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Sleep Duration and Type 2 diabetes Risk: A Prospective Study in a Population-based Mexican American Cohort.

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Abstract

The primary aim of the study was to estimate the effect of sleep duration on prospective type 2 diabetes (T2D) risk across demographic characteristics and follow-up periods, and test BMI as a mediator and moderator. Data included adults (M_{age} = 39.0 ± 12.7y) born in the US or Mexico recruited from 2001-2012 in a Mexican American cohort study conducted in Houston, Texas (n=15,779). Participants completed self-reported questionnaires at baseline related to health, health behaviors (sleep duration, physical activity, smoking, drinking) and sociocultural factors, and were followed up annually. Cox proportional hazard models estimated hazard ratios (HR) for the effect of sleep duration on T2D diagnosis at follow-up. Of the participants, 10.3% were diagnosed with T2D. Self-reported ≤5 hours of sleep, compared to 7-8 hours, at baseline predicted greater risk for T2D (HR=1.32, p=.001), yet was no longer significant after adjusting for sociodemographic characteristics and BMI. Notably, those with BMI < 25 kg/m^2 reporting ≤5 hours of sleep were at significant risk for T2D at 3 (HR=4.13, p=.024) and 5 year follow-up (HR=3.73, p=.008) compared to 7-8 hours. Obesity status accounted for 31.6% and 27.3% of the variance in the association between ≤5 and 6 hours of sleep and increased T2D risk, respectively. Results highlighted the mediating and moderating role of BMI, and its effect on T2D risk at earlier follow-up among those without obesity. T2D prevention and control for Mexican American adults should consider the role of chronic sleep loss.

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Keywords

Type 2 diabetes risk; obesity; health behaviors; sleep; Mexican Americans

Introduction

The last few decades have seen an upward trend in type 2 diabetes (T2D) incidence, which is beginning to level off in the general population.[1] However, Hispanic/Latinx (heretofore Hispanic, the preferred term in Texas) adults disproportionately experience increasing rates of T2D[2] with rates greater than non-Hispanic white adults.[3] In addition to increasing vulnerability to cardiovascular diseases and cancers,[4,5] T2D can economically burden patients given the costly comorbidities and T2D-related complications.[6,7] It is likely that the T2D disparities among Hispanic individuals is due in part to high rates of obesity in this group compared to non-Hispanic White individuals.[8,9] Excessive bodyweight is an important and mutable predictor of T2D,[10] and is partly responsible for its rise.[11] Indeed, excessive fat can decrease insulin sensitivity and impair pancreatic beta cells to increase risk for T2D.[12,13] Thus, understanding the behavioral risk factors for T2D in this population can inform community health promotion efforts.

While poor diet and physical inactivity are well-known risk factors for T2D,[10] a wealth of evidence also highlights insufficient sleep as an important,[14-16] if not more important,[17] contributor. The American Academy of Sleep Medicine and Sleep Research Society recommend adults (18-60 years) obtain at least 7 hours of sleep a night,[18] and evidence suggests that short sleep (≤ 6 hours/night) can increase obesity risk among diverse racial/ethnic minority adults.[19-22] According to the energy allocation theory of sleep functioning,[23] insufficient sleep upregulates energy requirements during the day to compensate for waking hour activation of biological processes that normally occur during nighttime sleep. That is, short sleep encourages a positive energy balance by increasing the energy intake required to maintain homeostasis during waking hours. Thus, short sleep can dysregulate metabolic processes by increasing insulin resistance and daily levels of ghrelin, and dampening daily levels of leptin—altogether increasing risk for obesity and T2D.[24-26]

The question of how obesity modifies (i.e., moderates) the link between short sleep and T2D among Mexican American adults remains unknown and an important area of inquiry. First, understanding the potential modifying nature of BMI can provide conceptual clarity in the role of obesity in the sleep duration and T2D risk link. Drawing upon an exposure and reactivity model,[27] conceptualizing BMI as a mediator highlights an *exposure* process by which short sleep increases exposure to T2D risk factors such as elevated BMI, whereas conceptualizing BMI as a moderator suggests that *reactivity* to short sleep varies across levels of BMI. A majority of theory[23,26,28] and epidemiological research[29-35] in this area focus on the exposure model (i.e., BMI as a mediator) leaving open opportunities to test alternative conceptualizations such as the reactivity model (i.e., BMI as a moderator). Studying the reactivity model furthers our theoretical understanding of BMI, but also provides helpful insight into T2D screening efforts by identifying for which groups short sleep increases T2D risk. Although no known study has stratified the effect of sleep duration

on T2D risk across BMI among Mexican American adults, studies in other countries do not find a modifying effect of BMI.[36,37] However, results may not generalize to the US context given the high rates of obesity. The reactivity model is indirectly supported in one study, which found that short sleep was more strongly related to hypertension risk among those with normal weight statuses.[38] Thus, we seek to test both the exposure and reactivity model by examining BMI as both an intermediary factor (i.e., mediator) and modifying factor (i.e., moderator) in the link between short sleep and T2D risk.

Second, there are few large-scale epidemiological studies examining the link between short sleep, obesity, and T2D among Hispanic adults—a fast growing ethnic group in the US. The mixed evidence has suggested both significant[39] and non-significant[40,41] relationships between short sleep and T2D among this group. Differences in study characteristics such as ethnic composition of the sample, acculturation level, and use of covariates likely account for the mixed findings. For example, samples likely included multiple ethnic groups varying in acculturation level, which was only identified one study,[40] and studies used different health behaviors (e.g., diet, physical activity, smoking) and demographic characteristics (e.g., income, shift work) as covariates. The heterogeneity within samples of Hispanic individuals is particularly important as one study found that sleep duration was related to body size only among Mexican American individuals.[42] Our study contributes to the literature by clarifying the sleep duration and T2D risk relationship among Mexican American adults born in the US or Mexico. Given that Mexican American adults have the highest prevalence of T2D compared to other Hispanic groups,[43] greater research is needed to understand how other behavioral risk factors in addition to physical activity and diet impact T2D risk in this population.

