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Original Article

Diabetes management mediates the association between sleep duration and glycemic control in youth with type 1 diabetes mellitus

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ABSTRACT

Objective/background: The purpose of this study was to examine the associations of diabetes management and sleep duration with glycemic control in youth with type 1 diabetes mellitus.
Patients/methods: 111 participants (mean age = 13.59 ± 2.11 years, 52.3% male, 50.5% non-white) wore actigraphy (average duration = 5.5 nights) and completed self-reported daily sleep diaries (average duration = 5.3 nights). Parents and participants each completed the Diabetes Management Scale (DMS) as part of a neurobehavioral evaluation. Glycated hemoglobin (HbA1c) and daily frequency of self-monitored blood glucose (SMBG) were collected from patient medical records.
Results: Youth with T1DM slept below the recommended amount of sleep for this age group (*M* = 7.45, *SD* = 0.74), which is approximately 9 h for school aged youth. They were in poor glycemic control with an average HbA1c of 9.11% (*SD* = 1.95) and their SMBG frequency was 4.9 (*SD* = 2.71). Average sleep duration from actigraphy was significantly correlated with average SMBG frequency and inversely related to HbA1c, indicating that less sleep was associated with worse management and glycemic control. When entered into a mediation model, diabetes management (SMBG frequency) completely mediated the relationship between sleep duration and glycemic control (HbA1c). Different sleep parameters of sleep quality, time to sleep, and sleep consistency also significantly correlated with HbA1c, SMBG, and parent and child-reports of various aspects of diabetes management. In particular, later bedtimes and a greater social jetlag predicted worse glycemic control.
Conclusions: In a sample of sleep deprived and poorly controlled youth with T1DM, diabetes management was an intermediary factor between sleep duration and glycemic control. Additional analyses of data supported circadian influences on glycemic control. These results highlight the importance of addressing sleep duration, quality, and consistency as part of routine diabetes management in this population.

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1. Introduction

Type 1 diabetes mellitus (T1DM) is an autoimmune condition that affects about one in every 400 children and adolescents [1]. Blood glucose is regulated through the administration of exogenous insulin based on healthcare providers' recommendations, results of frequent self-monitoring of blood glucose (SMBG), and adjustments for carbohydrate intake and physical activity. It is estimated that over two-thirds of youth with T1DM struggle to achieve adequate glycemic control and many experience volatile variations in glucose during adolescence [2]. The gold standard measure of glycemic control is glycated hemoglobin (HbA1c), of which less than 7.5% is considered to be in good control for pediatric patients. Across published studies, blood glucose monitoring via a glucometer contributes to about 30% of the variance in HbA1c levels [2]. Specifically, a study of younger adolescents demonstrated that one less daily SMBG predicted an increase of 1.26% in HbA1c values over a two-year time span [3]. Sleep deficiency may be one barrier to an individual being able to properly manage their diabetes and may have physiological impacts due to the counterregulatory hormones that maintain metabolic control during sleep.

Most attention has been given to glycemic control. However, several studies have not found a significant association between sleep duration and HbA1c in the pediatric population [4–7], with

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the exception of one study [8]. Nonetheless, a meta-analysis concluded that adults with shorter sleep (eg, 6–6.5 h) had significantly higher HbA1c compared to those who obtained the recommended amount of sleep [9]. Moreover, emerging evidence has suggested a potential association between sleep duration and diabetes management [4,5]. Thus, it is possible that the impact that sleep has on glucose levels is at least partially attributable its influences on diabetes management. Additionally, the amount of sleep may not be the only factor that influences disease control. Accordingly, some studies have shown that sleep quality and consistency (eg, keeping the same sleep schedule) also contribute to diabetes-related outcomes [4,6,7,10].

