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Insufficient Sleep in Young Patients with Diabetes and Their Families

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Abstract

Objective—We examined sleep in families of individuals with type 1 diabetes and the relationship of sleep with obesity, diabetes, and insulin resistance.

Methods—Probands with type 1 diabetes diagnosed before age 18 and 1st and 2nd degree relatives were included ($n = 323$). Demographic, anthropometric and clinical variables and self-reported sleep duration and napping were assessed.

Results—On average, adults (≥ 20 years) slept 7.5 (SD 1.5) hr, whereas children (5–11 years) and adolescents (12–19 years) slept 9.8 (SD 1.1) and 8.5 (SD 1.9) hr, respectively ($p < .01$). Based on national recommendations, 40.9% of participants slept insufficiently, particularly young people (vs. adults, $p < .01$). In age-group stratified analysis, there were no significant associations of insufficient sleep or sleep duration with obesity, diabetes status, or insulin resistance after adjustment for age, race/ethnicity, and gender. 42% of participants reported napping regularly (1/week), with adolescents significantly more likely to do so (vs. adults, OR = 1.95, $p < .01$). Non-Hispanic Blacks and Hispanics also had higher odds of regular napping (vs. Non-Hispanic Whites, OR = 3.74, $p < .01$, and OR = 2.52, $p = .03$, respectively). In adjusted analysis, leaner (vs. obese) adolescents, whether measured by body mass index, percent body fat, or waist circumference, were significantly more likely to nap regularly.

Conclusions—We found that insufficient sleep was significantly more likely in children and adolescents compared with adults in families with type 1 diabetes. Lower adiposity was associated with regular napping in adolescents. The high prevalence of insufficient sleep in young patients with type 1 diabetes and their relatives detected in the current study may have significant health consequences.

Keywords

sleep; type 1 diabetes; obesity; family studies

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With the current rise in the prevalence of diabetes and obesity in the United States (Pearson, 2007), it is important to investigate potentially modifiable risk factors for these conditions in order to design effective prevention programs. One such risk factor is insufficient sleep. Sleep is not only a restorative process that allows the mind and body to rest, but it is also a dynamic activity during which many processes vital to health and well-being take place (National Sleep Foundation, 2006b). Previous research in adults has shown that reduced self-reported sleep duration is associated with an increase in 10-year diabetes incidence in women from the Nurses Health Study (Ayas et al., 2003) and a higher 12-year incidence of diabetes in Swedish men (Mallon, Broman, & Hetta, 2005). Reduced amounts of self-reported total sleep time per 24 hr have been associated with overweight and obese status in adults from primary care practices (Vorona et al., 2005); and controlled sleep restriction in men has caused a level of impaired glucose tolerance similar to that seen in type 2 diabetes (Spiegel, Leproult, & Van Cauter, 1999). Shortened sleep duration therefore appears to be associated with obesity, impaired glucose tolerance, and risk of type 2 diabetes in adults.

While the problems of increasing obesity and diabetes in children have received much attention, children and adolescents are also currently sleeping less than needed. Recent National Sleep Foundation (NSF) polls report that 54% of school-aged children sleep less than the recommended 10 hr (National Sleep Foundation, 2004), and 80% of adolescents get less than the optimal 9 hr of sleep on school nights (National Sleep Foundation, 2006a). Despite this, most epidemiological research on the association of reduced sleep with diabetes and obesity has focused on healthy adults and those with type 2 diabetes (Ayas et al., 2003; Gangwisch et al., 2007; Gangwisch, Malaspina, Boden-Albala, & Heymsfield, 2005; Knutson, Ryden, Mander, & Van Cauter, 2006; Mallon et al., 2005). Authors of a meta-analysis of epidemiological studies in the general pediatric populations of different countries found evidence of an association between short sleep duration and increased risk of childhood obesity (Chen, Beydoun, & Wang, 2008): for each hr increase in sleep, the risk of overweight/obesity was reduced by 9%. Additionally, a recent German study found that short self-reported sleep duration was associated with homeostasis-model assessed insulin resistance and elevated insulin levels in young girls (Hitze et al., 2009). These findings suggest that counseling to promote regular bedtime habits that ensure sufficient sleep for children may comprise an important nursing intervention for childhood obesity and associated risk factors for diabetes.

