

Title: Sleep in Children with Type 1 Diabetes and their Parents in the T1D Exchange

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Short Title: Sleep in Children with Type 1 Diabetes

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Highlights

- Survey of sleep in children ages 2-12 with type 1 diabetes
- 67% of children met criteria for poor sleep quality
- Child sleep quality was related to glycemic control
- Sleep may be a modifiable factor to improve glycemic control

Abstract (Word Count: 248)

Objectives: Sleep has physiological and behavioral impacts on diabetes outcomes, yet little is known about the impact of sleep disturbances in children with type 1 diabetes. The current study sought to characterize sleep in children with type 1 diabetes and in their parents, and to examine the associations between child sleep, glycemic control and adherence, parent sleep and well-being, parental fear of hypoglycemia, and nocturnal caregiving behavior.

Methods: Surveys were emailed to parents of participants 2-12 years old in the T1D Exchange clinic registry. Clinical data were obtained from the registry for the 515 respondents.

Results: In our sample, 67% of children met criteria for poor sleep quality. Child sleep quality was related to glycemic control (HbA1c of 7.9% [63 mmol/mol] in children with poor sleep quality vs 7.6% [60 mmol/mol] in children with non-poor sleep quality; $P<0.001$) but not mean frequency of blood glucose monitoring (BGM) (7.6 times/day vs 7.4 in poor/non-poor quality; $P=0.56$). Associations were similar for sleep duration. Children with poor sleep quality were more likely to experience severe hypoglycemia (4% in children with poor sleep quality vs 1% in children with non-poor sleep quality; $P=0.05$) and more likely to experience DKA (7% vs 4%, respectively; $P<0.001$). Poorer child sleep quality was associated with poorer parental sleep quality, parental well-being, and fear of hypoglycemia ($P<0.001$ for all). Child sleep was not related to use of diabetes-related technology (CGM, insulin pump).

Conclusions: Sleep may be a modifiable factor to improve glycemic control and reduce parental distress.

Keywords: sleep quality; type 1 diabetes; glycemic control

Abbreviations: blood glucose monitoring (BGM), continuous glucose monitoring (CGM), diabetic ketoacidosis (DKA), Child Sleep Habits Questionnaire (CSHQ), Pittsburgh Sleep Quality Index (PSQI), Hypoglycemia Fear Survey (HFS-P), World Health Organization-Five-Wellbeing Index (WHO-5), severe hypoglycemia (SH), Research Electronic Data Capture (REDCap)

1.1 Introduction

Sleep has physiologic and behavioral impacts on many health outcomes. Sleep disturbances, which include bedtime resistance or difficulty initiating sleep, night wakings, and insufficient sleep (1), are highly prevalent in children, occurring in 20-30% of the general population (2), and a recent meta-analysis found that children and adolescents with type 1 diabetes had significantly shorter sleep duration than controls (3). For children with type 1 diabetes, sleep disruptions can be the result of night wakings due to hypo/hyperglycemia, as well as parental nocturnal diabetes caregiving behaviors (4). Nevertheless, sleep characteristics, such as total sleep time, sleep/wake times, and sleep quality, are not routinely addressed in standards of care for youth with type 1 diabetes (5, 6).

Accumulating evidence indicates that short sleep duration and poor quality sleep contribute to problems with glycemic control and adherence in adolescents and adults with type 1 diabetes (7, 8). The effect of sleep on glycemic control likely occurs through a direct physiological pathway (decreased insulin sensitivity) (9-12) and an indirect behavioral pathway (insufficient sleep compromises the cognitive functions needed to effectively manage diabetes) (13). One of the only studies to examine sleep in youth with type 1 diabetes used actigraphy and polysomnography and found that adolescents with type 1 diabetes (n=40, ages 10-16) spent less time in slow wave sleep than matched controls. Adolescents with reduced slow wave sleep had worse glycemic control and poorer self-reported quality of life (8). In this study, poor sleep habits also were associated with behavior problems and academic difficulties, but sleep was not examined in relation to adherence. In a more recent study of adolescents and young adults with type 1 diabetes (ages 13-20), shorter self-reported sleep duration was significantly associated with a lower frequency of blood glucose checks, and sleep quality was significantly related to

glycemic control for males but not females (14). In a smaller study of adolescents (n=45), sleep duration was significantly associated with adherence, measured with frequency of blood glucose monitoring (BGM) and insulin bolus (15). While these studies generally support the link between sleep disturbances and glycemic control, they have not examined the role of sleep on adherence to diabetes management in younger children, or the influence of diabetes-related technology, such as continuous glucose monitoring (CGM) or insulin pumps on sleep quality.

Characterizing sleep patterns and disturbances and their impacts in youth with type 1 diabetes will inform future studies designed to improve diabetes management and diabetes outcomes. Previous studies of sleep in type 1 diabetes have been limited by small sample sizes and narrow age ranges, and children with type 1 diabetes are likely to experience unique sleep disturbances related to nocturnal caregiving. We used validated survey measures to evaluate sleep patterns in youth enrolled in the T1D Exchange clinic registry and their parents. In addition, we examined the associations between sleep and diabetes outcomes (hemoglobin A1c [HbA1c], hypoglycemia, diabetic ketoacidosis [DKA]), use of diabetes-related technology (insulin pump, CGM), and BGM. Finally, we explored relationships between parental well-being, fear of hypoglycemia, nocturnal caregiving and children's sleep patterns.

2.1 Materials and Methods

2.2 Survey Components

We used parental surveys to characterize sleep patterns in youth 2-12 years of age with type 1 diabetes and their parents. We administered both the validated Child Sleep Habits Questionnaire (CSHQ) (16) and Pittsburgh Sleep Quality Index (PSQI) (17). The CSHQ is a parent-reported measure of child sleep habits and sleep disturbances, validated for use in ages 2-12 (18, 19). CSHQ scores range from 33 to 99, with higher scores indicating greater sleep

disturbance. A score of 41 or higher is considered clinically significant. The PSQI is a self-reported measure of sleep quality and was used to assess parental sleep quality. PSQI scores range from 0 to 21 with a higher score indicating poor sleep quality. A score greater than 5 indicates a clinically significant sleep disturbance.

In addition to capturing data regarding sleep patterns, we included validated measures for parental fear of hypoglycemia (Hypoglycemia Fear Survey [HFS-P]; range 0-104 with higher score indicating more fear) and parental emotional wellbeing (World Health Organization-Five-Wellbeing Index [WHO-5]; range 0-100 with high score indicating better well-being).

Nighttime caregiving habits also were captured from parents and reported as frequency per week of a caregiver checking the child's blood sugar after bedtime. Information regarding diabetes history and demographic variables (gender, race/ethnicity, parent education, annual household income, and insurance status) were also obtained from the parent. Parents were asked to report on most recent HbA1c value, episodes of severe hypoglycemia (SH), and episodes of DKA over the previous 3 months.