The purpose of this study was to fill a key gap in the literature by utilizing objective, multiday assessment of sleep via actigraphy to investigate the mediating role of diabetes management in the relations between sleep duration and glycemic control in a sample of youth with T1DM. It was hypothesized that shorter sleep duration would be associated with worse glycemic control. Furthermore, the relation between total sleep time (TST) and HbA1c was expected to weaken when the mediator of SMBG frequency was added to the model. Additionally, the study explored relations between objectively measured indicators of sleep quality, including sleep efficiency (SE), percent time awake (%Awake), and sleep onset latency (SOL), as well as metrics of sleep timing and consistency, including the standard deviation of TST (TST SD), sleep debt, bedtime, and social jet lag, with glycemic control and diabetes management.

2. Method

2.1. Participants

Participants (n = 111) were recruited from a pediatric endocrinology clinic, based in a large urban hospital in the southwest to participate in the Glucose Regulation and Neurobehavioral Effects of Sleep (GRANES) study. Inclusion criteria for participants in the larger study were: diagnosis of T1DM confirmed by medical records; between the ages of 10–16 years old; ability to speak and understand English; child assent; and at least one parent willing to participate. Participants were excluded if they had psychiatric, cognitive, genetic (eg, Down syndrome), or neurological conditions that would interfere with their ability to participate in the study or if they were hospitalized within one month prior to participation. Four participants did not have HbA1c values within three months of participating and an additional seven did not have SMBG frequency recorded in their medical records. Actigraphy was not available for one participant who took the device off after one day, but that same participant also did not have HbA1c or SMBG frequency data. All other participants averaged 5.5 nights (range 3–7 nights) of actigraphy data. Less than 12% had fewer than five nights due to scheduling closer to their evaluation (n = 7), device issues (n = 3), or watch removal without putting it back on during a sleep interval (n = 3).

2.2. Measures

As described previously [11], data collection included the assessment of sleep, glycemic control, diabetes management, other neurobehavioral measures such as standardized assessments and multi-informant psychosocial questionnaires. Only the measures relevant to the present study are included below.

2.2.1. Sleep duration

Participants wore a wrist actigraph (Actiwatch Spectrum, Philips Respironics) on their non-dominant hand to objectively measure their sleep. Algorithms from the company's software was utilized, each record was individually reviewed, and manual adjustments were made as needed. Instances that warranted adjustment included discrepancy between autoscored sleep onset when lights and activity were still apparent or when a sleep interval was not registered by the software. Participants also completed a daily sleep diary each morning for the duration of the study. Information provided on the sleep diary included: napping onset and duration, sleep onset and duration, night time awakenings frequency and duration, use of sleep medication, and self-reported sleep quality. Average sleep duration in minutes was calculated from sleep onset and wake time, subtracting any awakenings.

2.2.2. Sleep quality, timing, and consistency

Although sleep duration was the primary sleep parameter of interest, this study explored indicators of sleep quality, timing, and consistency. Using actigraphy, sleep quality was evaluated with SOL (time it took to fall asleep), SE (time slept/time in bed), and percent time awake after sleep onset (%Awake). Sleep timing and consistency were conceptualized with four metrics, the SD of TST, sleep debt (the absolute difference between TST on school nights and non-school nights), average bedtime, and social jetlag (the absolute difference between the average mid-point of the sleep period on school nights and the average mid-point on non-school nights) [7,12].

2.2.3. Glycemic control

HbA1c reflects the average blood glucose level over a three-month period and is regarded as the medical standard for evaluating diabetes control [13]. Participants' HbA1c values, derived from blood samples, were collected from medical records with values extracted if they occurred within three months prior to participation. If participant did not have pre-participation HbA1c values that met that criteria, HbA1c values within three months following participation were used.

2.2.4. Diabetes management

Diabetes management was assessed two ways. Average daily frequency of SMBG, which was obtained via participants' medical records, provided an objective metric of how often participants checked their blood glucose levels based on readings downloaded from blood glucose meters. The average number of SMBG is automatically computed in the software by dividing the number of blood glucose readings by the number of days during the recording period (eg, typically one month). The Diabetes Management Scale (DMS) was completed by participants and their parents as another indicator of diabetes management. Questions included the percentage of time the participant engaged in different management activities, including administering insulin (Insulin Subscale), engaging in safety behaviors (eg, alerting others of their diagnosis; Safety Subscale), becoming aware of physiological changes (Symptom Recognition Subscale), exhibiting sufficient autonomy (Supervision Subscale), monitoring their glucose (Blood Glucose Subscale), or exercising (Exercise Subscale). Scores on these subscales are derived by averaging the percentages for the items in the subscales, as well as a total score reflecting the average percentage of time spent engaging in different aspects of diabetes management (Table 2). The instrument has been found to have good reliability [14].