Focusing on sleep in those with type 1 diabetes may be another way to understand the relationship of sleep and diabetes. Children with type 1 diabetes, and possibly their siblings, are at increased risk for metabolic disruption. Children and adolescents with type 1 diabetes experience frequent nocturnal hypoglycemia (Beregszaszi et al., 1997) as well as alterations in sleep architecture (Pillar et al., 2003). To our knowledge, no previous family studies have examined sleep in those with type 1 diabetes. The analysis reported below describes self-reported sleep duration and nap frequency in young probands with type 1 diabetes and their relatives who participated in the Chicago Childhood Diabetes Family Study and the associations of these sleep variables with obesity, diabetes, and insulin resistance. We hypothesized that among people in these families, less sleep would be associated with greater adiposity, the presence of diabetes, and higher insulin resistance.

Materials and Methods

Participants

The Chicago Childhood Diabetes Registry (CCDR) Family Study was designed to cross-sectionally describe genetic and behavioral risk factors for diabetes and cardiovascular disease in families identified as high risk by virtue of having a member diagnosed with diabetes in childhood. Families were eligible if the proband was diagnosed with diabetes

prior to age 18, irrespective of the diabetic proband's current age, and if they currently resided in the Chicago metropolitan area. Probands with diabetes were recruited through clinics, health fairs, and mailings. First- and second-degree relatives were eligible if they had no serious chronic diseases (except diabetes). Families participated in the study in their homes or in the General Clinical Research Center at the University of Chicago after an overnight fast. Trained research staff collected data after obtaining written informed consent from participants 18 years of age and from parents of children <18 years of age as well as assent from children 10–17 years old. The Institutional Review Board at the University of Chicago approved the research. Of 331 participants aged five years or older at the time of data collection from families with type 1 diabetes, 8 participating family members were excluded for missing information on sleep. Thus, this analysis includes 323 participants (78 probands with type 1 diabetes, 227 of their relatives without diabetes, and 18 relatives with diabetes).

Demographics

Participants completed questionnaires, with parents answering for their children as necessary. Age was divided into three categories: 5–11 years (children), 12–19 years (adolescents), and 20+ years (adults). Race/ethnicity was self-reported and was categorized as non-Hispanic (NH) White, NH Black, Hispanic, and other (which includes Asian, American Indian, and mixed race).

Anthropometric Measurements

Research staff measured height using a portable stadiometer rod and weight and percent body fat using a bioelectric impedance analysis scale (Tanita TBF-300A, Arlington Heights, IL; Jebb, Cole, Doman, Murgatroyd, & Prentice, 2000). Percent body fat was available on participants 10 years of age. We transformed body mass index (BMI) into percentiles using age- and sex-matched reference data for children < 20 years old (National Center for Health Statistics, 2000) and adults 20 years old (McDowell, Fryar, & Ogden, 2009), and then categorized them using Centers for Disease Control BMI definitions: underweight (< 5th percentile), normal weight (5th to 84th percentile), overweight (85th to 94th percentile), and obese (95th percentile; Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults, 1998; National Center for Health Statistics, 2000). For this analysis, we dichotomized BMI categories into underweight/normal weight and overweight/obese, as results were similar between underweight and normal weight and between overweight and obese categories. We measured waist circumference (WC) twice, averaged the measures, and then dichotomized participants using age- and sex-specific 85th percentiles of WC (McDowell, Fryar, & Ogden, 2009).

Blood Collection

Research staff performed venipuncture after participants had completed an overnight fast. We determined glucose levels using a portable meter (One Touch SureStep, Lifescan, Milpitas, CA) and measured insulin levels in the University of Chicago Diabetes Research and Training Center laboratory with a solid-phase, two-site chemiluminescent immunometric assay (Immulite 1000, Siemens Medical Solutions Diagnostics, Los Angeles, CA). The intra-assay coefficient of variation was < 8.0%. We used glucose and insulin levels to calculate insulin resistance in the 220 of the 227 nondiabetic relatives who underwent venipuncture using the revised Homeostatic Model Assessment version 2.0 (HOMA2) method (Levy, Matthews, & Hermans, 1998). We defined insulin resistance as a HOMA2 value 3.2 for those < 20 years old (Keskin, Kurtoglu, Kendirci, Atabek, & Yazici, 2005) and 2.5 for those 20 years old (Levy, Matthews, & Hermans, 1998).