2.3 T1D Exchange Clinic Registry Data

In addition to self-reported survey data, supplementary demographic and clinical data were obtained from T1D Exchange clinic registry medical chart extraction for survey respondents and non-respondents. Demographic T1D Exchange variables used to compare respondents and non-respondents included the following: age, sex, race/ethnicity, health insurance type, use of insulin pump, CGM use, most recent HbA1c, and age at type 1 diagnosis.

Self-reported HbA1c values were used for the main respondent analysis because a significant number of respondents did not have clinic-reported HbA1c values within 6 months of survey administration. However, a sub-analysis of participants who had recent T1D Exchange

clinical HbA1c data (93 (18%) participants) revealed that self-reported HbA1c was only slightly lower than clinic-reported values (median difference 0.1% (inter-quartile range -0.2% to 0.5%)). Further, the difference in self-reported and clinic-reported HbA1c was not associated with CSHQ score or PSQI score ($P=0.76, 0.19, 0.98$, respectively, from separate linear regression models).

2.4 Survey Administration

The T1D Exchange Clinic Network has enrolled over 30,000 individuals with type 1 diabetes across 74 U.S.-based pediatric and adult endocrinology practices. Details on the eligibility criteria, informed consent process, and data collection for the T1D Exchange Clinic Network have been previously published (20). Parents of T1D Exchange clinic registry participants 2-12 years of age with type 1 diabetes duration of at least one year who consented to receive emailed information regarding T1D Exchange studies were invited to participate via 2 email waves between June 2015 and September 2015. After signing an institutional review board-approved electronic informed consent form, parents of eligible participants completed the online surveys. Study data were collected and managed using Research Electronic Data Capture (REDCap) tools hosted at the Jaeb Center for Health Research (21). REDCap is a secure web-based application designed to support data capture for research studies, providing; 1) an intuitive interface for validated data entry, 2) audit trails for tracking data manipulation and export procedures, 3) automated export procedures for seamless data downloads to common statistical packages, and 4) procedures for importing data from external sources. Survey respondents received their choice of a \$20 electronic gift card or donation to a diabetes charity.

2.5 Statistical Analysis

The study was designed to include a feasibility sample (not based on statistical principles) of 500 participants. Summary statistics were calculated for duration of sleep

according to age group (2-4 years old, 5-12 years old, parents) and for CSHQ score. The proportion of CSHQ scores meeting the definition of poor sleep quality (score >41) was calculated. The association between each of the following parent-reported outcomes and CSHQ score were examined: most recent HbA1c, frequency of BGM, use of insulin pump, CGM use, WHO-5 score, PSQI score, occurrence of ≥ 1 SH events in the 3 months prior to questionnaire completion, and occurrence of ≥ 1 DKA events in the 3 months prior to questionnaire completion. For continuous outcomes (HbA1c, frequency of BGM, WHO-5 score and PSQI score), separate multivariable linear regression models were used to assess the association with continuous CSHQ score; for categorical outcomes (insulin modality, CGM use, occurrence of ≥ 1 SH events, and occurrence of ≥ 1 DKA events), separate multivariable logistic regression models were used to assess the association with CSHQ score. The associations between CSHQ (outcome) and parental fear of hypoglycemia and nocturnal caregiver behavior were assessed through separate multivariable linear regression models. The association between parental nocturnal caregiver behavior and parental fear of hypoglycemia was examined through an ordinal logistic regression model. Methods assessing association between outcomes and CSHQ score were repeated to assess the association between outcomes and parental report of child sleep duration.

Summary statistics of the PSQI score were calculated, and the proportion of PSQI scores meeting the definition of poor sleep quality (score >5) was determined. The association between PSQI score and parental fear of hypoglycemia and nocturnal caregiver behavior was assessed through separate multivariable linear regression models.

Results are expressed as mean \pm Standard Deviation for normally distributed variables or median (interquartile range) for non-normally distributed variables. To account for possible confounding, the following covariates were assessed for association with each outcome through

univariate analysis and selection models: race/ethnicity, age, sex, age at T1D diagnosis, insurance status, clinic center, and insulin modality (assessed if insulin modality was not the outcome of interest). If an association with an outcome was present, the covariate was included in the model for the outcome.

Data analyses were performed using SAS software version 9.4 (SAS Institute Inc., Cary, NC). In view of the multiple comparisons, only p-values <0.01 were considered significant.

3.1 Results

Between June 2015 and September 2015, 2,356 parents of eligible participants from 54 pediatric diabetes centers were sent a survey invitation via email; 598 (25%) accessed the survey and 515 (22%) from 50 of the 54 clinics completed the survey (all who completed the survey were analyzed).

The 515 surveys were completed by parents of children who had a mean age of 9 ± 3 years, 240 (47%) children were female, and 442 (86%) non-Hispanic White. Mean age of the child at diagnosis was 4 ± 2 years. Mean parent-reported HbA1c was $7.8\%\pm 0.9\%$ (62 ± 9.8 mmol/mol); median number of blood glucose checks per day was 7.0 (6.0, 9.0). Occurrence of at least one SH event in the 3 months prior to questionnaire completion was reported by parents of 15 children (3%), and occurrence of at least one DKA event was reported by parents of 30 children (6%). Additional characteristics of the cohort are shown in Table 1.

Compared with participants who did not complete the survey, participants who completed the survey were more likely to be non-Hispanic White (86% vs. 78%), use an insulin pump (77% vs. 68%), use a CGM (27% vs. 19%), and have lower clinic-reported HbA1c (mean 8.0% vs 8.4% [64 vs 68 mmol/mol]) (Supplemental Table 1).

3.2 Summary of Scores

Mean duration of sleep per night was 10.9 ± 1.2 hours in children 2-4 years old, 9.5 ± 1.0 hours in children 5-12 years old (22), and 6.5 ± 1.2 hours in parents (Supplemental Figure 1). Among the 511 children with sleep duration reported, 103 (20%) reported sleep duration below the recommended amount (<9 hours/night); among 504 parents with sleep duration

reported, 259 (51%) reported sleep duration below the recommended amount (<7 hours/night).

Three hundred forty-six (67%) children met criteria for poor sleep quality (CSHQ score >41). Among the 501 parents with PSQI score measured, 266 (53%) met criteria for poor sleep quality (score >5). About one-third of parents (163 [32%]) met the WHO-5 criteria for low mood (score \geq 50) (23). Most participants (355 [69%]) often or always had a caregiver check the child's blood glucose value after the child's bedtime; only 17 (3%) never checked the child's blood glucose value after bedtime.