2.3. Procedures

Participants were recruited during regularly scheduled appointments at a pediatric endocrinology clinic. Each participant was enrolled in the GRANES study for up to two weeks, including approximately six days of baseline data collection and six days of intervention. Only data from the baseline week were utilized in the present study. Detailed information about the about recruitment efforts has been published previously [11]. Participants provided demographic information and were fitted with study devices (ie, Actiwatch). All participants were instructed to follow their typical schedule for one week by going to sleep and waking up as they normally would. They were provided with a sleep diary to record their daily sleep. Participants and their parent/guardian each completed the DMS. HbA1c and frequency of SMBG were collected from medical records. Parental consent and assent were obtained from parents and participants, respectively. All study procedures were approved by the Institutional Review Board at the University of Arizona.

2.4. Statistical analyses

Means and SDs were computed for continuous variables and frequencies for categorical variables. Bivariate association between sleep and diabetes management variables were assessed with a pairwise correlation matrix. A series of simple linear regression models predicting glycemic control (HbA1c) were conducted separately by sleep duration (TST) and diabetes management (SMBG frequency; parent- and self-report DSM subscales). Additionally, we evaluated diabetes management predicted by sleep duration. Other variables were evaluated for statistical significance as potential independent variables to be included in a multiple independent variable mediation model (ie age, sex, and parental income). However, these variables were not significant and were not kept in the models. We also ran these models with the exploratory sleep parameters of sleep quality (SOL, SE, %Awake), and sleep timing and consistency (bedtime, TST SD, and social jet lag). We used mediation analysis to test our hypothesis that adherence in diabetes management (ie, SMBG frequency) would mediate the relations between the independent variable of sleep duration (ie, average TST measured via actigraphy) and the dependent variable of glycemic control (ie, HbA1c). We conducted exploratory analyses using the same process with the metrics of sleep quality, timing, and consistency. Significance was determined at $p < 0.05$. All analyses were performed using SPSS Statistics version 25 (IBM, Armonk, NY, USA).

3. Results

3.1. Characteristics of the sample

3.1.1. Demographics

Participant characteristics are presented in Table 1. The sample was 52.3% male, with a mean age of 13.59 ± 2.11 years. Approximately half (50.5%) of participants identified as non-white, with 28.7% identifying as Hispanic/Latino, 0.9% as Black/African American, 0.9% as Asian, 3.7% as Native American/American Indian, and 15.8% as multiracial, which is comparable to the overall demographic characteristics of the clinic from which participants were recruited. Males and females did not differ on any diabetes outcome, with the exception of SMBG frequency, $t(97) = -2.34$, $p = 0.021$. Females had an average SMBG frequency of 5.58 ($SD = 3.06$), whereas males had an average SMBG frequency of 4.33 ($SD = 1.88$). With regard to sleep, males had a later bedtime $t(108) = 2.51$, $p < 0.014$. Age was significantly related with SMBG frequency, $r(97) = -0.47$, $p < 0.001$, but not with HbA1c, $r(107) = 0.12$, $p = 0.210$. Additionally, participants who were older had more difficulties falling asleep (SOL), $r(110) = 0.26$, $p = 0.007$, and staying asleep (%Awake), $r(110) = 0.22$, $p = 0.020$, lower SE, $r(110) = -0.33$, $p < 0.001$, shorter sleep duration, $r(110) = -0.35$, $p < 0.001$, and later bed times $r(110) = 0.25$, $p < 0.009$. Estimated median income as determined by census tract data using participants' zip codes only related to HbA1c, $r(107) = -0.26$, $p = 0.007$ and TST SD, $r(110) = -0.20$, $p = 0.003$.

Table 1
Demographic characteristics of the sample.