Previous studies highlight modifying effects of other important sociodemographic characteristics in the sleep duration and T2D link. For example, subgroups of Hispanic individuals are disproportionately impacted by inadequate sleep. Large-scale epidemiological studies suggest that the health consequences of sleep duration can vary by country of origin among Hispanic individuals,[40,44] likely reflecting different sociocultural and immigration histories. While Hispanic individuals who acculturate to the US culture experience worsened sleep habits,[45,46] it remains unclear if inadequate sleep increases risk of T2D among more acculturated individuals. Furthermore, past studies also suggest the link between sleep duration and T2D may differ across sex (i.e., null findings among women,[34,41,47] in contrast to significant effects found among men[30]), and age (i.e., significant effects of short sleep on T2D among those under 65 years[48], and variable effects of sleep timing on HbA1c levels among Hispanic individuals younger than 35 years[44]).

The current study builds upon previous research limited by cross-sectional study designs, racially/ethnically aggregated samples, and shorter follow-up periods to examine the prospective effect of sleep duration on self-reported T2D diagnosis among Mexican-origin adults. The primary aim of our study was to examine the baseline effect of sleep duration on T2D risk among Mexican-origin adults. We proposed the following hypotheses: (1) sleep duration will be inversely related to T2D risk; (2) obesity will moderate the relationship between sleep duration and T2D risk such that the effect of sleep duration on T2D risk will be stronger among those without obesity; (3) obesity will mediate the relationship between

sleep duration and T2D risk; and (4) the association between sleep duration and T2D risk will be stronger among individuals who reported greater acculturation, being born in the US, being male, or being younger.

Methods

Study cohort

Data were from the Mexican American Cohort Study conducted in Houston, TX. Details of study data collection are described elsewhere.[49] Briefly, participants completed an in-person interview related to current and past health, health behaviors, anthopometry, work history, and demographic and psychosocial characteristics. Participants were followed-up annually with a short telephone interview to update contact information and diagnoses for chronic diseases. When a new diagnosis of T2D was present, participants provided a date of diagnosis, type of diabetes and treatment, and names of the physician and hospital/clinic for diagnosis/treatment.

The final sample (n = 15,779) included Mexican-origin adults (≥21 years) born in the US or Mexico recruited between 2001-2012 with complete data for self-report sleep, BMI, and demographics at baseline, a follow-up interview prior to November 2018, and no reported history of T2D at study enrollment. There were 415 (2.6%) deaths at follow-up with 386 (2.4%) without T2D. Figure 1 displays the inclusion/exclusion flowchart for the current study resulting in no missing data for 15,779 participants. Overall, the distribution of responses to study variables were largely similar between the complete and final sample based on descriptive statistics.

The Mexican American Cohort study is conducted under the University of Texas MD Anderson Cancer Center Institutional Review Board approved protocols 2009-0379 and CPN00-367.

Measures

Diabetes.—Self-reported T2D was previously validated by reviewing medical records for a subset of participants (n = 235) and showed 98% agreement.

Sleep duration.—Sleep duration was based on one item related to number of hours slept including naps in the past year (i.e., “On the average, during the last year, how many hours in a day did you usually sleep including naps?”). Participants were asked to choose from 6 response options: ≤5, 6, 7, 8, 9, or ≥10 hours. The current study collapsed the 7 and 8 hour responses together based on a recent meta-analysis suggesting that the lowest T2D risk was between 7-8 hours of sleep per day.[50] Thus, sleep duration as categorized as: ≤5, 6, 7-8 [reference], 9, or ≥10 hours.

Acculturation.—Acculturation was measured using four items from the Bidimensional Acculturation Scale for Hispanics (BAS),[51] which assesses the frequency and ability to speak, read, and consume media in English and Spanish (e.g., How often do you watch television programs in English?). Participants rated each item on a 4-point Likert scale (1 = “almost always”/ “very well”; 2 = “often”/ “well”; 3 = “sometimes”/ “poorly”; 4 = “almost

never”/ “very poorly”). Items were reversed scored such that higher scores represented greater English or Spanish speaking, reading, or media use, and composite scores were calculated by averaging the 4 items for English and Spanish separately. Participants with mean scores greater than 2.5 on English or Spanish were classified as English or Spanish dominant, respectively. Participants with scores greater than 2.5 on both English and Spanish subscales were classified as bilingual.

Physical activity.—We measured physical activity using items from the California Teachers Study (CTS) survey.[52] Participants were asked to estimate the average hours per week (“Never,” “0.5,” “1,” “1.5,” “2,” “3,” “4–6,” “7–10,” or “11+ hours”) and the average number of months (“1–3,” “4–6,” “7–9,” and “10–12 months”) during the past year spent in strenuous, moderate or light exercise, sport or work activities. Average number of minutes per week spent doing strenuous and moderate physical activity was calculated by multiplying average hours per week and proportion of the year. The resulting time was then compared to national physical activity recommendations (i.e., 75 minutes of vigorous physical activity or 150 minutes of moderate activity) to create a dichotomous variable reflecting meeting/not meeting the physical activity guideline.

