in the latter or further studies and were thus not available for comparison. Experimentally restricting sleep resulted in emotional distress in adolescents and adults without chronic conditions.[1,21]Although the causality between sleep duration and distress remains unclear, short sleep duration plays a role given the linear association over time,[22] and the direct effects that were noted when sleep was restricted in adolescents and young adults in a previous study.[1,21]

A few limitations should be considered within the context of interpreting these results. First, the present study sample was primarily Caucasian (84.8%), socioeconomically advantaged (94%), and from a single recruitment site; therefore, demographic differences in these variables could not be determined. Also, some other traits were represented disproportionately relative to the national T1D population, including female sex (67% vs. 50%), optimal glycemia (44% vs. 30%), and CGM use (87% vs. 30%).[23] Finally, the relatively small sample size raises the possibility of type II statistical error regarding associations among total sleep time, sleep variability, and some diabetes symptoms or general emotional distress symptoms.

The study also had several strengths. Young adults were exclusively enrolled, a subset of people with T1D with unique chronobiological and environmental threats to sleep-wake characteristics. A comprehensive T1D-specific symptom panel and objective sleep-wake characteristics were measured over a longer timeframe than previous studies to capture typical variation (6–14 days). Finally, complex, multiple variable relationships beyond correlation were investigated to strengthen and verify the correlational findings.

Future investigators should clarify the directionality of these associations and the potential utility of promoting sleep (extending sleep duration and decreasing variability) in the mitigation of diabetes symptoms. This approach would help provide insight into whether poor sleep precedes higher symptoms, or vice versa, or acts bidirectionally. For example, having distress at night is associated with an inability to fall and stay asleep.[24] but in turn,

sleep that is of inadequate quantity or quality leads to an imbalanced mix between emotional regulation (controlled by the amygdala and prefrontal cortex) and mood hormones (e.g., serotonin, dopamine, etc.).[25] Clinicians working with young adults with T1D should aim to improve achievement of glycemic targets by addressing sleep-wake behaviors and diabetes symptoms, particularly those that may be interfering with nocturnal sleep.

The mechanisms between sleep disturbance and internalized problems (e.g., stress arousal, emotion-processing, and cognitive factors)[26] underlie several modifiable risks. Symptoms and sleep characteristics may be amenable to cognitive-behavioral interventions (CBT) to improve sleep.[24] CBT components may have the dual benefit of improving multiple sleep dimensions (perceived sleep quality, total sleep time, wake after sleep onset), emotional distress (general and diabetes), and diabetes symptoms.

The diabetes symptoms checklist was originally developed for type 2 diabetes, and later four subscales (cognitive distress, fatigue, hyperglycemia, and hypoglycemia) were psychometrically evaluated for adults with T1D (mean age 40 years, 65% Caucasian). More work needs to be done to further develop this scale for other symptoms (e.g., cardiology, ophthalmology, neurology-pain, etc.) experienced by adolescents and adults with T1D across the lifespan so that reference norms can be established. This is of particular importance to improve tracking in clinical trials and systematic screening in the clinical setting. This approach would allow for recommendations to be put into place to prevent premature micro and macrovascular complications (e.g., diet, exercise, and sleep interventions to lower BMI and A1C) before they arise.

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Table 1.

Contribution of total sleep time and sleep efficiency and covariates to diabetes emotional distress.(Regression Model)

	Outcome									
	Diabetes Distress									
Predictor	**B**	**SE**	**β**	**P value**		***Predictor***	**B**	**SE**	**β**	**P value**
Total Sleep Time	**−0.07**	**0.03**	**−0.30**	**.033**		Sleep Efficiency	**−1.02**	**0.45**	**−0.35**	**.028**
Covariates										
Sex	5.36	4.39	0.19	.229		Sex	5.89	4.42	0.20	.190
BMI	0.44	0.47	0.17	.354		BMI	0.21	0.50	0.07	.680
R^2	.165					.170				

Note. B is the unstandardized coefficient regression coefficient. SE standard error. β is the standardized regression coefficient. R^2 = coefficient of determination shown for each model. **Bolded values are significant.**

Table 2.

Contribution of sleep variability and covariates to neurology pain and total symptom burden. (Regression Model)

	Outcomes							
	Neurology pain R^2 = .270				**Total symptom burden R^2 = .198**			
Predictor	**B**	**SE**	**β**	**P value**	**B**	**SE**	**β**	**P value**
Sleep Variability	**1.84** [a]	**0.00**	0.32	**.020**	**1.59** [a]	**0.00**	**0.29**	**.041**
Covariates								
Sex	0.05	0.19	0.03	.806	0.29	0.19	0.22	.128
BMI	**0.06**	**0.02**	0.40	**.006**	0.03	0.02	0.22	.133

Note. B is the unstandardized coefficient regression coefficient. SE standard error. β is the standardized regression coefficient. R^2 = coefficient of determination shown for each model. **Bolded values are significant.**

[a]Unstandardized values B were multiplied by 10,000.

Table 3.

Contributions of sleep onset latency and covariates to emotional distress and diabetes symptoms. (Regression Model)

	Outcomes											
	Diabetes Distress R^2 = .171				**Psychology, cognitive R^2 = .196**				**Hyperglycemia R^2 = .154**			
Predictor	**B**	**SE**	**β**	**P value**	**B**	**SE**	**β**	**P value**	**B**	**SE**	**β**	**P value**
Sleep Onset Latency	**0.37**	**0.16**	0.35	**.027**	**0.04**	**0.01**	**0.44**	**.006**	**0.03**	**0.01**	**0.32**	**.042**
Covariates												
Sex	5.37	4.37	0.18	.227	−0.04	0.04	0.25	.356	0.40	0.32	0.19	.217
BMI	0.19	0.50	0.06	.698	0.57	0.25	−0.15	.093	0.01	0.04	0.06	.716

Note. B is the unstandardized coefficient regression coefficient. SE standard error. β is the standardized regression coefficient. R^2 = coefficient of determination shown for each model. **Bolded values are significant.**