Individuals with a fasting blood glucose <150 mg/dL, measured by a glucometer (One Touch Sure Step, Lifescan, Milpitas, CA), had a stimulated C-peptide measurement 90 min after ingestion of a 6 ml/kg standard nutrient solution (Boost, Novartis Nutrition Corporation, Minneapolis, MN). We determined plasma C-peptide with a solid-phase, competitive chemiluminescent enzyme immunoassay (Immulin 2000, Diagnostic Products Corporation, Germany) in the University of Chicago's Diabetes Research and Training Center Lab. The lower limit of detection was 0.17 nmol/L and the intra-assay coefficient of variation (CV) was 8%. Absent C-peptide was defined as a fasting and stimulated level below the detection limit. C-peptide is the remaining inactive portion of the endogenously secreted proinsulin molecule which is cleaved to make active insulin. C-peptide levels thus directly reflect the amount of insulin secreted by the pancreas. In contrast, insulin treatment for those who are unable to secrete insulin (i.e., those with type 1 diabetes) involves the active insulin molecule alone—there is no cleavage of proinsulin and thus no C-peptide. Therefore, the absence of C-peptide can be used as the gold standard for determining type 1 diabetes status among those whose blood levels of insulin reflect the administration of the hormone by injection. We quantified antibodies to radiolabelled recombinant human GAD65 (whole) and human IA2/ICA512 (349 AA cytoplasmic portion) by fluid phase immunoprecipitation assay (Woo et al., 2000). These antibodies are thought to indicate the presence of pancreatic autoimmunity and thus can be used to distinguish type 1 from type 2 (non-autoimmune) diabetes (Lieberman & DiLorenzo, 2003).

Diabetes Status

We assigned diabetes type based on information from a physical examination and questionnaires. We classified participants as having type 1 diabetes if they had 1) no endogenous insulin production as indicated by undetectable fasting and postchallenge C-peptide or 2) detectable C-peptide with < 2 years' diabetes duration and were either positive for islet autoantibodies or receiving intensive insulin treatment (≥ 3 insulin injections per day or on an insulin pump). We classified probands not meeting these criteria as non-type 1 and did not include them or their families in this analysis. We assigned relatives of type 1 probands as having type 1 diabetes ($n = 8$) based on these same criteria. We assigned them as having type 2 diabetes based on detectable C-peptide, no islet autoantibodies, and either not using insulin at all or using ≤ 2 insulin shots/day (with or without oral antidiabetic agents; $n = 8$). We were unable to type 2 relatives who self-reported diabetes because of ambiguous data.

Sleep

Participants reported their previous day's sleep and wake times; if these were not typical, the questionnaire asked them to clarify what their typical sleep and wake times were. We used the interval between these two times as a proxy for typical sleep duration. As sleep requirements vary with age (Iglowstein, Jenni, Molinari, & Largo, 2003), we categorized sleep as adequate or insufficient for three age groups based on American Academy of Sleep Medicine recommendations, with adequate sleep being ≥ 10 hr for children, ≥ 9 hr for adolescents, and ≥ 7 hr for adults (Lamb, 2006; National Institute of Neurological Disorders & Stroke, 2006). We asked participants about the frequency of daytime naps, which we categorized as either infrequent (< 1 weekly) or regular (≥ 1 time weekly), as research indicates that daily napping is very infrequent in those 5 years of age and older (Iglowstein et al., 2003).

Statistical Methods

We used a regression approach to examine associations of sleep with demographics, body habitus (BMI, percent body fat, and WC), diabetes status, and insulin resistance and linear mixed models to model the continuous outcome, sleep duration, with family as a random

effect (McCulloch & Searle, 2001). We assessed normality of the sleep duration distribution prior to analysis. We used generalized estimating equation (GEE) logistic regression to model the dichotomous outcomes of insufficient sleep and nap frequency, with family as a repeated factor with an exchangeable correlation structure (Liang, Zeger, & Qaqish, 1992) and evaluated statistical significance using Score Statistics for Type 3 GEE Analysis. All models therefore incorporate correlation due to clustering of participants within families. We fit univariate models first, followed by multivariable models adjusted for age, gender, and race/ethnicity. We also tested interactions of the main predictors (body habitus, diabetes, and insulin resistance) with age group, gender, and race, and there was evidence of an interaction with age group. Because of this effect modification, we stratified all final multiple variable models by age group. We considered all statistical tests significant at $\alpha = .05$. We performed analyses using SAS version 9.1 GENMOD and MIXED procedures (SAS Institute, Cary, NC).