Substance use.—Alcohol consumption was measured using one item assessing frequency of drinking any alcohol beverages (“Do you now or did you ever drink any alcoholic beverages, at least once per month for 1 year or more?”) with three options (“Yes, currently,” “Yes, but I quit,” or “No, never.”) Similarly, smoking was measured using one item assessing frequency of cigarette use (“Have you smoked at least 100 cigarettes (5 packs) in your entire life?”) with three options (“Yes, currently,” “Yes, but I quit,” or “No, never smoked.”)

Body mass index (BMI).—BMI was based on measured height and weight data. When objective measures were not available, self-reported height and weight data were used to calculate BMI. Participants were classified as normal/underweight (BMI < 25.0), overweight (25 ≤ BMI < 30.0), or obese (BMI ≥ 30.0). Normal and underweight was combined given the few underweight cases (n = 108). 71.6% of the BMI data were based on measured height and weight by trained staff.

Demographic characteristics.—Demographic information included self-reported age (median split), sex (Male or Female), marital status, education, and birth country (US, Mexico). Marital status was recoded as “Married” (“Married/living as married”) or “Not married” (combined “Widowed”, “Separated”, “Divorced”, and “Never married”). Education was recoded as “<High school” (combined “No formal schooling”, “Elementary”, “Middle school/junior high”), “High school grad” (combined “High school”, “High school graduate”, “GED”, “Technical/vocational training”, “Associate degree/some college”), or “College” (combined “Bachelor's degree”, “Post-graduate degree”).

Statistical analyses

Preliminary analyses included descriptive statistics to characterize T2D progression, and t-tests and Chi-square tests to compare T2D risk across demographic characteristics. To

test the first hypothesis (i.e., sleep duration will be inversely related to T2D risk), Cox proportional hazard models were used to estimate the adjusted and unadjusted hazard ratio (HR) for T2D at follow-up associated with baseline predictors including sleep duration, health behaviors, and demographic characteristics. Participants free of T2D diagnosis were censored at their last follow-up interview or date of death (n = 13,765). Follow-up time was defined as the difference between diagnosis date and enrollment date for those who self-reported T2D at follow-up. Participants who did not self-report T2D diagnosis and died during the annual follow-up (n = 386) were censored at time of death. The median follow-up time was 9.67 years.

To test the second hypothesis (i.e., the sleep duration and obesity risk link will be stronger among those without obesity, who were more acculturated, who were males, and who were younger) was first tested by conducting independent moderated regressions for each potential modifier. Cox proportional hazard models included sleep duration, demographic characteristics, and their interaction. For statistically significant interactions, we then stratified the effect of sleep duration and T2D by demographic characteristic.

We also conducted a sensitivity analysis that stratified the effect of sleep on T2D across BMI (< 25, 25 to < 30, <30, and ≥ 30) by follow-up duration. Recent meta-analyses suggested a stronger effect of sleep duration on T2D risk among studies with longer follow-up durations[16] up to 10 years.[48] Based on the cumulative incidence of T2D risk across follow-up period (Figure 2), we censored at 3, 5, and > 5 year follow-up, which was 25%, 42%, and 58%, respectively. Further, we aggregated participants with BMI < 30 to explore the impact of sleep duration among those without obesity.

Lastly, we tested the third hypothesis (i.e., obesity will mediate the association between short sleep and T2D risk) using mediation analyses. A SAS macro was used to estimate total, direct, and indirect effects and potential mediator interactions in a Cox proportional hazard model.[53] The macro was specified to conduct a Cox proportional hazard model with a logit link function that allowed censoring and mediator interactions. Indirect effects were calculated using the delta method. Mediation was determined to be present if the value “1” did not fall within the 95% confidence intervals. We also explored the potential mediation of baseline physical activity (meet/does not meet physical activity guidelines).

Covariates included in main analyses included age, sex, education, marital status, birth country, acculturation, alcohol use, smoking, and physical activity. SAS version 9.4 (Cary, NC) was used to perform all analyses. Vital status (living/deceased) was not associated with greater T2D risk for the overall follow-up, but was related to greater T2D risk at the 3- and 5-year follow-up (Table S1). Including vital status as an additional covariate did not impact the pattern of results, and was thus not included as a covariate in the models. All statistical tests were 2-sided, and p-values <0.05 were considered statistically significant.

Results

Table 1 provides descriptive statistics for baseline characteristics of the sample stratified by T2D diagnosis. On average, the sample was 39.0 years old (SD = 12.7), predominantly

female (79.2%), married (80.0%), less than high school educated (58.1%), born in Mexico (76.1%), and Spanish-speaking dominant (58.6%). Participants were predominantly obese (47.6%), never smokers (74.8%), never drinkers (67.5%), did not meet physical activity guidelines (78.9%), and reported a daily average of 7-8 hours of sleep in the past year (62.3%). Risk of T2D at follow-up across sociodemographic and health behaviors was associated with being older, female, having less than a high school education, obese, not meeting physical activity guidelines, and reporting less sleep duration.

Results partially supported the hypothesis that short sleep would be related to increase T2D risk. To examine the association between sleep duration and T2D risk after controlling for covariates, Table 2 displays the results from Cox-proportional hazard models with the overall sample and stratified by sociodemographic and lifestyle behaviors. Results showed that the unadjusted effect of self-reported ≤ 5 hours of sleep, compared to 7-8 hours, on T2D at follow-up (HR = 1.315, p = .001) was no longer significant after adjusting for sociodemographic factors, BMI, and lifestyle behaviors (HR = 1.153, p = .092).

