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Multiple sleep dimensions and type 2 diabetes risk among women in the Sister Study: differences by race/ethnicity

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ABSTRACT

Objective Poor sleep has been associated with type 2 diabetes. Since racial/ethnic minorities experience a disproportionately high prevalence of poor sleep and type 2 diabetes, we sought to determine the relationships between multiple sleep dimensions and incident type 2 diabetes and to investigate if these relationships vary by race/ethnicity.

Research design and methods Prospective data were analyzed from the Sister Study, which enrolled 50 884 women from 2003 to 2009. Participants self-reported sleep duration, sleep latency, night awakenings, and napping at baseline, and a physician's diagnosis of type 2 diabetes at follow-up. Multivariable-adjusted HRs and 95% CIs were estimated using Cox proportional hazards models.

Results Among the 39 071 eligible participants, 87% self-identified as white, 8% black and 5% Hispanic/Latina. The mean follow-up period was 8.5±2.1 years and 1785 type 2 diabetes cases were reported. The incidence rate per 1000 person-years was 5.4 for whites, 13.3 for blacks and 11.6 for Hispanics/Latinas. There was a positive but non-significant increased risk of type 2 diabetes among women who reported short sleep, latency >30 min and frequent night awakenings. In fully-adjusted models, frequent napping was associated with a 19% (HR 1.19, 95% CI 1.04 to 1.37) higher type 2 diabetes risk in the overall sample. Poor sleep among racial/ethnic minorities ranged from a 1.4-fold to a 3.2-fold higher type 2 diabetes risk than whites with recommended sleep.

Conclusions Frequent napping was associated with higher type 2 diabetes risk. Racial/ethnic minorities with poor sleep had a higher type 2 diabetes risk than whites with recommended sleep.

Significance of this study

What is already known about this subject?

- Poor sleep has been associated with type 2 diabetes in previous studies, but more prospective data are necessary to help establish it as a risk factor.
- Racial/ethnic minorities experience a disproportionately high prevalence of both poor sleep and type 2 diabetes, but few studies have explicitly investigated if racial/ethnic differences in the relationship exist.

What are the new findings?

- We observed positive but non-significant associations between all sleep dimensions and type 2 diabetes risk, with the exception of a significantly increased type 2 diabetes risk among women who reported frequent napping.
- Black and Hispanic/Latina women remained at a significantly increased risk for type 2 diabetes across every poor sleep characteristic when compared with white women with recommended sleep, and frequent napping was most strongly associated with type 2 diabetes risk among blacks and night awakenings among Hispanics/Latinas.

How might these results change the focus of research or clinical practice?

- The results of this study contribute prospective data demonstrating that unfavorable sleep characteristics are associated with type 2 diabetes risk and should be considered a risk factor.
- High-risk individuals, especially racial/ethnic minorities, may need to be screened and treated for unfavorable sleep as a means of preventing or delaying type 2 diabetes, but more research is warranted.

INTRODUCTION

Type 2 diabetes is of great public health concern. Between 1990 and 2010, the number of US adults living with diabetes tripled, and the number of incident cases doubled.[1] It is estimated that by 2040, 1 in 10 adults will suffer from diabetes.[2] In particular, between 2005 and 2050, diabetes prevalence is expected to increase by 99% among non-Hispanic whites (from 5.4% to 10.6%), by 107% among non-Hispanic blacks (from 7.4% to 15.3%), by 127% among Hispanics (from 5.5% to 12.4%), and by 158% among other races (from 5.4% to 14.0%).[3]