Results

Participants ranged in age from 5.0 to 82.5 years, with means (standard deviations [SDs]) of 14.4 (6.3) years for probands and 33.7 (19.2) years for relatives (see Table 1). Participants were ethnically diverse, with the sample being 37.5% NH Black, 42.4% NH White, 14.5% Hispanic, and 5.6% other race. Overall, 53.3% of participants were overweight/obese by BMI, mean percent body fat was 31.7%, and 25.1% were 85th percentile in WC. In addition to the probands, 18 relatives (7.4%) reported having been diagnosed with diabetes, and among relatives without diabetes, 9.1% met the criteria for insulin resistance.

Figure 1 shows the distribution of sleep duration by age group. Participants averaged 8.3 (1.8) hr of nighttime sleep (range 4.0–16.0 hr); adults slept 7.5 (1.5) hr on average, whereas children and adolescents slept 9.8 (1.1) and 8.5 (1.9) hr, respectively ($p < .01$ for both age groups vs. adults; see Table 2). Overall, 40.9% of participants had insufficient sleep. The odds of insufficient sleep were 2.98 times greater in children and 5.85 times greater in adolescents compared with adults ($p < .01$ for both comparisons). Sleep duration and insufficiency were not significantly different between genders and among race/ethnicity groups. After adjusting for age at visit, gender, and race/ethnicity, there were no significant associations of sleep duration or sleep adequacy with body habitus, diabetes, or insulin resistance (data not shown). However, in adults, those with 85th percentile WC showed marginally decreased sleep duration (vs. < 85th percentile WC, mean difference = -0.5 hours, $p = .08$).

With regard to napping, overall 42.2% of participants reported taking naps regularly (Table 2). Adolescents were significantly more likely than adults to take regular naps (odds ratio [OR] = 1.87, $p < .01$), but children were not (OR = 0.95, $p = .98$). Regular napping was more prevalent among NH Black participants (OR = 3.74, $p < .01$) and Hispanic participants (OR = 2.52, $p = .03$) than among NH White participants. Those with insufficient nighttime sleep duration were not more likely to nap regularly during the day (OR = 1.47, $p = .15$). In the adjusted models, among adults and children, regular napping was not associated with body habitus, diabetes, or insulin resistance (data not shown). However, in adolescents, regular napping was significantly and inversely associated with overweight/obese BMI (vs. normal BMI, OR = 0.19, $p < .01$), higher percent body fat (per 10 unit increase, OR = 0.39, $p < .01$) and 85th percentile WC (vs. < 85th percentile WC, OR = 0.20, $p = .05$). NH Black race/ethnicity (vs. NH White) remained significantly associated with regular daytime napping in all multivariable models.

Discussion

To our knowledge, this study is the first description of sleep duration and nap frequency in families of those with type 1 diabetes. We found that 41% of the CCDR Family Study participants were sleeping insufficiently. Our results on sleep duration in children and adolescents are similar to NSF polls (National Sleep Foundation, 2004; National Sleep Foundation, 2006a), which found that 52% of children aged 5–11 and 80% of adolescents aged 12–19 had insufficient sleep. The proportions with insufficient sleep in the current study were 52% and 64%, respectively. The NSF polls also reported that 18% of school-aged children and 38% of adolescents take regular naps, with the proportions being higher in the current study (33% and 53%, respectively), but not significantly so. Taken together, these results demonstrate that children and adolescents do not get sufficient amounts of sleep. Because adequate sleep is particularly vital as children and adolescents pass through important physical, psychological, and developmental stages, sleep deprivation at a young age may represent a significant health burden. For example, insufficient sleep contributes to excessive daytime sleepiness and school performance problems, and it is associated with depressed mood (Fallone, Owens, & Deane, 2002). There is also increasing evidence that insufficient sleep is a risk factor for obesity, insulin resistance, and diabetes (Spiegel et al., 1999; Vorona et al., 2005).

Previous studies in adults have found that less sleep increased the risk of developing diabetes and insulin resistance (Ayas et al., 2003; Gangwisch et al., 2007; Knutson et al., 2006; Mallon et al., 2005). Our analysis showed no association of either diabetes or insulin resistance with sleep duration or insufficient sleep, possibly because the sample size was relatively small. However, it is also possible that, in subjects already at elevated risk of diabetes by virtue of being part of a family with an individual with type 1 diabetes, sleep may not add any additional risk beyond the genetic and lifestyle factors within these families.