3.3 Associations with CSHQ

Children with poor sleep quality (high CSHQ score) had higher HbA1c than children with non-poor sleep quality ($P<0.001$ for continuous score adjusted for race/ethnicity, insurance status, and clinic center) (Figure 1A). Frequency of BGM per day was not associated with child sleep quality ($P=0.56$ adjusted for race/ethnicity, insurance status, diagnosis age, insulin modality, and clinic center). Children with poor sleep quality were more likely to experience at least one SH event in the 3 months prior to questionnaire completion ($P=0.05$ adjusted for race/ethnicity, gender, type of insurance, and HFS score; Figure 1B) and were more likely to experience at least one DKA event in the 3 months prior to questionnaire completion ($P<0.001$ adjusted for race/ethnicity, type of insurance, and use of an insulin pump; Figure 1C). Poor child sleep quality was associated with worse parental wellbeing ($P<0.001$ adjusted for race/ethnicity and child age; Figure 1D) and worse parental sleep quality ($P<0.001$ adjusted for race/ethnicity and sex; Figure 1E). Insulin modality was not associated with child sleep quality ($P=0.13$ adjusted for race/ethnicity, sex, insurance status, and diagnosis age); use of CGM also was not associated with child sleep quality

($P=0.96$ adjusted for race/ethnicity, child age, sex, insurance status, age at diagnosis, and insulin modality).

Children of parents with more fear of hypoglycemia (high HFS score) had worse sleep quality than children of parents with less fear of hypoglycemia ($P<0.001$ adjusted for race/ethnicity, child age, insurance status, clinic center; Figure 2A). Parents with more fear of hypoglycemia were more likely to more frequently check their child's blood glucose level after the child's bedtime ($P=0.005$ adjusted for race/ethnicity, child age, age at diagnosis, and insulin modality). However, child sleep quality was not associated with frequency of nocturnal checking of blood glucose ($P=0.66$; Figure 2A).

3.4 Associations with Parental Report of Child Sleep Duration

Associations with sleep duration were similar to associations with child sleep quality. Participants with shorter sleep duration had: higher HbA1c (8.0% in participants with <9 hours/night vs 7.8% in participants with ≥ 9 hours/night; adjusted $P=0.02$), worse parental wellbeing (WHO score 54 for participants with <9 hours/night vs 58 with ≥ 9 hours/night; adjusted $P=0.01$), and worse parental sleep quality (PSQI score 7 in participants with <9 hours/night vs 6 in participants with ≥ 9 hours/night; adjusted $P=0.01$). Sleep duration was not associated with frequency of blood glucose checks (adjusted $P=0.66$) or CGM use ($P=0.31$), however, there was a trend for participants with longer duration of sleep to be more likely to use an insulin pump (68% in participants with <9 hours/night vs 82% in participants with ≥ 9 hours/night; adjusted $P=0.06$).

3.4 Associations with PSQI

Similar to child sleep quality, parents with more fear of hypoglycemia had worse sleep quality than parents with less fear of hypoglycemia ($P<0.001$ adjusted for race/ethnicity and

sex; Figure 2B). However, frequency of nocturnal checking of blood glucose was not associated with parental sleep quality ($P=0.35$; Figure 2B).

4.1 Discussion

The current study is the largest descriptive study of sleep in children with type 1 diabetes and their parents and yielded several important findings. A significant percentage of children and parents met clinical criteria for sleep disturbances. Further, 20% of children and 50% of parents did not meet the recommended duration for sleep. Child sleep disturbances were significantly associated with glycemic control, such that those who met the clinical criteria for sleep disturbances had significantly poorer glycemic control than those who did not have clinically significant sleep disturbances (parent-reported HbA1c=7.9% vs 7.6% [63 vs 60 mmol/mol]), and children with poor sleep quality were more likely to have experienced at least one event of SH and DKA. In addition, child sleep disturbances were significantly related to parents' own reported sleep quality and parental well-being.

In line with our hypothesis, child sleep quality and duration were significantly related to HbA1c. While the relationship between sleep and glycemic control is likely bidirectional, experimental studies offer support for the effect of sleep restriction or disturbances to sleep quality (suppressing slow wave sleep) on insulin resistance and elevated blood glucose (9, 11, 12). However, contrary to our hypothesis, we did not find a significant association between child sleep and a measure of adherence (frequency of BGM). Given that parents are primarily responsible for diabetes management in younger children, it may be that associations between sleep disturbances and adherence will be evident in older individuals, who engaged in more self-management (adolescents and adults). Further, as we only assessed one adherence behavior, it remains to be determined whether other adherence behaviors (e.g., mealtime

insulin bolus, corrections for hyperglycemia) are associated with sleep quality or sleep duration in children.

Child sleep also was associated with parent sleep and parental well-being, in line with findings from smaller studies of sleep in young children with type 1 diabetes (4, 24). Child sleep disturbances are a source of stress for parents, and parents' own lack of sleep and poor sleep quality are likely to have a negative impact on their emotional wellbeing (25). Even though parents' fear of hypoglycemia was associated with greater nocturnal caregiving and poorer sleep quality in children, there was no association between nocturnal caregiving and child sleep. Further, the lack of association between CGM use and parental or child sleep quality was surprising, given that many parents express interest in using CGM as a way to reduce anxiety around nocturnal hypoglycemia and reduce the need for nocturnal caregiving. It may be that objective measures of sleep are needed to demonstrate the impact of CGM on sleep, rather than self-report (26). Alternatively, it is possible that parents with the greatest fear of hypoglycemia were early adopters of CGM, but given the cross-sectional design of the study, we cannot know if fear of hypoglycemia changed with CGM use. Finally, the potential for alterations in insulin delivery based on CGM data (i.e., closed loop systems) may have greater potential to improve parents' sleep quality than data alone.

The current study is limited by the use of parent-report measures of child sleep and HbA1c and the cross-sectional design. Future studies should use more objective measures of sleep (actigraphy, polysomnography), longitudinal designs, and consider including a matched sample of children without diabetes. It is also important to note that the HFS-P was validated for use in children ages 8-15, and our sample included children ages 2-12. In addition, given the demographic and clinical differences in parents who completed the survey as compared to

the total clinic registry sample, findings may not generalize to other samples with lower use of diabetes-related technology or poorer glycemic control. However, this study was the first to examine sleep in a large, nationally distributed sample of young children with type 1 diabetes.

5.1 Conclusions

Sleep is an understudied and potentially important influence on glycemic control, and sleep quality may be a viable target for interventions to improve outcomes in youth with type 1 diabetes. In children without diabetes, behavioral interventions have resulted in significant improvements in sleep disturbance (2); a recent study demonstrated that an increase of even 15-20 minutes of sleep was associated with an additional BGM check and additional insulin bolus (15). Further, sleep disturbances observed in young children are likely to persist into adolescence, so it may be important to intervene at an early age (27). Given that most children with type 1 diabetes are not meeting treatment goals (28), and that parents of children experience high levels of distress related to diabetes management (29), the current study offers support for targeting sleep as a potentially modifiable risk factor for these outcomes in children with type 1 diabetes.

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Contributors' Statement

Sarah S. Jaser: Dr. Jaser contributed to data interpretation and wrote and edited the manuscript, and approved the final manuscript as submitted.