Variable	Mean (SD)
Age (years)	13.59 (2.11)
Male Sex (%)	52.3
Ethnicity/Race (%)	
White	49.5
Ethnic Minority	50.5
BMI Percentile (%)	69.75 (25.49)
Insulin Delivery Method	
On Pump (%)	65.8%
Multiple Dose Insulin (%)	32.4%
Tanner Stage	
Pre-pubertal (stages 1–2)	30.3
Early puberty (stages 3–4)	33.0
Late puberty (stage 5)	36.7
Mean Median Income	52,055 (19,088)
Self-Reported Highest Maternal Education Level (%)	
Less than high school	19.3
High school graduate	14.7
Some college/specialized training	31.2
Bachelors and above	34.9

Note. SD = standard deviation. Means (standard deviation) are provided for continuous variables.

3.1.2. Glycemic control and diabetes management

Diabetes-related characteristics of the sample are presented in Table 2. Participants had an average HbA1c of 9.11% ± 1.95%, which is considered poor diabetic control (>7.5%). The average SMBG frequency was 4.90 ± 2.71 (range 1–14 checks per day). Per the DMS, both parent and self-report indicated that youth adhered to the different aspects of diabetes management an average of 70.85% and 69.76% of the time, respectively.

Table 2
Diabetes and sleep related characteristics of the sample.

Variable	Mean (SD)
Duration of Diagnosis (years)	4.98 (3.61)
SMBG Frequency	4.90 (2.71)
DMS Self Report (%)	
Safety Subscale	59.89 (30.59)
Insulin Subscale	81.9 (13.98)
Blood Glucose Subscale	65.50 (23.42)
Exercise Subscale	63.29 (32.62)
Symptom Recognition Subscale	78.53 (13.81)
Supervision Subscale	66.73 (17.02)
Total Score	69.76 (12.47)
DMS Parent Report (%)	
Safety Subscale	59.89 (30.59)
Insulin Subscale	59.16 (30.10)
Blood Glucose Subscale	60.37 (24.66)
Exercise Subscale	63.21 (31.52)
Symptom Recognition Subscale	80.40 (12.76)
Supervision Subscale	64.30 (17.38)
Total Score	70.85 (12.87)
$HbA1_c$ (%)	9.11 (1.95)
Actigraphy Sleep Efficiency (%)	86.82 (3.39)
Actigraphy Sleep Percent Awake (%)	8.25 (2.42)
Actigraphy Sleep Onset Latency (mins)	15.44 (11.78)
Actigraphy TST (hours)	7.45 (0.74)
Actigraphy TST SD (mins)	1.07 (0.54)
Actigraphy Sleep Onset to Offset (hours)	15.45 (11.77)
Bedtime	22:34 (1:05)
Social Jetlag	1.12 (0.83)
Sleep Debt	75.36 (145.28)

Note. SMBG = self-monitored blood glucose. $HbA1_c$ = glycated hemoglobin. SD = standard deviation. TST = total sleep time. DMS = Diabetes Management Scale. Means (standard deviation) are provided for continuous variables.

3.1.3. Sleep parameters

With regard to sleep duration, participants' average TST was 7.45 ± 0.74 h (range 5.61–9.57 h), with 99.1% of participants sleeping less than 9 h based on actigraphy, which is considered insufficient sleep duration. Based on sleep diary, participants' average TST was 8.4 ± 0.89 h (range 5.57–10.69 h), with 76.6 percent sleeping less than 9 h. A one-sample t-test further supported that the sample slept significantly less than the recommended amount of sleep (ie, 9 h) based on both actigraphy, $t(110) = -21.97$, $p < 0.001$, and sleep diaries, $t(106) = -6.80$, $p < 0.001$, respectively. With regard to sleep quality, the sample had an average SOL of 15.44 ± 11.78 min, average %Awake during the night of 8.25 ± 2.42 min, and SE of 86.82 percent ± 3.39 percent. Participants' average TST SD and sleep debt were 1.09 h ($SD = 0.56$ h) and 1.26 h ($SD = 2.42$ h), respectively. Participants average bedtime was 10:34 PM ($SD = \pm1.12$ h), and they experienced a social jet lag of a little more than one hour (mean = 1.12; $SD = 0.83$).