The current analysis also did not demonstrate significant associations between sleep duration and obesity, in contrast to other research (Chen et al., 2008; Knutson et al., 2006; Taheri, Lin, Austin, Young, & Mignot, 2004; Vorona et al., 2005). However, we did find significant associations between regular napping and lower adiposity as measured by BMI, percent body fat, and WC in adolescents.

Research on the relationship between napping and nighttime sleep has been limited, and interpretations based on this research are inconclusive. The results of the current study showed no association of insufficient nighttime sleep with regular napping. Similarly, other research has found that there were no significant differences in nighttime sleep quantity or quality between adults who did and did not nap (Pilcher, Michalowski, & Carrigan, 2001). While regular daytime napping may not fully compensate for insufficient nighttime sleep, it may help to decrease negative effects such as daytime sleepiness and poor performance (Vgontzas et al., 2007). Also, as was indicated by our results, regular napping in adolescents may offer specific protective effects against obesity in addition to nighttime sleep. However, further research is needed. The highly significant association of NH Black race/ethnicity with regular naps remained when adjusted for other covariates in all age groups. Others have found similar racial differences in regular nap behavior in children (Crosby, LeBourgeois, & Harsh, 2005), perhaps reflecting cultural differences in napping acceptability.

Many potential biological mechanisms have been proposed to explain how obesity and diabetes may be associated with decreased sleep duration. One study showed that sleep debt significantly lowers glucose tolerance and increases cortisol in young, healthy adults, indicating that insufficient sleep affects carbohydrate metabolism and endocrine function

(Spiegel et al., 1999). Another study reported that perceived sleep debt (desired hours of sleep minus actual hours of sleep) and shorter weekday sleep duration were significantly associated with higher hemoglobin A1c, indicative of poorer blood sugar control, in adults with diabetes (Knutson et al., 2006). Reduced levels of leptin, a hormone that regulates hunger and metabolism, have been associated with shortened sleep in adults (Taheri et al., 2004). Studies in children and adolescents have shown that obese children with shorter sleep duration also have increased insulin resistance and that children with obstructive sleep apnea (OSA) have higher levels of insulin resistance when compared to those without OSA (Flint et al., 2007). Unfortunately, the current study did not include a measure of OSA. Thus, obesity, diabetes, and sleep may be linked through alterations in energy metabolism, endocrine dysfunction, leptin, and/or OSA.

One limitation of the present study is the use of self-reported sleep. However, other subjective sleep assessments have been found to be valid when compared to actigraphic sleep measurements (Lockley, Skene, & Arendt, 1999). The measurement of sleep duration over 1 day may also not accurately reflect usual sleep patterns, though follow-up questions were asked to determine if the participants' reported sleep was typical. Furthermore, the interval between reported sleep and wake times is a relatively crude measure for sleep duration, as it more correctly estimates time in bed which could include time spent trying to fall asleep (sleep latency). Unfortunately, we did not collect information on OSA and nap duration; therefore, total sleep duration over a 24-hr period cannot be assessed. The fact that many of the children's questionnaires were completed by their parents might have led to some inaccuracies, although good agreement has been found between child- and parent-completed questions on sleeping problems and tiredness (Sundblad, Saartok, & Engström, 2006). Finally, using the age-specific AASM recommendations (Lamb, 2006; National Institute of Neurological Disorders & Stroke, 2006) for categorizing adequate vs. insufficient sleep duration is also a limitation because sleep needs may vary from person to person. In future studies, examining perceived sleep debt may be a way to address this variation.

The use of a cross-sectional study design precludes conclusions about the direction of the association between insufficient sleep and diabetes. Previous research supports an increased risk of diabetes in individuals with reduced sleep, but diabetes may also alter sleep. For example, almost 50% of children and adolescents with type 1 diabetes experience nocturnal hypoglycemia (Beregszaszi et al., 1997). Young individuals with type 1 diabetes also experience more awakenings during sleep from rapid changes in glucose levels (Pillar et al., 2003). Therefore, diabetes may affect, not only the sleep of the individual with diabetes, but also the sleep of their families due to frequent nightly arousals and increased anxiety over nocturnal hypoglycemia.

Conclusions

The high prevalence of insufficient sleep in young patients with type 1 diabetes and their relatives detected in the current study may have several significant health consequences. There is clearly a role for nurses and health educators in addressing modifiable risk factors such as insufficient sleep in young people. Promoting regular sleep habits in childhood carries the potential for lifelong health benefits in addition to dealing with childhood obesity.