Poor sleep is pervasive and has been positively associated with common metabolic conditions like obesity, hypertension, and type 2 diabetes.[4–14] Experimental and observational data suggest that various parameters

of sleep can lead to upregulation of the hunger-inducing ghrelin hormone and downregulation of the leptin hormone, which is associated with satiety.[15 16] Dysregulation of these hormones may lead to overeating and eventually obesity—a strong risk factor for type 2 diabetes. Data also suggest that poor sleep can contribute to diabetes risk through insulin resistance and glucose intolerance by compromising homeostatic metabolic pathways.[17] Additionally, sleep restriction may activate reward areas of the brain that lead to greater propensity to overeat, thereby contributing to diabetes risk.[18] Black and Hispanic/Latino populations have been consistently shown to have disproportionately higher prevalences of poor sleep and type 2 diabetes than their white counterparts,[10 14 19–23] with some studies estimating that blacks, Hispanic/Latinos and Asians are more than twice as likely as whites to report very short sleep (<5 hours).[20] Understanding whether race/ethnicity modifies the sleep–diabetes relationship may provide additional strategies to address these recalcitrant disparities. Previous studies have published mixed results regarding the impact of race/ethnicity on the association between poor sleep and type 2 diabetes.[10 24] While a nationally representative cross-sectional US study concluded that black participants with short or long sleep duration had a greater prevalence of diabetes independent of sociodemographic or medical characteristics,[24] another representative study found that socioeconomic status attenuates both the positive association between short sleep and diabetes in black individuals and the racial/ethnic difference in the short sleep–diabetes association.[10] Furthermore, a cross-sectional study among white, Filipina and African-American postmenopausal women found an association between sleep duration and prevalent diabetes in Filipina women only and an association between daytime napping and diabetes among only white women.[12] A prospective study found a positive association between short sleep and diabetes risk among non-Hispanic whites and Hispanics and reported a non-significant negative association for African-Americans.[25] However, in a meta-analysis of 10 prospective studies of over 107 000 participants, short (28%) and long (48%) sleep duration and difficulty in initiating (57%) and maintaining (84%) sleep were associated with increased risk of type 2 diabetes.[4] While the number of prospective studies examining the sleep–diabetes relationship in recent years has nearly doubled,[6] most existing studies and most prospective as well as experimental studies include mainly men,[17 26 27] do not include sufficient racial/ethnic minorities, and lack measurement of sleep dimensions beyond sleep duration.

Therefore, using a prospective cohort of US women, we aimed to determine the independent relationship between multiple measures of poor sleep and type 2 diabetes risk and to examine whether these relationships vary by race/ethnicity. We hypothesized that women with poor sleep dimensions will have higher incidence of type 2 diabetes than women with recommended sleep dimensions and that this relationship will be stronger for racial/ethnic minority women.

METHODS

The Sister Study

We used data from the Sister Study (Data Release 6.0), a prospective cohort of 50 884 women aged 35–74 years from the continental US including Puerto Rico focused on identifying environmental and genetic risk factors for breast cancer.[28] From 2003 to 2009, a national multimedia campaign and a network of professionals and volunteers recruited women who had a sister diagnosed with breast cancer but were themselves free of breast cancer. Special efforts were made to recruit older women, racial/ethnic minority women, and women of lower socioeconomic status. Collection of baseline sociodemographic, medical, and lifestyle data included a computer-assisted telephone interview and self-administered questionnaires. Follow-up consisted of brief annual health updates and detailed questionnaires approximately every 2 or 3 years. All participants provided written informed consent.

Study participants

Participants included non-Hispanic white, non-Hispanic black (referred to hereafter as white and black), and Hispanic/Latina women. Among 50 884 participants, women were excluded if they had type 1 diabetes (n=122). Type 1 diabetes was defined as: (1) self-report of type 1 diabetes, (2) being diagnosed with diabetes before age 20 years, or (3) being diagnosed with diabetes between the ages of 20 and 35 years, taking insulin continuously, and having a difference of a year or less between the start of taking insulin medication and the diagnosis of diabetes. Participants were further excluded if they answered yes (as well as 'refused' or responded 'do not know') to having prevalent or borderline type 2 diabetes at baseline or currently taking insulin or diabetes medications (n=4312); having unknown timing of diabetes onset (n=479); pregnant or breastfeeding (n=54); and self-identifying their race/ethnicity as other than white, black or Hispanic/Latina (n=1154, 3%). Participants were also excluded if they were missing age at type 2 diabetes diagnosis (n=49), race/ethnicity (n=15), or sleep data (n=179); had implausible sleep values (awakening more than 20 times per night, n=2); or had history of cancer excluding non-melanoma skin cancer (n=2632), stroke (n=407) or transient ischemic attack (n=724), or heart disease (angioplasty (n=313), angina (n=934), heart attack (n=208), congestive heart failure (n=168), or heart bypass surgery (n=61)). The final analytic sample consisted of 39 071 women.