Nicole C. Foster: Ms Foster performed statistical analysis, wrote and edited the manuscript, and approved the final manuscript as submitted.

Bryce A. Nelson: Dr. Neslon contributed to data interpretation, discussion, reviewed/edited the manuscript, and approved the final manuscript as submitted.

Julie M. Kittelsrud: Dr. Kittelsrud contributed to data interpretation, discussion, reviewed/edited the manuscript, and approved the final manuscript as submitted.

Linda A. DiMeglio: Dr. DiMeglio contributed to data interpretation, discussion, reviewed/edited the manuscript, and approved the final manuscript as submitted.

Maryanne Quinn: Dr. Quinn contributed to data interpretation, discussion, reviewed/edited the manuscript, and approved the final manuscript as submitted.

Steven M. Willie: Dr. Willi contributed to data interpretation, discussion, reviewed/edited the manuscript, and approved the final manuscript as submitted.

Jill H. Simmons: Dr. Simmons contributed to data interpretation, discussion, reviewed/edited the manuscript, and approved the final manuscript as submitted.

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A listing of the T1D Exchange Clinic Network sites with participating principal investigators (PI), co-investigators (I) and coordinators (C) ordered by the number of participants recruited per site as of August 1, 2012 is included below:

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Stacy Hurst (C); Sarah Kissel (C); Carol Recklein (C) **Iowa City, IA University of Iowa Children's Hospital** (n=327) Eva Tsalikian (PI); Michael Tansey (I); Joanne Cabbage (C); Julie Coffey (C); Sarah Salamati (C) **Kansas City, MO Children's Mercy Hospital** (n=323) Mark Clements (PI); Sripriya Raman (I); Angela Turpin (I); Jennifer Bedard (C); Cyndy Cohoon (C); Aliza Elrod (C); Amanda Fridlington (C); Lois Hester (C); **Detroit, MI Henry Ford Health System** (n=316) Davida Kruger (PI); Andreana Tassopoulos **Gainesville, FL University of Florida** (n=306) Desmond Schatz (PI); Michael Clare-Salzler (I); Kenneth Cusi (I); Colleen Digman (I); Becky Fudge (I); Mike Haller (I); Collette Meehan (I); Henry Rohrs (I); Janet Silverstein (I); Sujata Wagh (I); Miriam Cintron (C); Eleni Sheehan (C); Jamie Thomas (C) **Orange, CA Children's Hospital of Orange County** (n=305) Mark Daniels (PI); Susan Clark (I); Timothy Flannery (I); Nikta Forghani (I); Ajanta Naidu (I); Christina Reh (I); Peggy Scoggin (I); Lien Trinh (I); Natalie Ayala (C); Rebeca Quintana (C); Heather Speer (C) **Columbus, OH Central Ohio Pediatrics Endocrinology and Diabetes Services** (n=303) William Zipf (PI); Diane Seiple (C) **Sioux Falls, SD Avera Research Institute** (n=281) Julie Kittelsrud (PI); Ashutosh Gupta (I); Vikki Peterson (C); Ashley Stoker (C) **San Diego, CA University of California** (n=280) Michael Gottschalk (PI); Marla Hashiguchi (C); Katheryn Smith (C) **Tampa, FL University of South Florida Diabetes Center** (n=276) Henry Rodriguez (PI); Craig Bobik (C); Danielle Henson (C) **Nashville, TN Vanderbilt Eskind Diabetes Clinic** (n=276) Jill Simmons (PI); Amy Potter (I); Margo Black (C); Faith Brendle (C) **Cleveland, OH Case Western Reserve University** (n=251) Rose Gubitosi-Klug (PI); Beth Kaminski (I); Susan Bergant (C); Wendy Campbell (C); Catherine Tasi (C) **Oklahoma City, OK University of Oklahoma Health Sciences Center Dept. of Pediatric Diabetes and Endocrinology** (n=243) Kenneth Copeland (PI); Joni Beck (I); Joane Less (C); Jill Schanuel (C); Jennifer Tolbert (C) **San Francisco, CA University of California, San Francisco Medical Center (UCSF)** (n=237) Saleh Adi (PI); Andrea Gerard-Gonzalez (I); Stephen Gitelman (I); Nassim Chettout (C); Christine Torok (C) **Seattle, WA Seattle Children's Hospital** (n=226) Catherine Pihoker (PI); Joyce Yi-Frazier (I); Susan Kearns (C) **Pittsburgh, PA Children's Hospital of Pittsburgh of UPMC** (n=217) Ingrid Libman (PI); Vicky Bills (C); Ana Diaz (C); Julie Duke (C) **Minneapolis, MN University of Minnesota** (n=204) Brandon Nathan (PI); Antoinette Moran (I); Melena Bellin (I); Shannon Beasley (C); Anne Kogler (C); Janice Leschyshyn (C); Kara Schmid (C); Anne Street (C) **Greenville, SC Greenville Hospital System Pediatric Endocrinology** (n=196) Bryce Nelson (PI); Carrie Frost (C); Erin Reifeis (C) **Houston, TX Baylor College of Medicine / Texas Children's Hospital** (n=187) Morey Haymond (PI); Fida Bacha (I); Maria Caldas-Vasquez (I); Sara Klinepeter (I); Maria Redondo (I); Rosa Berlanga (C); Teresa Falk (C); Elizabeth Garnes (C); Janette Gonzalez (C); Cecilia Martinez (C); Mariam Pontifes (C); Ronald Yulatic (C) **Ocean Springs, MS The Diabetes Center, PLLC** (n=187) Kathleen Arnold (PI); Traci Evans (I); Sharon Sellers (C) **Salt Lake City, UT University of Utah - Utah Diabetes Center** (n=181) Vandana Raman (PI); Carol Foster (I); Mary Murray (I); Vandana Raman (I); Trina Brown (C); Hillarie Slater (C); Karen Wheeler (C) **Worcester, MA University of Massachusetts Medical School** (n=179) David Harlan (PI); Mary Lee (I); John-Paul Lock (I); Celia Hartigan (C); Lisa Hubacz (C) **Durham, NC University of North Carolina Diabetes Care Center** (n=179) John Buse (PI); Ali Calikoglu (I); Joseph Largay (I); Laura Young (I); Helen Brown (C); Vinnie Duncan (C); Michelle Duclos (C); Julie Tricome (C) **Sioux Falls, SD Sanford Research/USD** (n=178) Verdayne Brandenburg (PI); Julie Blehm (I); Julie Hallanger-Johnson (I); Dawn Hanson (C); Corliss Miller (C); Jennifer Weiss (C) **Columbus, OH The Research Institute at Nationwide Children's Hospital** (n=168) Robert