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References

- Ayas NT, White DP, Al-Delaimy WK, Manson JE, Stampfer MJ, Speizer FE, Hu FB. A prospective study of self-reported sleep duration and incident diabetes in women. *Diabetes Care*. 2003; 26:380–384. [PubMed: 12547866]
- Beregszaszi M, Tubiana-Rufi N, Benali K, Noel M, Bloch J, Czernichow P. Nocturnal hypoglycemia in children and adolescents with insulin-dependent diabetes mellitus: Prevalence and risk factors. *Journal of Pediatrics*. 1997; 131:27–33.
- Chen X, Beydoun MA, Wang Y. Is sleep duration associated with childhood obesity? A systematic review and meta-analysis. *Obesity*. 2008; 16:265–274. [PubMed: 18239632]
- Crosby B, LeBourgeois MK, Harsh J. Racial differences in reported napping and nocturnal sleep in 2- to 8-year-old children. *Pediatrics*. 2005; 115:225–232. [PubMed: 15866856]
- Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: Executive summary. *American Journal of Clinical Nutrition*. 1998; 68:899–917. [PubMed: 9771869]
- Fallone G, Owens JA, Deane J. Sleepiness in children and adolescents: Clinical implications. *Sleep Medicine Reviews*. 2002; 6:287–306. [PubMed: 12531133]
- Flint J, Kothare SV, Zihlif M, Suarez E, Adams R, Legido A, DeLuca F. Association between inadequate sleep and insulin resistance in obese children. *Journal of Pediatrics*. 2007; 150:364–369. [PubMed: 17382111]
- Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG, Malaspina D. Sleep duration as a risk factor for diabetes incidence in a large US sample. *Sleep*. 2007; 30:1667–1673. [PubMed: 18246976]
- Gangwisch JE, Malaspina D, Boden-Albala B, Heymsfield SB. Inadequate sleep as a risk factor for obesity: Analyses of NHANES I. *Sleep*. 2005; 28:1289–1296. [PubMed: 16295214]
- Hitze B, Bosy-Westphal A, Bielfeldt F, Settler U, Plachta-Danielzik S, Pfeuffer M, Muller MJ. Determinants and impact of sleep duration in children and adolescents: Data of the Kiel Obesity Prevention Study. *European Journal of Clinical Nutrition*. 2009; 63:739–746. [PubMed: 18665183]
- Iglowstein I, Jenni OG, Molinari L, Largo RH. Sleep duration from infancy to adolescence: Reference values and generational trends. *Pediatrics*. 2003; 111:302–307. [PubMed: 12563055]
- Jebb SA, Cole TJ, Doman D, Murgatroyd PR, Prentice AM. Evaluation of the novel Tanita body-fat analyser to measure body composition by comparison with a four-compartment model. *British Journal of Nutrition*. 2000; 83:115–122. [PubMed: 10743490]
- Keskin M, Kurtoglu S, Kendirci M, Atabek ME, Yazici C. Homeostasis model assessment is more reliable than the fasting glucose/insulin ratio and quantitative insulin sensitivity check index for assessing insulin resistance among obese children and adolescents. *Pediatrics*. 2005; 115:e500–e503. [PubMed: 15741351]
- Knutson KL, Ryden AM, Mander BA, Van Cauter E. Role of sleep duration and quality in the risk and severity of type 2 diabetes mellitus. *Archives of Internal Medicine*. 2006; 166:1768–1774. [PubMed: 16983057]
- Lamb, C. Sleep & children. 2006. Retrieved from SleepEducation.com <http://www.sleepeducation.com/Topic.aspx?id=8>
- Levy JC, Matthews DR, Hermans MP. Correct homeostasis model assessment (HOMA) evaluation uses the computer program. *Diabetes Care*. 1998; 21:2191–2192. [PubMed: 9839117]
- Liang KY, Zeger SL, Qaqish B. Multivariate regression analysis for categorical data (with discussion). *Journal of the Royal Statistical Society*. 1992; 54:3–40. Series B.