Exposure: sleep dimensions

Assessment of multiple sleep dimensions

At baseline, participants reported their average number of sleep duration hours per night/day, the average number of minutes they take to fall asleep (latency hereafter), the average number of times per week

they wake up while sleeping (and number of times per night/day they wake up once asleep, or night awakenings), and the average number of times they nap per week. Sleep duration was classified as <7 hours (short), 7–9 hours (recommended), and >9 hours (long).[29] Latency was dichotomized as 'no' (≤30 min) versus 'yes' (>30 min). Night awakenings was classified as 'no' (<3 times per night/day) versus 'yes' (≥3 times per night/day ≥3 times per week). Frequent napping was categorized as 'no' (<3 times per week) versus 'yes' (≥3 times per week). Napping frequency was examined and categorized as 'never', '<once per month', '≥once per month and <3 times per week), and ≥3 times per week. A cumulative sleep score was developed by assigning a value of 1 to 'yes' responses for short sleep duration, long latency, frequent night awakenings, and frequent napping, and taking the sum of these values for each participant. The cumulative sleep score was categorized as '0', '1', and '2 to 4.'

Outcome: type 2 diabetes

Type 2 diabetes

Participants were asked, 'Has a doctor or other health care provider ever told you that you had diabetes or high blood sugar, or that you had borderline diabetes other than during pregnancy?'. The results were dichotomously coded as 'yes' or 'no'. Participants who reported 'yes' and were not assumed to have type 1 diabetes (as described above) were considered as having type 2 diabetes. Incident diabetes was ascertained in annual follow-up questionnaires until 15 September 2016.

Confounders

All covariates were measured at baseline and based on self-report, except body mass index (BMI), which was measured by an examiner.

Age

Age (years) was measured continuously.

Race/ethnicity

Participants self-identified their race/ethnicity after being asked 'What race do you consider yourself to be? You may choose one or more of the following' and 'Do you consider yourself to be Hispanic or Latina?'. Categories were those defined using the Office of Management and Budget according to Standards for the Classification of Federal Data on Race and Ethnicity used in all Federal surveys.[30]

Educational attainment

Educational attainment was classified as less than high school (<HS), HS or general education degree, some college, or at least a bachelor's degree (≥bachelor).

Shift work

Participants were asked if their current job included irregular hours or shift work (yes/no). Participants were also asked if they had ever worked shifts at ≥1 job, followed by the total years they had worked shifts (corrected for job overlap). Years of shift work was classified as '0', '1–2', '3–4' and '>4' in the analysis.

Health behaviors

Both smoking and alcohol consumption status were classified as 'Current', 'Past', or 'Never'. Dietary information was assessed using a standardized self-administered food frequency questionnaire (FFQ), a modified version of the 110-item 1998 Block FFQ (NutritionQuest, Berkeley, California, USA), and average daily glycemic load (glucose scale) was calculated.[31] Physical activity was assessed by total metabolic equivalent of task (MET) hours per week, summed over all reported sports/exercise and daily activities.

Clinical characteristics

Height (cm), weight (kg), waist (cm), and hip (cm) measurements were taken by examiners at baseline. BMI (kg/m^2) was calculated from height and weight measurements, and waist-to-hip ratio (WHR) was calculated by dividing waist circumference by hip circumference. Diastolic and systolic blood pressure (BP) measurements were taken by examiners and was averaged over three measurements. Hypertension was defined as having a systolic BP of ≥130 mm Hg, or diastolic BP of ≥80 (according to recommendations of the American College of Cardiology/American Heart Association Task Force),[32] or self-report of a physician's diagnosis of hypertension and antihypertensive medication use.