Hoffman (PI); Monika Chaudhari (I); David Repaske (I); Elizabeth Gilson (C); Jesse Haines (C) **Billings, MT St. Vincent Healthcare/Internal Medicine and Diabetes** (n=165) Justen Rudolph (PI); Charles McClave (I); Doris Biersdorf (C) **Bismarck, ND Medcenter One** (n=156) Anthony Tello (PI); Julie Blehm (I); Donna Amundson (C); Rhonda Ward (C) **Philadelphia, PA University of Pennsylvania School of Medicine/Rodebaugh Diabetes Center** (n=156) Michael Rickels (PI); Cornelia Dalton-Bakes (C); Eileen Markman (C); Amy Peleckis (C); Nora Rosenfeld (C) **Cincinnati, OH Cincinnati Children's Hospital Medical Center** (n=148) Lawrence Dolan (PI); Sarah Corathers (I); Jessica Kichler (I); Holly Baugh (C); Debbie Standiford (C) **Spokane, WA Rockwood Research Center, P.S.** (n=132) Jeanne Hassing (PI); Jennifer Jones (I); Stephen Willis (I); Stephen Willis (I); Carol Wysham (I); Lisa Davis (C) **Baltimore, MD Johns Hopkins University Pediatric Endocrinology** (n=120) Scott Blackman (PI); Kimber-Lee Abel (C); Loretta Clark (C); Andrea Jonas (C); Ellie Kagan (C) **Miami, FL University of Miami, Diabetes Research Institute** (n=119) Jay Sosenko (PI); Carlos Blashke (C); Della Matheson (C) **Rapid City, SD Regional Health Clinical Research** (n=118) Rachel Edelen (PI); Thomas Repas (I); Denise Baldwin (C); Trista Borgwardt (C); Christina Conroy (C); Kelly DeGrote (C); Rod Marchiando (C); Michelle Wasson (C) **Jacksonville, FL Nemours Children's Clinic** (n=116) Larry Fox (PI); Nelly Mauras (I); Ligeia Damaso (C); Kim Englert (C) **Cleveland, OH Cleveland Clinic Department of Endocrinology, Diabetes and Metabolism** (n=111) Marwan Hamaty (PI); Laurence Kennedy (I); Michelle Schweiger (I); Pantelis Konstantinopoulos (C); Carolyn Mawhorter (C); Amy Orasko (C); Denise Rose (C) **Tallahassee, FL Tallahassee Memorial Diabetes Center** (n=108) Larry Deeb (PI); Kim Rohrbacher (C) **Findlay, OH Blanchard Valley Medical Associates** (n=100) Leroy Schroeder (PI); Amanda Roark (C) **Milwaukee, WI The Medical College of Wisconsin/ Children's Hospital of WI** (n=99) Omar Ali (PI); Joanna Kramer (C); Donna Whitson-Jones (C) **Nashville, TN Vanderbilt Eskin Diabetes Clinic** (n=98) Amy Potter (PI); Margo Black (C); Faith Brendle (C) **Vallejo, CA Kaiser Permanente** (n=74) Heidi Gassner (PI); Sobha Kollipara (I); Vicky Bills (C); Julie Duke (C) **Paterson, NJ St. Joseph's Children's Hospital** (n=53) Katerina Harwood (PI); Vijaya Prasad (I); Judy Brault (C)

Table 1. Parent-Reported Characteristics

	Overall N=515
Child Characteristics	
Age (years) - <i>mean ± SD</i>	9 ± 3
Age at diagnosis (years) – <i>median (25th %ile, 75th %ile)</i>	3 (2,5)
Female ^a – <i>n(%)</i>	240 (47%)
Race/ethnicity – <i>n(%)</i>	
White Non-Hispanic	442 (86%)
Black Non-Hispanic	20 (4%)
Hispanic or Latino	16 (3%)
Other Race/Ethnicity	37 (7%)
Insurance status ^a – <i>n(%)</i>	
Private insurance	408 (81%)
Other insurance	90 (18%)
No insurance	3 (<1%)
Insulin modality ^a – <i>n(%)</i>	
Insulin pump	403 (80%)
Multiple daily insulin injections	103 (20%)
Uses a Continuous Glucose Monitor – <i>n(%)</i>	199 (39%)
Parent-reported Most Recent HbA1c – <i>mean ± SD</i>	7.8% ± 0.9%

Frequency of monitoring of blood glucose – <i>mean ± SD</i>	7.4 ± 2.4
Occurrence of ≥1 severe hypoglycemia events ^{ab}	15 (3%)
Occurrence of ≥1 DKA events ^b	30 (6%)
Parent Characteristics	
Education level ^{ab} – <i>n(%)</i>	
High school diploma or less	134 (26%)
Vocational or college degree	255 (50%)
Graduate or professional degree	124 (24%)

^a1 transgender participant; education level missing for 2 participants; insurance status missing for 14 participants; insulin modality missing for 9 participants; frequency of self-monitoring of blood glucose missing for 1 participant; occurrence of severe hypoglycemia event information missing for 2 participants

^bAt least one event experienced in the 3 months prior to questionnaire completion

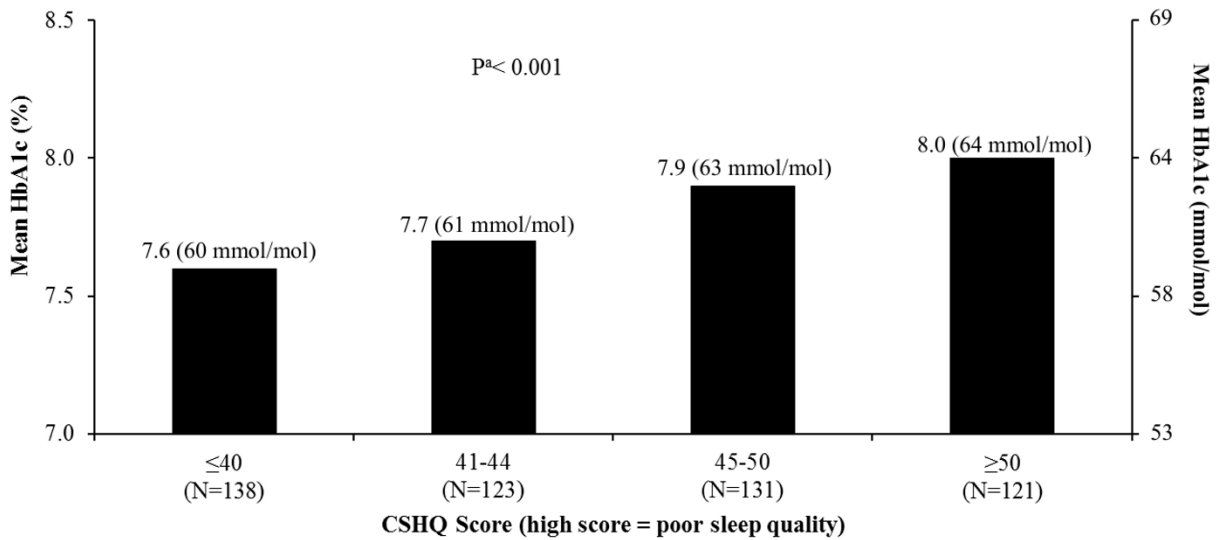
^cHighest parental education level

Table 2. Summary of Scores

	Overall N=515
CSHQ (child sleep disturbance) – <i>mean ± SD</i> Score >41 (poor sleep quality) – <i>n</i>(%)	45.8 ± 7.7 346 (67%)
PSQI (parental sleep quality) ^a – <i>mean ± SD</i> Score >5 (poor sleep quality) – <i>n</i>(%)	6.6 ± 3.4 266 (53%)
Hypoglycemia Fear – <i>mean ± SD</i>	43.2 ± 12.8
WHO-5 – <i>mean ± SD</i>	57.4 ± 17.5
Score ≥50 (low mood) – <i>n</i>(%)	163 (32%)
Frequency of nocturnal checks – <i>n</i> (%)	
Never	17 (3%)
Sometimes	143 (28%)
Often/always	355 (69%)