- Lieberman SM, DiLorenzo TP. A comprehensive guide to antibody and T-cell responses in type 1 diabetes. *Tissue Antigens*. 2003; 62:359–377. [PubMed: 14617043]
- Lockley SW, Skene DJ, Arendt J. Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. *Journal of Sleep Research*. 1999; 8:175–183. [PubMed: 10476003]
- Mallon L, Broman JE, Hetta J. High incidence of diabetes in men with sleep complaints or short sleep duration: A 12-year follow-up study of a middle-aged population. *Diabetes Care*. 2005; 28:2762–2767. [PubMed: 16249553]
- McCulloch, CE.; Searle, SR. Generalized, linear and mixed models. New York, NY: Wiley; 2001.
- McDowell MA, Fryar CD, Ogden CL. Anthropometric reference data for children and adults: United States, 1988–1994. National Center for Health Statistics. *Vital Health Statistics*. 2009; 11(249)
- National Center for Health Statistics. 2000 CDC growth charts: United States. 2000. Retrieved from <http://www.cdc.gov/growthcharts>
- National Institute of Neurological Disorders & Stroke. Sleep need. 2006. Retrieved from SleepEducation.com at <http://www.sleepeducation.com/Topic.aspx?id=39>
- National Sleep Foundation. Final report: Sleep in America poll 2004. Washington, DC: National Sleep Foundation; 2004.
- National Sleep Foundation. Final report: Sleep in America poll 2006. Washington, DC: National Sleep Foundation; 2006a.
- National Sleep Foundation. Sleep-wake cycle: Its physiology and impact on health. Washington, DC: National Sleep Foundation; 2006b.
- Pearson WS. Ten-year comparison of estimates of overweight and obesity, diagnosed diabetes and use of office-based physician services for treatment of diabetes in the United States. *Preventive Medicine*. 2007; 45:353–357. [PubMed: 17707497]
- Pilcher JJ, Michalowski KR, Carrigan RD. The prevalence of daytime napping and its relationship to nighttime sleep. *Behavioral Medicine*. 2001; 27:71–76. [PubMed: 11763827]
- Pillar G, Schusheim G, Weiss R, Malhotra A, McCowen KC, Schlitner A, ... Shehadah N. Interactions between hypoglycemia and sleep architecture in children with type 1 diabetes mellitus. *Journal of Pediatrics*. 2003; 142:163–168. [PubMed: 12584538]
- Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet*. 1999; 354:1435–1439. [PubMed: 10543671]
- Sundblad GM, Saartok T, Engström LM. Child-parent agreement on reports of disease, injury and pain. *BMC Public Health*. 2006; 6:276. [PubMed: 17092332]
- Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Medicine*. 2004; 1:e62. [PubMed: 15602591]
- Vgontzas AN, Pejovic S, Zoumakis E, Lin HM, Bixler EO, Basta M, Chrousos GP. Daytime napping after a night of sleep loss decreases sleepiness, improves performance, and causes beneficial changes in cortisol and interleukin-6 secretion. *American Journal of Physiology, Endocrinology, & Metabolism*. 2007; 292:E253–E261. [PubMed: 16940468]
- Vorona RD, Winn MP, Babineau TW, Eng BP, Feldman HR, Ware JC. Overweight and obese patients in a primary care population report less sleep than patients with a normal body mass index. *Archives of Internal Medicine*. 2005; 165:25–30. [PubMed: 15642870]
- Woo W, LaGasse JM, Zhou Z, Patel R, Palmer JP, Campus H, Hagopian WA. A novel high-throughput method for accurate, rapid, and economical measurement of multiple type 1 diabetes autoantibodies. *Journal of Immunological Methods*. 2000; 244:91–103. [PubMed: 11033022]

Figure 1.

Distribution of Hours of Sleep by Age Group in the Chicago Childhood Diabetes Registry Family Study. A. Ages 5–11 ($n = 64$), B. Ages 12–19 ($n = 92$), C. Ages 20+ ($n = 167$). Vertical line = the recommended number of hours for “adequate” versus “insufficient” sleep for each age group according to the American Academy of Sleep Medicine (Lamb, 2006; National Institute of Neurological Disorders & Stroke, 2006).

Table 1Sample Description (*N* = 323).