Potential modifiers: Sleep medication, menopausal status, and depression

Sleep medication was classified as 'yes' if participants reported using prescription sleep medications, antihistamines (for any reason), or melatonin. Women were considered postmenopausal if they had gone >12 months without menstruating, had both ovaries removed, or underwent a hysterectomy with ovarian retention and were >55 years old. Depression status was determined by self-report of a physician's diagnosis of clinical depression (yes/no).

Statistical analysis

Person-time in the overall sample and for each sleep and race/ethnicity category was accumulated from baseline until the: (1) participants were diagnosed with type 2 diabetes, (2) end of follow-up (15 September 2016), or (3) participant experienced loss to follow-up or death. Incidence rates in the overall sample and in each sleep and race/ethnicity category were calculated by dividing the number of type 2 diabetes events by person-time (in years) of follow-up. Incidence rates for type 2 diabetes were represented per 1000 person-years. We tested the proportional hazards assumption by creating interactions of the exposure and covariates and a function of survival time. Multivariable Cox proportional hazards models were used to assess HRs

and 95% CIs for the associations between sleep and type 2 diabetes event. Model I included adjustment for age (in years) measured continuously. Model II was further adjusted for race/ethnicity, educational attainment, and shift work/working irregular hours (yes/no) as confounders. Model III was additionally adjusted for smoking status, alcohol consumption, diet, and physical activity. Model IV was also adjusted for WHR, BMI, and hypertension. Model V was model IV without adiposity measures WHR and BMI.

We examined a potential interaction of sleep with race/ethnicity by adding interaction terms to each model to determine the impact of race/ethnicity on the relationship between sleep and diabetes risk, using white women with recommended sleep as a reference group. We then separately stratified by race/ethnicity, shift/irregular work hours at baseline, BMI classification (under/normal weight, overweight, and obese), and age category (35–44, 45–54, 55–64, 65+ years) to determine their roles as modifiers of the association between sleep and diabetes. We performed separate sensitivity analyses to examine differences in estimates of the impact of sleep on diabetes by excluding participants who reported sleep medication use, person years and cases diagnosed in the first year to address potential confounding by symptoms related to undiagnosed diabetes, postmenopausal women, and women with depression. SAS statistical software, V.9.4 was used for all analyses, and a two-sided p value <0.05 was considered statistically significant.

RESULTS

Among 39 071 women, mean age was 54.8±8.8 years with 87% self-identifying as white, 8% black, and 5% Hispanic/Latina (table 1). The majority of women had an educational attainment level ≥bachelor's (53%), never worked shift or irregular hours (62%), were current drinkers (83%), never smoked (57%), had a mean BMI classified as overweight (27.2±5.8), and were postmenopausal (62%). Eleven percent reported recent use of sleep medications, 37% were hypertensive, and 19% reported a physician's diagnosis of clinical depression. Participants who reported poor sleep were slightly more likely to have less than a college degree, use sleep medication, report being a former drinker or current smoker, and have suboptimal clinical characteristics (eg, higher daily glycemic load, higher BMI, postmenopausal, hypertension, and clinical depression). Blacks and Hispanics/Latinas were more likely than whites to report short sleep (53% and 37% vs 25%), longer latency (27% and 27% vs 15%), and frequent napping (13% and 13% vs 9%) (online supplementary table 1). Blacks reported the lowest frequent night awakenings compared with whites and Hispanics/Latinas (11% vs 13% and 14%). Black (27%) and Hispanic/Latina (23%) women were more likely to experience multiple poor sleep dimensions (cumulative sleep score of 2–4) compared with white (14%) women.