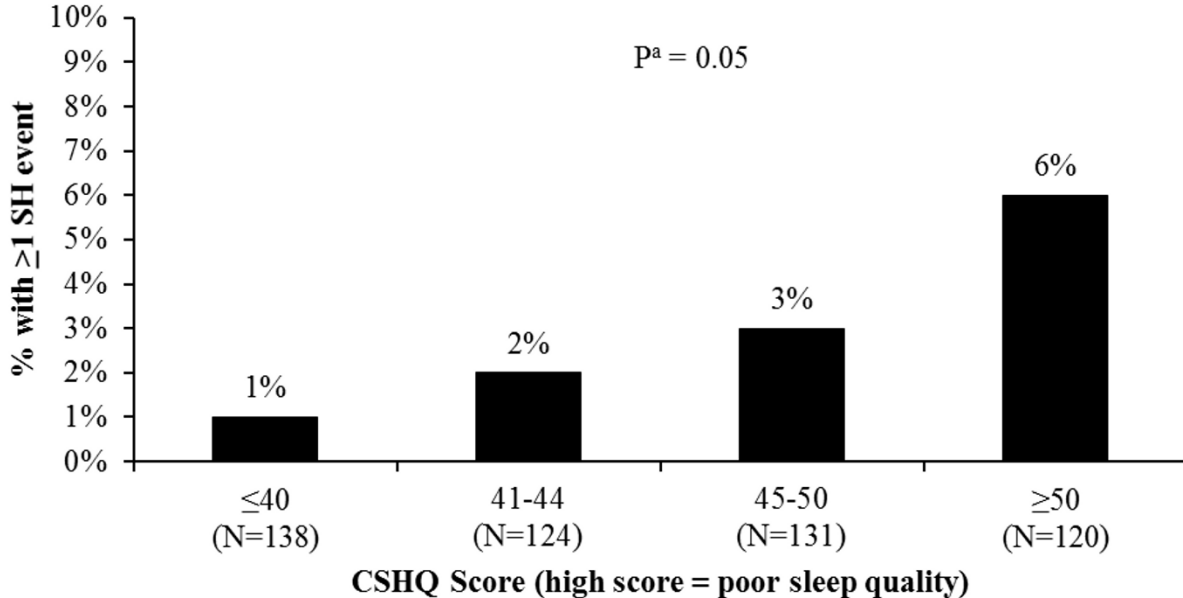
^aPSQI score missing for 14 participants

Figure 1A. Association between Parent-Reported HbA1c and Child Sleep Disturbances (CSHQ)



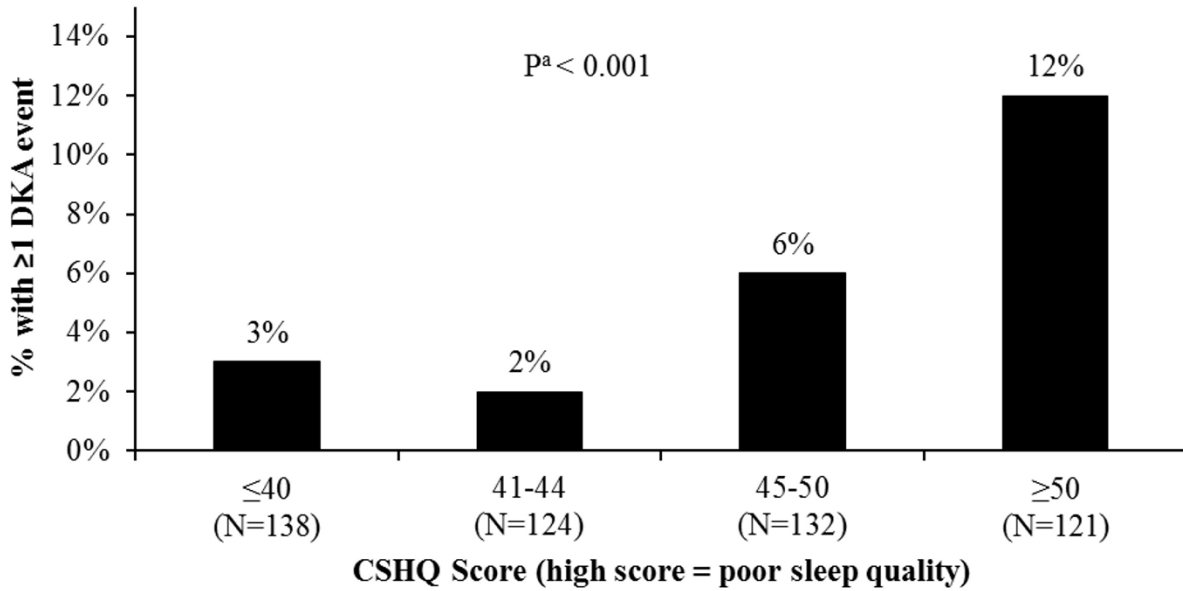
^aP-value calculated from linear regression model adjusting for clinic site, race/ethnicity, and insurance status

Figure 1B. Association between Occurrence of ≥ 1 Severe Hypoglycemia Events in the 3 Months Prior to Questionnaire Completion and Child Sleep Disturbances (CSHQ)