Results did not support the hypothesis that short sleep would be differentially related to T2D risk across age, sex, acculturation, or birth country (Table S2). However, results partially supported the hypothesis for BMI. In overall follow-up, the effect of short sleep on T2D risk did not differ across BMI, but censoring at different follow-up periods (3, 5, and >5 year follow-up) revealed significant effects (Table 3). Specifically, among those with BMI < 25 kg/m^2 (underweight + normal weight), ≤ 5 hours of sleep, compared to 7-8 hours, was related to increased T2D risk at 3 year (HR = 4.131, p = .024) and 5 year (HR = 3.733, p = .008) follow-up. Among those with BMI 25 to < 30 kg/m^2 (overweight), 6 hours of sleep, compared to 7-8 hours, was related to an increased risk of T2D at 3 year follow-up (HR = 1.827, p = .039). Among those with BMI < 30 kg/m^2 (non-obese), ≤ 5 hours of sleep, compared to 7-8 hours, was related to an increased risk of T2D at 3 year (HR = 2.209, p = .016) and 5 year follow-up (HR = 1.689, p = .034); and 6 hours of sleep compared to 7-8 hours was related to an increased risk of T2D at 3 year follow-up (HR = 1.781, p = .028).

Mediation results supported the hypothesis that the indirect effect of short sleep on T2D through obesity would be significant. Results from Cox proportional hazard models in Table 4 suggested that obesity accounted for a significant proportion of the variance in the association of ≤ 5 (31.6%) and 6 hours (27.3%) of sleep and increased T2D risk. Specifically, results showed that baseline short sleep (≤ 5 and 6 hours), compared to 7-8 hours, was related to greater likelihood of obesity, and obesity was related to a greater likelihood of T2D.

Exploratory analyses.

Table S3 displays results examining the association of sleep duration on T2D across non-significant interactions with sociodemographic factors. There was a significant effect of ≤ 5 hours of sleep compared to 7-8 hours of sleep among those aged ≤ 36 years (HR = 1.371, p = .038), and English dominant or bilingual (HR = 1.342, p = .017). Further, 6 hours of sleep compared to 7-8 hours was related to increased T2D risk among those born in the US (HR = 1.336, p = .019). Lastly, ≥ 10 hours compared to 7-8 hours was related to increased T2D

risk among males (HR = 1.970, p = .016). Exploratory mediation analyses testing physical activity as a mediator were not significant (Table S4).

Discussion

The current study is the largest prospective cohort study to examine the impact of self-reported sleep duration on T2D risk among Mexican American adults. Based on Cox proportional hazard models, the adjusted estimate of sleep duration on T2D risk was not significant. However, results revealed selective effects across multiple demographic characteristics and different follow-up periods. Further, mediation analyses suggested that baseline BMI significantly mediated the association between ≤ 5 hours of sleep and T2D risk.

The health profile of the sample suggested a population at-risk for chronic health conditions. While most of the sample reported sufficient sleep (73.6%) similar to Hispanic adults nationally (68.6%),[54] many reported not meeting physical activity guidelines (78.9%) and had BMI within the obesity range (47.6%). The elevated rates of physical inactivity and obesity likely contribute to the high rates of T2D at follow-up (10.3%).

Consistent with our hypotheses, sleep duration was related to future T2D risk and baseline BMI mediated this relationship. First, the modest unadjusted effect observed in our study for ≤ 5 hours of sleep was similar to adjusted pooled estimates in previous meta-analyses,[16,17,48,50] and consistent with epidemiological studies[29-35] demonstrating that the addition of BMI removed the significant effect of sleep duration. Thus, highlighting the important link between sleep duration, obesity, and T2D risk.

Second, results from mediation models supported theories highlighting excessive weight as an explanatory factor linking short sleep and T2D risk.[23,26,28] Specifically, baseline short sleep duration (≤ 5 and 6 hours compared to 7-8 hours) was related to increased baseline obesity risk, which was related to increased T2D risk. Notably, baseline obesity explained 31.6% and 27.3% of the variance in the effect of ≤ 5 and 6 hours of sleep on T2D risk, respectively. Because obesity is a major risk factor for dysregulated glycemic control and a host of comorbid health conditions, it is likely that an accumulation of multiple risk factors combine to impact T2D risk. Indeed, short sleep is linked to greater insulin resistance[55-57] and lower inhibitory responses to foods high in fat and carbohydrate content.[58] Chronic short sleep may increase unhealthy food intake thereby increasing risk for T2D. It is also possible that short sleep may increase T2D risk by interfering with pre-T2D management. Although this remains to be studied among adults, a recent study showed that shorter sleep duration was related to less engagement in T2D management behavior in youth with type 1 diabetes.[59] Chronic short sleep may increase forgetfulness to engage in glucose monitoring and decrease motivation to engage in self-regulatory behavior. While our study did not find physical activity as a significant mediator explaining the sleep duration and T2D relationship, this is an area deserving greater attention given the putative links between insufficient sleep and sedentary behavior.[60]

A notable finding was that reporting ≤ 5 hours of sleep at baseline was related to a 4.1 and 3.7 times increase in T2D risk among normal and underweight individuals at the 3 and > 5 year follow-up, respectively. These results suggest that sleep duration may be a stronger predictor of T2D at shorter follow-up durations and among non-obese individuals. First, it is possible that sleep duration is a better proximal rather than distal predictor of T2D risk. One study found that T2D risk was greater among those who slept ≤ 7 hours per night, compared to ≥ 8 hours, at the 6 year follow-up, yet this effect was not observed at the 11-year follow-up.[61] A meta-analysis also found a non-significant effect of sleep duration on T2D risk among studies with > 10 year follow-up.[16] It is possible that the predictive ability of sleep duration for T2D decreases with additional follow-up time due to changes in health conditions or health behaviors. Thus, insufficient sleep may be useful for early T2D detection among individuals without obesity. Indeed, a recent study in China found that short sleep improved the predictive ability of a T2D risk factor score,[62] highlighting the promise of considering short sleep as a screening factor.