Overall association between poor sleep and type 2 diabetes risk

Diabetes incidence rates across sleep dimensions in the overall sample are provided in table 2. There were non-significant positive associations with type 2 diabetes risk for all sleep dimensions. Only frequent napping remained statistically significant in fully adjusted models (HR 1.19 (95% CI 1.04 to 1.37)). When examining the four-category napping variable (online supplementary table 9), napping ≥once per month and <3 times per week and ≥3 times per week remained significant in fully adjusted models. A dose–response was observed for poor sleep but was attenuated after adjusting for clinical characteristics, including WHR, BMI, and hypertension. There were non-significant associations for a cumulative sleep score of 1 ($HR_{sleep\ score=1}$ 1.04 (95% CI 0.93 to 1.16)) but a significantly higher risk of type 2 diabetes for women with a cumulative sleep score of 2–4 ($HR_{sleep\ score=2\text{-}4}$ 1.15 (95% CI 1.01 to 1.30)).

Impact of race/ethnicity and poor sleep on type 2 diabetes risk

With the exception of frequent night awakenings among black women, all sleep measures for blacks and Hispanics/Latinas compared with white women with recommended sleep were significantly associated with type 2 diabetes risk in fully adjusted models, ranging from 41% to an over threefold increased risk (table 3). For white women, only napping frequency was significantly associated with type 2 diabetes risk in fully adjusted models (HR 1.22 (95% CI 1.04 to 1.43)). Hispanic/Latina participants who reported frequent night awakenings (HR 3.18 (95% CI 2.24 to 4.51)) had the highest type 2 diabetes risk and, among black women, those who reported frequent napping (HR 1.65 (95% CI 1.20 to 2.27)) had the highest type 2 diabetes risk.

In table 4, associations between poor sleep and diabetes risk are stratified by race/ethnicity. Except for frequent napping for whites (HR 1.20 (95% CI 1.02 to 1.41)) and frequent night awakenings for Hispanics/Latinas (HR 1.93 (95% CI 1.27 to 2.92)), there were no other significant associations between sleep dimensions and risk of type 2 diabetes based on within-race/ethnicity comparisons.

Modification of the sleep–diabetes relationship

Although risk ratios did not reach statistical significance in fully adjusted models, there was generally a higher risk of type 2 diabetes among women working shift/irregular hours versus not working shift/irregular hours across all sleep dimensions (online supplementary table 2). When stratified by BMI classification, the association between poor sleep and type 2 diabetes risk was attenuated with higher BMI class and strengthened with increasing cumulative sleep score for underweight/normal and

Table 1 Sociodemographic characteristics, health behaviors, and clinical characteristics overall and by sleep characteristics among 39 071 adult women in the Sister Study

		Sleep duration*		Time to fall asleep		Frequent night awakenings†		Napping frequency	
	All	Recommended (7–9 hours)	Short (<7 hours)	≤30 min	>30 min	No	Yes	<3 times/ week	≥3 times/ week
Sample size, *N* (%)	39 071	27 880 (72)	10 835 (28)	32 566 (83)	6505 (17)	34 025 (87)	5046 (13)	35 390 (91)	3681 (9)
Sociodemographic characteristics									
Age, years	54.8±8.8	54.9±9.0	54.6±8.5	54.8±8.8	55.2±8.8	54.7±8.9	56.0±8.4	54.6±8.7	57.5±9.5
Race/ethnicity									
White	87	91	79	88	79	87	88	88	83
Black	8	5	15	7	13	8	7	8	11
Hispanic/Latina	5	4	6	4	8	5	5	5	6
Educational attainment									
<HS	1	1	1	1	2	1	2	1	2
HS/GED	13	13	15	13	17	13	16	13	15
Some college	33	32	35	32	38	32	36	32	35
≥Bachelor's	53	55	49	55	43	54	46	54	48
Marital status									
Married or living as married	76	79	70	77	71	76	76	77	71
Single/never married	5	5	7	5	7	6	5	5	6
Widowed/separated/ divorced	19	17	23	18	22	18	19	18	23
Currently working shift/ irregular hours, yes	15	15	16	15	15	15	15	15	14
Total years worked shifts									
None	62	62	62	62	62	62	61	62	63
1–2	3	3	3	3	3	3	4	3	3
3–4	8	9	8	9	8	8	8	9	7
>4	26	26	27	26	26	26	27	26	27
Sleep characteristics									
Cumulative sleep score‡									
0	53	73	0	63	0	60	0	58	0
1	32	23	54	30	40	31	40	30	53
2–4	16	4	46	7	60	9	60	12	47
Recent sleep medication use, yes§	11	10	12	9	19	10	15	10	13
Health behaviors									
Alcohol consumption status									
Current	83	84	81	84	79	84	81	84	76
Past	13	13	15	13	16	13	15	13	19
Never	3	3	4	3	4	3	4	3	5
Smoking status									
Current	8	7	10	7	11	8	8	7	10
Past	35	35	33	35	33	34	38	35	33
Never	57	58	57	58	56	58	54	57	57
Diet (daily glycemic load)	84.9±39.1	84.0±37.3	87.0±43.0	84.2±37.6	88.1±45.6	84.6±38.5	86.9±42.6	83.9±38.2	94.4±45.1