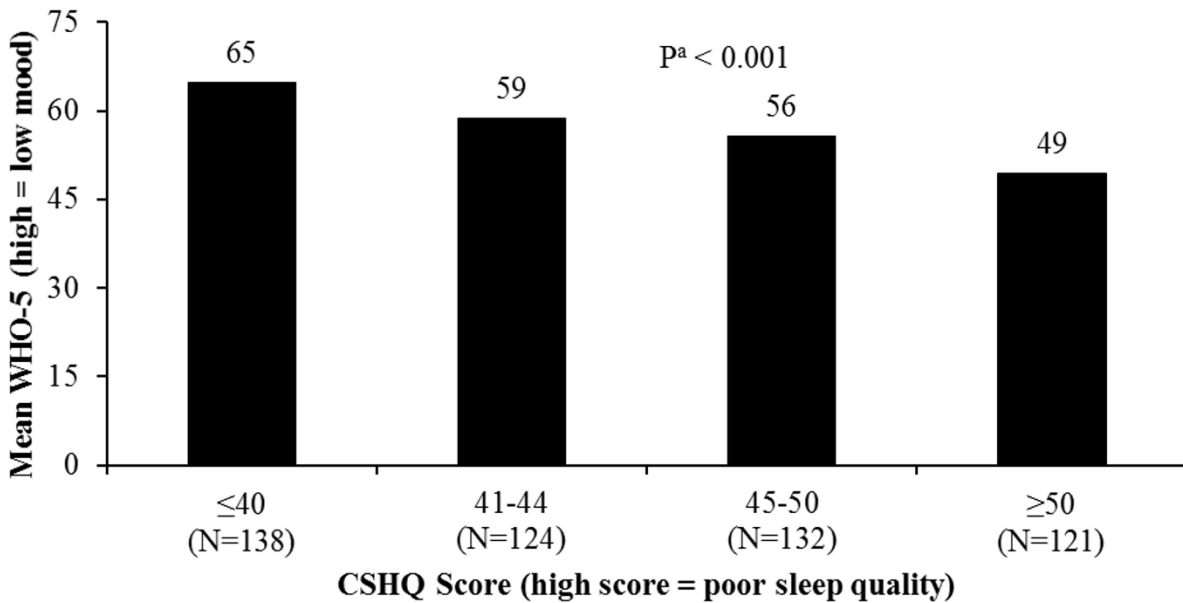
^aP-value from a multivariable logistic regression model adjusting for race/ethnicity, sex, type of insurance, and fear of hypoglycemia score (HFS survey score)

Figure 1C. Association between Occurrence of ≥ 1 DKA Events in the 3 Months Prior to Questionnaire Completion and Child Sleep Disturbances (CSHQ)



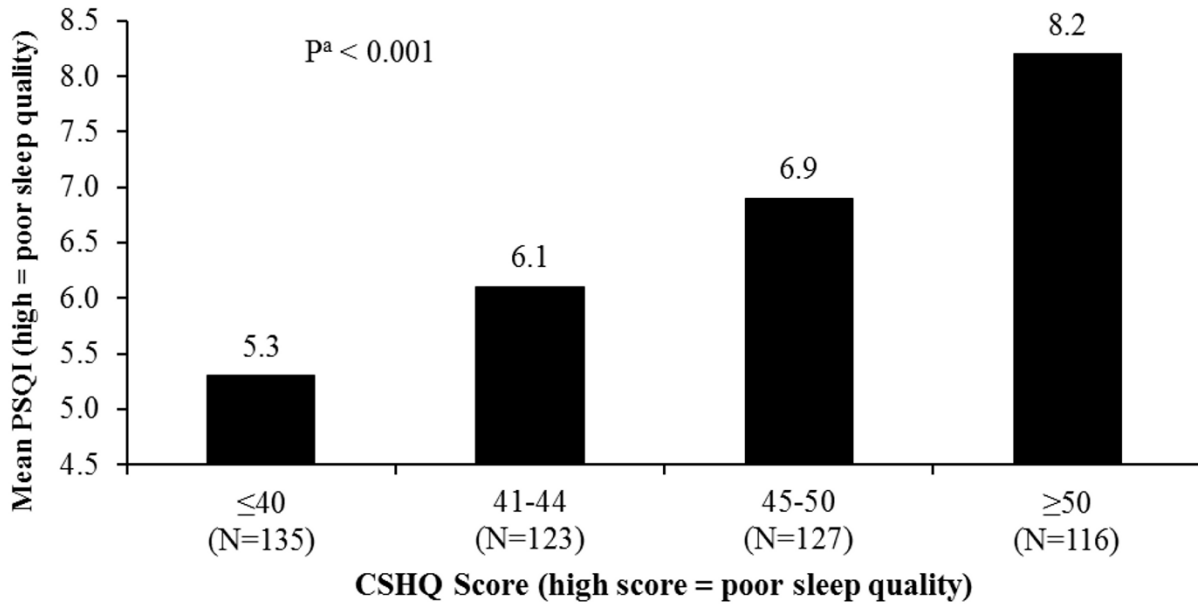
^aP-value from a multivariable logistic regression model adjusting for race/ethnicity, type of insurance, and use of an insulin pump

Figure 1D. Association between Parental Well-Being (WHO-5) and Child Sleep Disturbances (CSHQ)



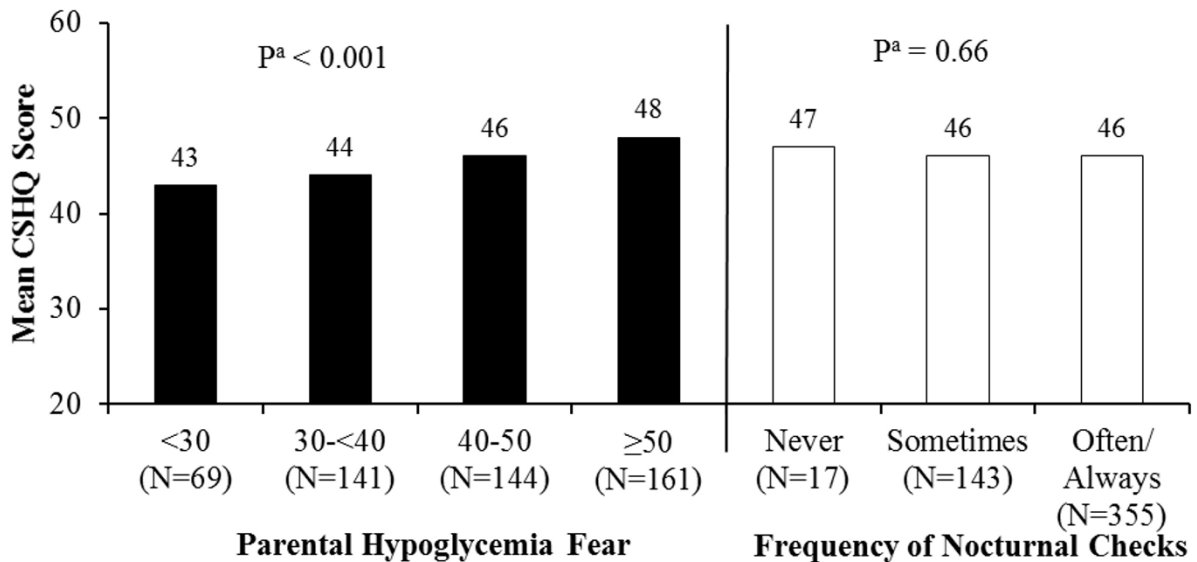
^aP-value calculated from linear regression model adjusting for race/ethnicity and child age at start of questionnaire