3.2. Relations between sleep duration with diabetes management and glycemic control

3.2.1. Sleep duration

At the bivariate level, objectively-measured TST was significantly correlated with both HbA1c, $r(107) = -0.20$, $p = 0.036$ and SMBG frequency, $r(98) = 0.24$, $p = 0.015$. Consistent with prior literature, HbA1c was related to SMBG frequency, $r(96) = -0.34$, $p = 0.001$. Sleep duration, as measured by the sleep diary, did not significantly relate to either HbA1c or SMBG frequency. Less sleep related to a lower percentage of time engaging in safety behaviors per parent report, $r(109) = 0.22$, $p = 0.020$. Data from bivariate analyses are presented in Table 3.

When entered into a mediation analysis (presented in Fig. 1), diabetes management, as measured by SMBG frequency, completely mediated the relation between objectively-measured sleep duration and glycemic control (ie, HbA1c) [15]. Accordingly, for every additional hour of sleep, HbA1c was reduced by 0.33% and the youth engaged in almost one additional SMBG check (0.88). However, the relation between TST and HbA1c was no longer significant after controlling for the mediator (ie, SMBG frequency). The estimate of the indirect effect of sleep duration on HbA1c through SMBG frequency was −0.20 ($z = -1.91$, $p = 0.050$). Using bootstrap of 3000 samples with a 95% confidence interval, the effect was significant as the true indirect effect was estimated to lie between −0.48 and −0.013, which does not comprise the value of zero. Similar findings were derived when diabetes management was represented by the DMS-P Safety subscale. Accordingly, when entered into a mediation analysis, diabetes management fully mediated the relation between actigraphy TST and HbA1c.

3.3. Relations between sleep quality with glycemic control and diabetes management

With regard to glycemic control, in addition to sleep duration as noted above, a greater %Awake, $r(107) = 0.22$, $p = 0.024$, and SE, $r(107) = -0.22$, $p = 0.023$, significantly correlated with higher Hb1Ac. With regard to diabetes management, more time spent awake during the overnight sleep period significantly correlated with more supervision in diabetes self-care per parent report, $r(109) = 0.24$, $p = 0.014$, and less time exercising per parent report, $r(108) = -0.23$, $p = 0.018$. Lower SE significantly related to less time spent exercising per parent report, $r(108) = 0.26$, $p = 0.007$, and engaging in safety behaviors per parent report, $r(109) = 0.22$, $p = 0.020$. Data from bivariate analyses are presented in Table 3.

Table 3
Bivariate relations between sleep and diabetes.