Second, sleep duration may be a better predictor of future T2D among specific BMI ranges. In particular, we found that individuals with BMI < 25 who reported ≤ 5 hours of sleep per night were at the highest risk for T2D at short-term follow-up. These results are consistent with studies of other health behaviors and outcomes showing that short sleep was related to greater hypertension risk among individuals with normal and obese BMI only,[38] modestly stronger association between sleep duration and self-rated health among individuals without obesity,[63] and improving diet and exercise may prevent more cases of T2D among women with normal body weight.[64] Another study found a significant effect of sleep duration on T2D risk among those with BMI < 26.1, but not those with BMI ≥ 26.1 although the sleep and BMI interaction was not significant.[36]

Our results theoretically replicate and extend previous research by highlighting the dual role of BMI as a moderator and mediator in the sleep duration and T2D risk association. In addition to supporting the exposure model largely focused by previous research (i.e., BMI as a mediator),[29-35] results also supported the reactivity model (i.e., BMI as a moderator) contrary to international studies.[36,37] It is possible that as the cumulative effects of short sleep increase weight gain and prospective T2D risk, the hazards of short sleep diminish over time as other health conditions arise. Thus, for those without obesity, short sleep may be a particularly strong risk factor given the greater potential for weight gain and development of problematic health conditions.

Inconsistent with our hypotheses, demographic characteristics largely did not modify the effect of short sleep on T2D risk. Although there were no significant sleep by acculturation or birth country interactions, exploration of stratified effects showed a pattern of results suggesting that insufficient sleep may be particularly deleterious for more acculturated Mexican American adults. Immigration scholars find that among Hispanic adults residing in the US, health and health behaviors deteriorate with subsequent generations—a phenomenon known as the immigrant paradox.[65] Thus, it is possible that there may be fewer protective factors such as healthy eating to buffer the deleterious effects of insufficient sleep among Hispanic adults. However, results stratified by acculturation must be interpreted with caution given the non-significant interactions. Future research should further explore and empirically

test these assertions. Lastly, stratified analyses did not reveal sex differences in the effect of short sleep duration and T2D risk, which is consistent with previous studies among women[34,41,47] and men only.[30]

The study results are interpreted within the context of the following limitations. First, only one aspect of sleep was examined, namely, sleep duration. Recent studies suggest that later bedtimes predict greater insulin resistance,[44] and frequent night awakenings predict increased T2D risk[41] among Hispanic adults. Further, a Finnish study found that poor sleep quality, rather than short sleep, was related to increased risk of cardiovascular disease.[66] Thus, future large-scale cohort studies with Mexican American adults should consider including objective measures of sleep such as those obtained using electronic wearables that track sleep, in addition to measuring subjective reports of sleep quality, bed times, and chronotype. Second, the results did not consider other important prospective risk factors for T2D. For example, we did not include socioeconomic status in our models due to the low response rate in the sample (60% of participants had available income data). We also did not have data on pre-diabetic status or dieting behavior at baseline. Third, data were self-reported and vulnerable to recall biases. Although we cross-validated T2D diagnosis with medical records, participants may not have accurately reported the date of diagnosis. Further, sleep duration and physical activity were measured at a low temporal resolution (i.e., within the past year). Future research should consider using objective measures of sleep duration and physical activity via actigraphy. Fourth, we were unable to test change in BMI as a mediator due to substantial missing data at follow-up (41% without weight data). Thus, future prospective studies should consider the role of weight change in the sleep and T2D risk link. Fifth, sleep duration and BMI were measured at the same time point precluding us from disentangling longitudinal relationships between sleep duration and BMI. Consequently, in such a model described as a “half-longitudinal” design,[67] inability to control for prior levels of X and M can bias estimates of the X → M relationship and the indirect effects. Further, while statistical justifications allow for the dual role of BMI as both a mediator and moderator,[68] our models must be interpreted with caution as lack of temporal precedence limits our ability to determine the true direction and nature of the relationships between sleep duration, BMI, and T2D risk. While the timing of the sleep and BMI measures were concurrent, the timing of the constructs were past year sleep and BMI at the day of assessment. Further, substantial theory[23,26,28] guided the hypothesized mediation model. Lastly, we did not have data related to fatigue. Fatigue is prevalent among individuals with T2D[69] and linked to sleep disturbances.[70] Thus, it is possible chronic short sleep, fatigue, and T2D may demonstrate a feedback loop that can undermine treatment efforts. However, additional future research is needed to disentangle these relationships.

Conclusions

In summary, selective effects of sleep duration on T2D risk were identified, and the results highlighted the potential mediating and moderating effects of BMI. Our findings suggest that T2D prevention and control should consider the important role of insufficient sleep among non-obese Mexican American adults. Health practitioners and researchers should consider Hispanic-focused explanatory models of T2D which highlight the role of self-care with

regards to being physically active, obtaining sufficient sleep, and eating a healthy diet as behaviors that help manage and decrease risk for T2D.[71,72]