Figure 1E. Association between Parental Sleep Quality (PSQI) and Child Sleep Disturbances (CSHQ)



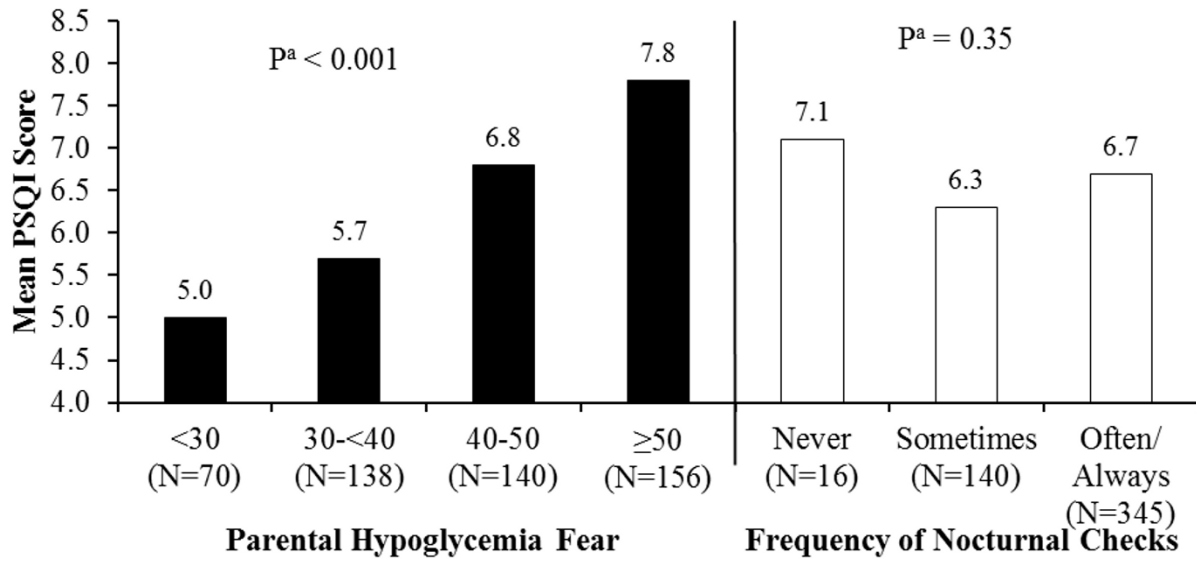
^a P-value calculated from linear regression model adjusting for race/ethnicity and sex

Figure 2A. Characteristics Associated with Child Sleep Disturbances (CSHQ)



^a P-value calculated from a linear regression model adjusted for clinic site, race/ethnicity, and child age at questionnaire start

Figure 2B. Characteristics Associated with Parental Sleep Quality (PSQI)



^a P-value calculated from a linear regression model adjusted for race/ethnicity and sex

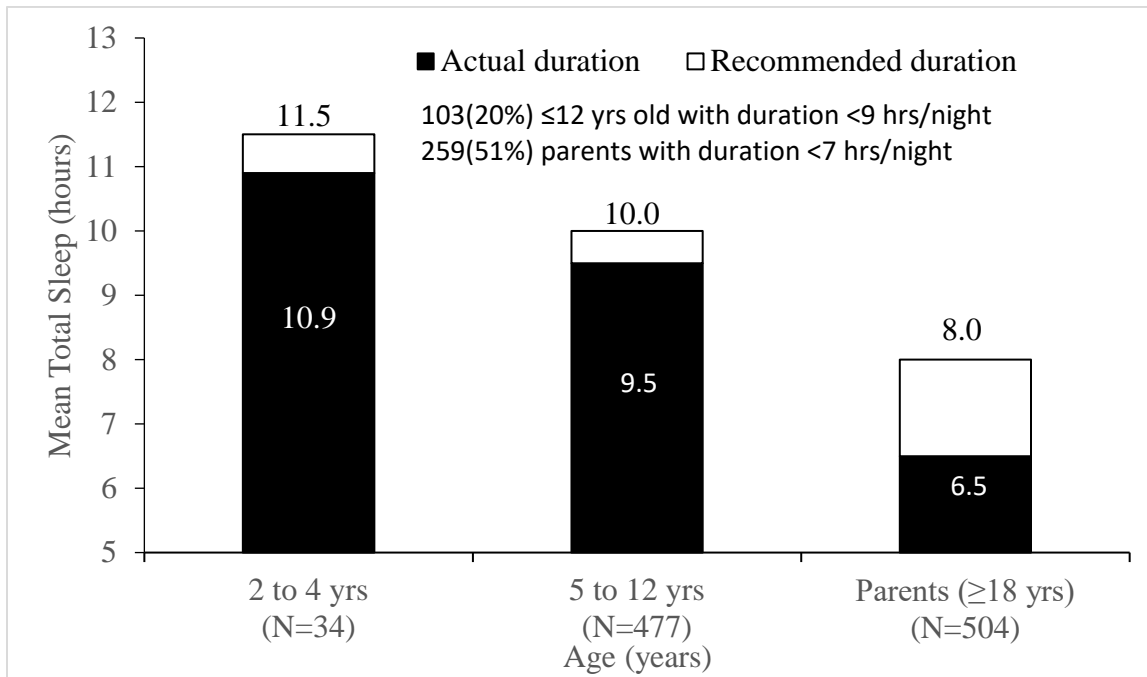
Supplemental Table 1. T1D Exchange Clinic Registry Characteristics of Study Completers vs. Study Non-Completers^a

	Completed Survey N=515	Sent Survey but Did not Complete Survey N=1841
Age (years) – mean ± SD	9 ± 3	10 ± 2
Female^b – n(%)	241 (47%)	848 (46%)
Race/ethnicity^b – n(%)		
White Non-Hispanic	439 (85%)	1438 (78%)
Black Non-Hispanic	16 (3%)	111 (6%)
Hispanic or Latino	27 (5%)	168 (9%)
Other Race/Ethnicity	26 (5%)	107 (6%)
Type of health insurance^b – (n%)		
Private insurance	383 (76%)	1305 (74%)
Other insurance	121 (24%)	444 (25%)
No insurance	2 (<1%)	4 (<1%)
Insulin modality^b – n(%)		
Insulin pump	394 (77%)	1250 (68%)
Multiple daily insulin injections	120 (23%)	590 (32%)
Uses a Continuous Glucose Monitor – n(%)	139 (27%)	343 (19%)
HbA1c – mean ± SD	8.0% ± 1.1%	8.4% ± 1.3%
Age at diagnosis (years) – median (25th %ile, 75th %ile)	3 (2,6)	4 (2, 6)

^aData collected and stored as part of the participant’s involvement in the T1D Exchange clinic registry.

^bRace/ethnicity information missing for 24 participants; insurance information missing for 97 participants; insulin modality information missing for 2 participant

Supplemental Figure 1. Average Reported Sleep Duration vs Recommended Sleep Duration



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