	HbA1c	SMBG Frequency	Duration		Quality			Consistency			
			ACTI TST	DSD TST	SE	SOL	%Awake	Average TST SD	Bedtime	Social Jetlag	Sleep Debt
Actigraphy											
Average TST	−0.203*	−0.244**	–	–	–	–	–	–	–	–	–
Average TST SD	0.018	−0.175	–	–	–	–	–	–	–	–	–
SE	−0.219*	0.177	–	–	–	–	–	–	–	–	–
SOL	0.046	−0.090	–	–	–	–	–	–	–	–	–
%Awake	0.219*	−0.145	–	–	–	–	–	–	–	–	–
Sleep Diary TST	−0.013	0.166	–	–	–	–	–	–	–	–	–
Bedtime	0.276**	−0.330**	–	–	–	–	–	–	–	–	–
Social Jetlag	0.192*	−0.046	–	–	–	–	–	–	–	–	–
Sleep Debt	0.066	0.030	–	–	–	–	–	–	–	–	–
DMS Self Report											
Safety Subscale	−0.191*	0.309**	−0.067	0.049	−0.100	0.022	−0.101	−0.023	−0.115	0.053	−0.080
Insulin Subscale	−0.053	0.134	−0.042	0.001	−0.037	0.108	0.036	−0.065	−0.052	−0.014	−0.149
Blood Glucose Subscale	−0.217*	0.152	0.096	0.082	0.154	−0.055	−0.103	−0.134	−0.142	0.040	−0.101
Exercise Subscale	0.028	0.188	0.118	0.081	0.096	0.024	0.146	−0.072	−0.157	0.010	0.110
Symptom Recognition Subscale	0.283**	−0.152	−0.064	0.049	−0.071	−0.052	0.123	0.058	0.002	−0.041	0.077
Supervision Subscale	0.092	0.068	0.081	0.062	−0.022	−0.068	0.095	−0.117	−0.154	0.020	−0.062
Total Score	−0.034	0.208*	0.042	0.087	−0.037	0.002	−0.164	−0.160	−0.222*	−0.064	−0.129
DMS Parent Report											
Safety Subscale	0.350**	0.316**	0.223*	−0.015	0.223*	−0.105	−0.078	−0.204*	−0.241*	0.026	−0.178
Insulin Subscale	−0.273**	0.110	0.156	0.092	0.089	−0.015	0.054	0.111	−0.208	−0.084	0.055
Blood Glucose Subscale	−0.324**	0.319**	0.066	0.034	0.11	−0.001	-0.0.054	−0.105	−0.280**	−0.063	−0.055
Exercise Subscale	−0.085	0.278**	0.151	−0.074	0.259**	−0.054	−0.226*	0.056	−0.068	0.008	0.148
Symptom Recognition Subscale	0.065	−0.125	0.037	−0.217*	−0.095	0.000	0.180	0.064	−0.089	0.004	−0.034
Supervision Subscale	0.012	0.003	0.002	−0.154	−0.152	−0.003	0.235*	0.107	−0.163	−0.078	0.119
Total Score	−0.229*	0.234*	0.132	−0.141	0.087	−0.054	-0.036	0.001	−0.269**	−0.101	0.013

Note. SD = standard deviation. ACTI TST = average total sleep time per actigraphy. DSD TST = average total sleep time per daily sleep diary. SE = sleep efficiency. SOL = sleep onset latency. %Awake = percent time awake. HbA1c = glycated hemoglobin. DMS = Diabetes Management Scale. SMBG = self-management blood glucose. Data are Pearson correlation coefficients. * = $p \leq 0.05$ ** = $p \leq 0.01$.

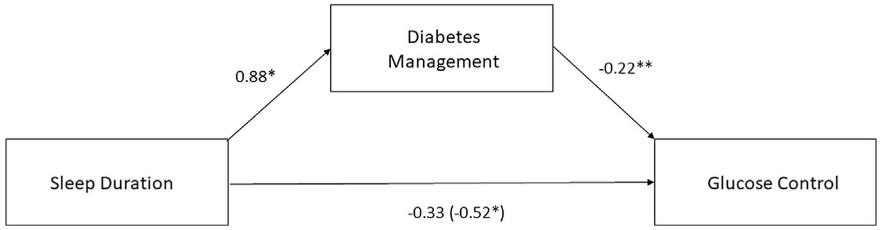

Fig. 1. Regression coefficients for the relationship between sleep duration and glucose control as mediated by diabetes management. The regression coefficient between sleep duration and glucose control is in parenthesis. *p<0.05, **p<0.01.

3.4. Relations between sleep consistency with glycemic control and diabetes management

Neither TST SD nor sleep debt significantly related to either glycemic control or diabetes management. Later bedtimes related to less frequent SBGM, $r(98), = -0.33$, $p = 0.001$, and higher HbA1c, $r(107), = 0.28$, $p = 0.004$. Average bedtimes were also significantly correlated with the total percent time spent in overall self-reported management, $r(110), = -0.22$, $p = 0.020$, and parents' reports of the percent time their child spent managing diabetes in the following areas on the DMS: Safety, $r(109), = -0.24$, $p = 0.012$; Insulin, $r(109), = -0.21$, $p = 0.030$; Glucose Monitoring, $r(109), = -0.28$, $p = 0.003$; and overall, $r(109), = -0.28$, $p = 0.005$. Since bedtime was significantly related to glucose and SMBG frequency, they were further evaluated in mediation analysis. Unlike the findings with TST, the data did not support that bedtime accounted for the effect on glycemic control through management. In other words, there was a direct effect of bedtime on HbA1c. Social jetlag significantly correlated with HbA1c, $r(106) = 0.19$, $p = 0.048$, with a greater discrepancy in school night and non-school night midsleep relating to worse glycemic control. However, social jetlag did not relate to any aspect of management.