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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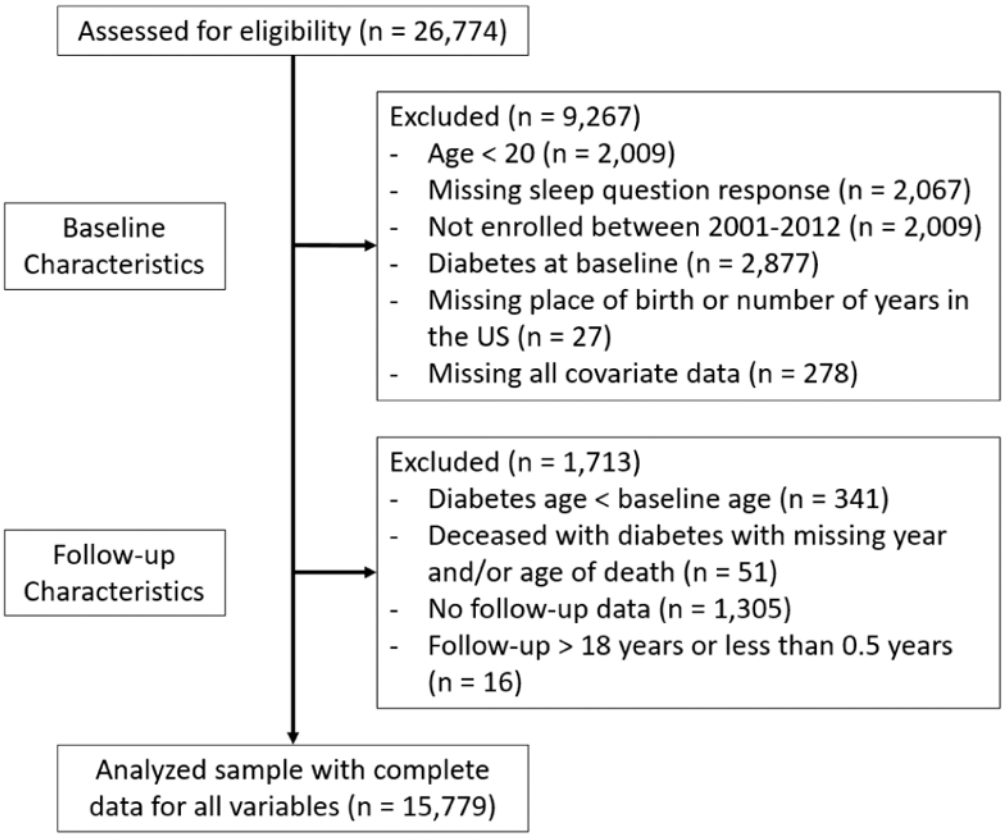

Figure 1.
Study inclusion/exclusion flow chart.

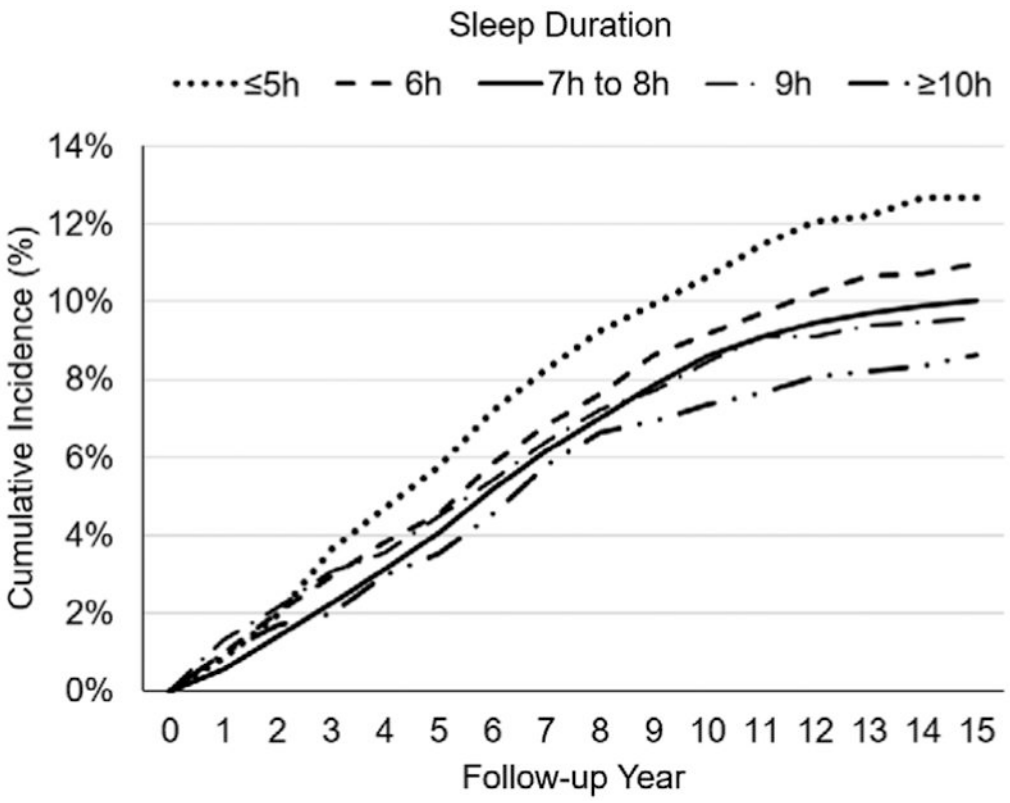

Figure 2.
Cumulative type 2 diabetes incidence by follow-up duration and sleep duration.

Table 1.

Baseline demographic characteristics by self-reported incident type 2 diabetes at follow-up.