Characteristic	Probands (<i>n</i> = 78)	Relatives (<i>n</i> = 245)
Demographic		
Male gender, <i>n</i> (%)	37 (47.4)	102 (41.6)
Age at visit in years, mean (SD, range)	14.4 (6.3, 5.0–45.3)	33.7 (19.2, 5.2–82.5)
5–11, <i>n</i> (%)	25 (32.1)	39 (15.9)
12–19, <i>n</i> (%)	44 (56.4)	48 (19.6)
20+, <i>n</i> (%)	9 (11.5)	158 (64.5)
Race/ethnicity, <i>n</i> (%)		
Non-Hispanic White	36 (46.2)	101 (41.2)
Non-Hispanic Black	24 (30.8)	97 (39.6)
Hispanic	13 (16.7)	34 (13.9)
Other	5 (6.4)	13 (5.3)
Clinical and anthropometric		
Self-reported diabetes, <i>n</i> (%)	N/A	18 (7.4)
HOMA2, mean (SD) ^a	N/A	1.2 (1.1)
Insulin resistant, <i>n</i> (%)	N/A	20 (9.1)
BMI ^b		
(< 20 years) BMI percentile, mean	21.2	21.2
(< 20 years) BMI 85th percentile, <i>n</i> (%)	23 (33.3)	26 (27.4)
(≥ 20 years) BMI, mean (SD)	30.4 (8.5)	31.0 (8.2)
Overweight or obese, <i>n</i> (%)	26 (33.3)	146 (60.1)
% body fat, mean (SD) ^c	27.4 (10.2)	33.0 (12.2)
Waist circumference in cm, mean (SD) ^d	72.4 (14.5)	87.4 (21.4)
Over 85th percentile, <i>n</i> (%)	10 (13.0)	71 (29.6)

^aHOMA2 = Homeostatic Model Assessment 2. HOMA2 was determined in 220 nondiabetic relatives. Insulin resistance criterion based on fasting insulin HOMA2:

if age < 20 years, insulin resistant = HOMA2 ≥ 3.2

if age ≥ 20 years, insulin resistant = HOMA2 ≥ 2.5

^bBMI = body mass index. BMI was transformed into percentiles using age- and sex-matched reference data (McDowell et al., 2009; National Center for Health Statistics, 2000). Overweight/obese was defined as ≥ 85th percentile (Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults, 1998; National Center for Health Statistics, 2000). BMI was available on 243 relatives.

^cPercent body fat was available on 60 probands and 213 relatives ≥ 10 years old.

^dWaist circumference was available on 77 probands and 240 relatives.

Table 2
Bivariate Associations of Sleep Duration, Insufficient Sleep, and Regular Naps with Demographic Characteristics.

Demographic Characteristic	Sleep Duration (hr)		Insufficient Sleep (vs. Adequate)		Regular Naps (vs. Infrequent) ^a	
	n (%)	Mean (95% CI)	Mean Difference from Reference (95% CI)	n (%)	Odds Ratio (95% CI)	n (%)
Total	323 (100)	8.3 (8.1, 8.5)	--	132 (40.9)	--	136 (42.2)
Age (years)						
5–11	64 (19.8)	9.8 (9.4, 10.3)	2.2 (1.8, 2.7)*	31 (48.4)	2.98 (1.45, 6.14)*	21 (33.3)
12–19	92 (28.5)	8.5 (8.2, 9.0)	1.0 (0.6, 1.4)*	60 (65.2)	5.85 (3.30, 10.34)*	49 (53.3)
20+	167 (51.7)	7.5 (7.3, 7.8)	Ref. ^b	41 (24.6)	Ref.	66 (39.5)
Gender						
Female	184 (57.0)	8.2 (7.8, 8.6)	−0.2 (−0.6, 0.2)	70 (38.3)	0.78 (0.52, 1.15)	83 (45.4)
Male	139 (43.0)	8.4 (8.1, 8.7)	Ref.	62 (44.3)	Ref.	53 (38.1)
Race/ethnicity						
NH Black	121 (37.5)	8.3 (7.7, 8.9)	0.2 (−0.4, 0.8)	57 (41.0)	1.24 (0.70, 2.22)	79 (56.8)
Hispanic	47 (14.5)	8.5 (7.7, 9.3)	0.4 (−0.4, 1.2)	21 (45.7)	1.40 (0.66, 2.93)	32 (26.7)
Other	18 (5.6)	8.6 (7.4, 9.7)	0.5 (−0.7, 1.6)	8 (44.4)	1.67 (0.43, 6.43)	19 (42.2)
NH White	137 (42.4)	8.1 (7.7, 8.5)	Ref.	46 (38.3)	Ref.	6 (33.3)

Note. NH = non-Hispanic.

* Significant mean difference or odds ratio ($p < .05$).

^a Regular naps: 1 per week; infrequent naps: < 1 per week; $n = 322$.

^b Ref: reference group for comparing means or calculating odds ratios.