4. Discussion

Using both diary and actigraphy, participants with T1DM slept below the recommended amount of sleep. In fact, actigraphy data showed that all but one of the participants had not achieved sufficient sleep duration. Further, the average HbA1c reflected that the majority of these youth have poorly controlled diabetes. Objectively measured sleep parameters were significantly associated with multiple aspects of diabetes management and glycemic control. Mediation analyses revealed that diabetes management served as an intermediary between sleep duration and glycemic control. In other words, the data supported the hypothesis that the SMBG frequency fully mediated the association between TST and HbA1c. Additionally, later bedtimes related to both glycemic control and diabetes management; however, SMBG frequency did not mediate the relations between bedtime and HbA1c. This study adds to the growing body of literature that support the cascading effects of sleep on multiple aspects of diabetes-related outcomes.

Consistent with prior research, SMBG frequency was related to HbA1c, supporting that self-management behaviors play a critical role in maintaining diabetes control [2,3]. The primary modality for assessing sleep duration in youth with T1DM has been self-report. Some recent research has begun to incorporate actigraphy to provide multi-day, objective assessment of sleep patterns in youth with T1DM [6,16]. Contrary to Patel et al. [6] the current study found a significant association between actigraphy TST and HbA1c, indicating that more sleep related to better glycemic control. Furthermore, their study also did not find a relationship between TST and diabetes self-management assessed via SMBG frequency or a diabetes self-care questionnaire, whereas the current study did find that the amount of sleep predicted the frequency of daily glucose monitoring and time spent engaging in safety behaviors per parent report. In fact, when taking into account diabetes management (eg, SMBG frequency, DMSParent Safety), the mediation models supported that increased sleep duration leads to better management, which in turn, leads to better glycemic control. These findings may suggest that inadequate sleep exerts behavioral effects on glycemic control such that longer sleep duration may relate to better glycemic control due to enhance management practices.

Sleep quality, such as more time spent awake during the sleep period and SE, was associated with HbA1c and various diabetes management behaviors, such as parent-reported supervision of diabetes self-care, safety behaviors, and less frequent exercising. These findings are consistent with previous research that found that self-reported sleep quality predicted blood glucose levels [4,10]. However, other research has not find a direct associated between sleep quality and glycemic control [6]. Sleep quality has also yielded mixed findings in the literature, with some studies not finding a relation with either SMBG frequency or self-reported management behaviors [4,6] and other research reporting significant associations [10]. In this study, although we did not find that objectively measured sleep quality correlated with SMBG frequency, we did find that it related to management behaviors. Sleep disruption could make it difficult to focus on managing diabetes as well as have a physiological effect on blood glucose. Future research should consider timing of when the awakenings are occurring and factors that contribute to the length of those awakenings. For example, awakenings during slow wave sleep, which predominates in the beginning of the night, may increase levels of sympathetic activation or accompany increased levels of cortisol [16]. Awakenings can also be indicative of arousals, which may be tied to sleep-disordered breathing. Additional research should examine how awakenings co-occur with glycemic excursions, such as found by Pillar and colleagues [17].

This study incorporated several metrics of sleep timing and consistency that have been reported in recent studies examining sleep in individuals with T1DM. With regard to inconsistent sleep duration, neither TST SD nor the difference between sleep duration on school and non-school nights (sleep debt) was associated with diabetes management or glycemic control. A recent study [6] found similar findings with regard to the TST SD not relating to scores on a self-care inventory as reported by either parents or youth. However, that study found that TST variability significantly related to both SMBG frequency and HbA1c, though that study did not examine the mediating role of diabetes management.