	Total	Type 2 diabetes (*N* = 1,628)	No Type 2 diabetes (*N* = 14,151)	
Characteristic	***N* (%)/Mean (*SD*)**	***N* (%)/Mean (*SD*)**	***N* (%)/Mean (*SD*)**	***p***
Age	38.98 (12.7)	42.8 (11.2)	38.5 (12.7)	<.0001
≤ 36 years	8012 (50.8%)	535 (32.9)	7477 (52.8)	
> 36 years	7767 (49.2%)	1093 (67.1)	6674 (47.2)	<.0001
Sex				
Male	3287 (20.8%)	292 (17.94)	2995 (21.16)	
Female	12492 (79.2%)	1336 (82.06)	11156 (78.84)	0.002
Marital status				
Married	12618 (80.0%)	1311 (80.53)	11307 (79.90)	
Not married	3161 (20.0%)	317 (19.47)	2844 (20.10)	0.550
Education				
<High school	9177 (58.2%)	1070 (65.72)	8107 (57.29)	
High school grad	6176 (39.1%)	523 (32.13)	5653 (39.95)	
College	426 (2.7%)	35 (2.15)	391 (2.76)	<.0001
Place of birth				
Mexico	12008 (76.1%)	1236 (75.92)	10772 (76.12)	
US	3771 (23.9%)	392 (24.08)	3379 (23.88)	0.858
Acculturation				
Spanish dominant	9251 (58.6%)	999 (61.74)	8252 (58.75)	
English dominant	1553 (9.8%)	168 (10.38)	1385 (9.86)	
Bilingual	4861 (30.8%)	451 (27.87)	4410 (31.39)	0.015
Body mass index (kg/m^2)				
<25	2777 (17.6%)	71 (4.36)	2706 (19.12)	
25 to <30	5502 (34.9%)	379 (23.28)	5123 (36.20)	
≥30	7500 (47.5%)	1181 (72.36)	6322 (44.68)	<.0001
Cigarette smoking				
Never	11803 (74.8%)	1205 (74.02)	10598 (74.89)	
Former	2052 (13.0%)	232 (14.25)	1820 (12.86)	
Current	1924 (12.2%)	191 (11.73)	1733 (12.25)	0.268
Alcohol drinking				
Never	10653 (67.5%)	1122 (68.92)	9531 (67.35)	
Former	1341 (8.5%)	149 (9.15)	1192 (8.42)	
Current	3785 (24.0%)	357 (21.93)	3428 (24.22)	0.098
Physical activity guidelines				
Not meeting	12442 (78.9%)	1321 (81.14)	11121 (78.59)	
Meeting	3337 (21.2%)	307 (18.86)	3030 (21.41)	0.017
Sleep duration (h)				
≤5	1318 (8.4%)	167 (10.26)	1151 (8.13)	

	Total	Type 2 diabetes (*N* = 1,628)	No Type 2 diabetes (*N* = 14,151)	
Characteristic	***N* (%)/Mean (*SD*)**	***N* (%)/Mean (*SD*)**	***N* (%)/Mean (*SD*)**	***p***
6	2844 (18.0%)	312 (19.16)	2532 (17.89)	
7-8	9636 (62.3%)	985 (60.50)	8851 (62.55)	
9	1075 (6.8%)	103 (6.33)	872 (6.87)	
≥10	706 (4.5%)	61 (3.75)	645 (4.56)	0.012

Note. SD = standard deviation; h = hours.

Table 2.

Sleep duration predicting type 2 diabetes (T2D) risk across sociodemographic and lifestyle factors.

Model	Sleep (Hours)*	T2D/ No T2D	HR	95% CI		p
Overall Unadjusted	≤5	167/1151	**1.315**	**1.116**	**1.550**	**0.001**
	6	312/2532	1.104	0.972	1.254	0.127
	7-8	985/8851	1 [Ref]	--	--	--
	9	103/972	0.975	0.796	1.195	0.810
	≥10	61/645	0.914	0.706	1.183	0.494
			Adj HR	**95% CI**		**p**
Overall Adjusted	≤5	167/1151	1.153	0.977	1.360	0.092
	6	312/2532	1.067	0.938	1.212	0.324
	7-8	985/8851	1 [Ref]	--	--	--
	9	103/972	0.980	0.800	1.201	0.846
	≥10	61/645	0.954	0.736	1.236	0.720
BMI (kg/m^2)						
< 25	≤5	9/165	2.034	0.968	4.274	0.061
	6	16/472	1.521	0.846	2.732	0.161
	7-8	39/1732	1 [Ref]	--	--	--
	9	5/193	1.35	0.528	3.451	0.530
	≥10	2/144	0.697	0.168	2.893	0.619
25 to < 30	≤5	35/382	1.159	0.811	1.657	0.418
	6	69/892	1.041	0.795	1.363	0.772
	7-8	238/3313	1 [Ref]	--	--	--
	9	25/340	1.038	0.687	1.570	0.858
	≥10	12/196	1.002	0.558	1.800	0.994
≥ 30	≤5	123/604	1.109	0.915	1.345	0.291
	6	227/1168	1.052	0.905	1.223	0.510
	7-8	708/3806	1 [Ref]	--	--	--
	9	73/439	0.956	0.751	1.217	0.717
	≥10	47/305	0.956	0.711	1.286	0.767
Sex						
Male	≤5	30/278	1.041	0.706	1.536	0.839
	6	48/605	0.822	0.597	1.131	0.229
	7-8	185/1887	1 [Ref]	--	--	--
	9	15/147	0.942	0.556	1.596	0.823
	≥10	14/78	**1.970**	**1.133**	**3.424**	**0.016**
Female	≤5	137/873	1.174	0.978	1.409	0.086
	6	264/1927	1.125	0.978	1.294	0.100
	7-8	800/6964	1 [Ref]	--	--	--
	9	88/825	0.987	0.791	1.230	0.904
	≥10	47/567	0.826	0.615	1.110	0.205

Note. Adjusted model includes age, sex, education, marital status, birth country, BMI, acculturation, alcohol, smoking, and physical activity as covariates. HR = hazard ratio; CI = confidence interval.