In the current study, sleep timing contributed to diabetes-related outcomes. Accordingly, later bedtimes positively associated with HbA1c, SMBG frequency, and overall management adherence as reported by both parents and youth, as well as specific management behaviors such as parent-reported safety behaviors, insulin adjustments, and glucose monitoring. The findings that bedtime continued to be directly associated with HbA1c in the mediation model, and the fact social jetlag related to HbA1c, support circadian influences on glycemic control. From a behavioral perspective, individuals with later bedtimes may engaged in other behaviors, such as late-night eating or sedentary activities, that compete with diabetes management and may contribute to elevated blood glucose levels. Physiologically, delayed bedtimes could impact the regulation of counterregulatory hormones, such as epinephrine, cortisol, and growth hormone. Bedtimes and the discrepancy in mid-sleep between school/work and non-school/work nights should be further explored given the mixed findings that have been yielded in the T1DM population [7,18]. These findings suggest that discussions regarding sleep should not only focus on increasing overall sleep duration, but also establishing earlier bedtimes and maintaining a consistent sleep period across the week. Future research needs to investigate the role of circadian rhythm in T1DM disease progression. Validating and examining additional metrics of sleep consistency in adolescents may also further elucidate the role of consistency in diabetes management and glycemic control. Accordingly, the sleep regularity index (SRI) is characterized by "the percentage probability of an individual being in the same state (asleep vs. awake) at any two time-points 24 h apart," (page 2) [19] whereas, the composite phase deviation (CPD) reflects similarity in sleep timing on a given day to 'typical' sleep [20].

Several limitations apply to the present study. First, actigraphy data were only collected over a one-week period. Some research has suggested that actigraphy overestimates sleep due to misclassifying motoric activity as awake [21]. However, multiple sleep manipulation studies have used accelerometers to document compliance. Furthermore, multiple published studies, including several studies that examined naturalistic sleep in relation to daytime functioning, as well as those that examined the effects of sleep restriction and extension have typically used 3–5 days of actigraphy recordings to assess sleep duration and changes in sleep [22–26]. Nonetheless, two weeks of actigraphy is recommended to capture circadian rhythm patterns and differences between school/work and non-school/work nights [26,27]. Second, this study relied on medical records to ascertain HbA1c and SMBG, rather than collecting these variables at the same time as the actigraphy. However, HbA1c, which is a measure of blood glucose levels over a three-month period, should have encompassed participants' glucose levels at the time of participation. Finally, this study did not specifically examine insulin administration in relation to sleep. Recent research has found several sleep parameters, such as duration and sleep consistency, to be related to bolus frequency and compliance [5,6]. Future research should investigate insulin requirements, as sleep may contribute to differential responses to exogenous insulin. In particular, experimental sleep manipulation studies such as the GRANES study [11], and those conducted by Beebe and colleagues [24,28,29] in naturalistic settings, would provide the opportunity to examine cause and effect relationships in ecologically valid contexts. However, in-lab or residential settings would allow for further examination of varying of doses of sleep duration, including prescribed bedtimes or variability of TST. Furthermore, this study utilized data from the baseline of a randomized control trial (RCT) in which all participants had T1DM to examine the efficacy of a sleep intervention to target diabetes management and glycemic control. Extant research, including our team's previously published findings, have examined sleep differences in youth with T1DM and those without diabetes [9,16].

In conclusion, the results of this study highlight the importance of assessing sleep in this population that appears to be at high risk for insufficient sleep duration. The effects of inadequate sleep may be more pronounced given the relationship between sleep and self-management behaviors necessary to maintain optimal glycemic control. Studies are needed to investigate the putative physiological and behavioral mechanisms underlying sleep's role in diabetes-related outcomes. Diabetes management indicators should be included as a key outcome of interventions designed to address sleep problems in this population. Furthermore, clinicians are encouraged to consider sleep when addressing diabetes management issues. Youth with T1DM should be educated on the importance of sleep and provided with sleep recommendations as part of treatment. Most critically, in order to have sleep be integrated into clinical care, the standards of diabetes management need to review and assess all published research on sleep in individuals with T1DM to determine the evidence for changing clinical care guidelines.

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