*Reference group is 7-8 hours. Bolded highlights statistically significant effects.

Table 3.

Sleep duration on type 2 diabetes risk across BMI at 3, 5, and >5 year follow up.

Characteristic	Sleep (Hours)*	Adj. HR	95% CI		*p*
3 Year Follow-Up					
BMI group (kg/m^2)					
< 25	≤5	**4.131**	**1.202**	**14.201**	**0.024**
	6	1.798	0.538	6.015	0.341
	7-8	1 [Ref]	--	--	--
	9	2.733	0.569	13.128	0.209
	≥10	0.000	0.000	0.000	0.993
25 to < 30	≤5	1.709	0.789	3.703	0.174
	6	**1.827**	**1.031**	**3.237**	**0.039**
	7-8	1 [Ref]	--	--	--
	9	1.757	0.737	4.188	0.204
	≥10	1.173	0.279	4.932	0.828
< 30	≤5	**2.209**	**1.160**	**4.210**	**0.016**
	6	**1.781**	**1.063**	**2.984**	**0.028**
	7-8	1 [Ref]	--	--	--
	9	1.900	0.891	4.051	0.097
	≥10	0.819	0.197	3.400	0.784
≥ 30	≤5	1.173	0.818	1.682	0.387
	6	1.118	0.836	1.495	0.452
	7-8	1 [Ref]	--	--	--
	9	1.265	0.832	1.924	0.271
	≥10	0.892	0.496	1.602	0.701
5 Year Follow-Up					
BMI group (kg/m^2)					
< 25	≤5	**3.733**	**1.413**	**9.867**	**0.008**
	6	1.434	0.554	3.710	0.458
	7-8	1 [ref]	--	--	--
	9	1.944	0.558	6.770	0.296
	≥10	0.909	0.120	6.891	0.927
25 to < 30	≤5	1.302	0.736	2.303	0.364
	6	1.194	0.775	1.839	0.421
	7-8	1 [ref]	--	--	--
	9	1.232	0.638	2.380	0.535
	≥10	1.186	0.477	2.954	0.713
< 30	≤5	**1.689**	**1.040**	**2.744**	**0.034**
	6	1.220	0.824	1.806	0.321
	7-8	1 [Ref]	--	--	--
	9	1.341	0.750	2.395	0.322

Characteristic	Sleep (Hours)*	Adj. HR	95% CI		*p*
	≥10	1.079	0.471	2.469	0.858
≥ 30	≤5	1.119	0.839	1.491	0.444
	6	1.033	0.819	1.303	0.782
	7-8	1 [ref]	--	--	--
	9	1.076	0.758	1.527	0.684
	≥10	0.870	0.547	1.385	0.558
>5 Year Follow-Up					
BMI group (kg/m^2)					
< 25	≤5	2.034	0.968	4.274	0.061
	6	1.521	0.846	2.732	0.161
	7-8	1 [Ref]	--	--	--
	9	1.350	0.528	3.451	0.530
	≥10	0.697	0.168	2.893	0.619
25 to < 30	≤5	1.159	0.811	1.657	0.418
	6	1.041	0.795	1.363	0.772
	7-8	1 [Ref]	--	--	--
	9	1.038	0.687	1.570	0.858
	≥10	1.002	0.558	1.800	0.994
< 30	≤5	1.331	0.966	1.835	0.080
	6	1.089	0.853	1.391	0.492
	7-8	1 [Ref]	--	--	--
	9	1.073	0.736	1.565	0.715
	≥10	0.869	0.507	1.491	0.611
≥ 30	≤5	1.109	0.915	1.345	0.291
	6	1.052	0.905	1.223	0.510
	7-8	1 [Ref]	--	--	--
	9	0.956	0.751	1.217	0.717
	≥10	0.956	0.711	1.286	0.767

Note. Models adjusted for age, sex, education, marital status, birth country, acculturation, alcohol, smoking, and physical activity. HR = hazard ratio; CI = confidence interval. Bolded highlights statistically significant effects.

Table 4.

Total, direct, and indirect effects of baseline sleep duration on prospective type 2 diabetes risk through baseline obesity.

Sleep Duration	Total effect[T]	Obesity independent[*]	Obesity mediated[*]	Interaction	Proportion
Hours	**HR [95% CI]**	**HR [95% CI]**	**HR [95% CI]**	***b* (SE)**	**%**
≤ 5	1.274 [1.078, 1.507]	1.188 [1.006, 1.402]	1.073 [1.038, 1.109]	−0.217 (0.185)[ns]	31.60
6	0.160 [0.964, 1.251]	1.071 [0.942, 1.218]	1.025 [1.002, 1.048]	−0.010 (0.142)[ns]	27.30
7-8 (ref)	--	--	--	--	--
9	0.988 [0.812, 1.227]	0.988 [0.806, 1.211]	1.010 [.979, 1.043]	−0.081 (0.225)[ns]	--
≥ 10	0.952 [0.762, 1.290]	0.954 [0.736, 1.237]	1.039 [.993, 1.088]	0.128 (0.310)[ns]	--

Note. Effect estimates represent the effect of sleep duration relative to 7-8 hours on type 2 diabetes including covariates with ("Total Effect") and without ("Obesity independent") obesity, and the indirect effect through obesity including covariates ("Obesity mediated"). Interaction = estimate for sleep duration X obesity predictor. Proportion = percent of variance explained by obesity status. HR = hazard ratio. CI = confidence interval. SE = standard error. NS = not significant at $p < .05$ level.

[*] Adjusted for age, sex, education, marital status, birth country, acculturation, alcohol and smoking, and physical activity.

[T] Adjusted for above including obesity.