Continued

Table 1 Continued

	All	Sleep duration*		Time to fall asleep		Frequent night awakenings†		Napping frequency	
		Recommended (7–9 hours)	Short (<7 hours)	≤30 min	>30 min	No	Yes	<3 times/ week	≥3 times/ week
Physical activity (Metabolic equivalent of task hours)	51.6±31.4	51.6±31.0	51.6±32.4	51.7±31.3	50.8±31.8	51.5±31.3	51.6±31.7	51.8±31.3	49.5±31.6
Clinical characteristics									
Mean waist-to-hip ratio	0.80±0.08	0.80±0.08	0.81±0.07	0.80±0.08	0.81±0.07	0.80±0.08	0.81±0.07	0.80±0.08	0.81±0.07
Waist circumference (cm) over NHLBI guidelines (yes)	36	34	41	35	43	36	41	35	46
Mean BMI (kg/m^2)	27.2±5.8	26.9±5.6	28.0±6.3	27.0±5.8	28.0±6.0	27.1±5.8	27.7±6.2	27.1±5.8	28.4±6.4
Menopause, yes	62	62	63	61	67	61	71	61	71
Hypertension, yes	37	36	41	36	42	37	41	36	46
Clinical depression, yes	19	19	19	18	27	19	24	19	27

Waist circumference>88 cm for women was defined as exceeding guidelines per the National Heart, Lung and Blood Institute (NHLBI).
The following variables had missing data: diet (daily glycemic load), n=843 (2%). All other variables with missing values were <1% of the total sample.
*Long sleep duration (>9 hours) is not reported (n=356).
†Frequent night awakenings was classified as 'No' if participant reported waking up while sleeping less than three times per week and 'Yes' if participant reported waking up while sleeping more than three times per night/day for more than three nights/days per week.
‡Cumulative sleep score was calculated by summing each participant's 'yes' response for each sleep characteristic. Scores range from 0 to 4, with poor sleep characteristics being combined into the category '2 to 4'.
§Recent sleep medication use was defined as use of the following medications within the past 4 weeks: inclusive of medication classes containing medications specifically used for sleep (directly affecting sleep), antihistamines and melatonin.
HS, high school; GED, general education diploma; BMI, body mass index.

obese participants (online supplementary table 3). In analysis stratified by age category, only women who were older than 65 years had a dose–response relationship with latency >30 min (HR=1.44 (95% CI 1.09 to 1.91)) and increased poor sleep dimensions ($HR_{sleep\ score=1}$ 1.02 (95% CI 0.77 to 1.36) and $HR_{sleep\ score=2–4}$ 1.50 (95% CI 1.10 to 2.02)) (online supplementary table 4).

Potential modifiers: sleep medications, menopausal status, and depression

When separately excluding women who reported sleep medication use, incident cases of type 2 diabetes during the first year of follow-up, postmenopausal women, or women with depression, sleep–diabetes associations remained significant across sleep dimensions but attenuated with adjustment for hypertension and adiposity measures (online supplementary tables 5–8).

DISCUSSION

Among a large cohort of women, we observed positive but non-significant associations between all sleep dimensions and type 2 diabetes risk, with the exception of a significantly increased type 2 diabetes risk among women who reported frequent napping. After controlling for adiposity measures, including WHR and BMI, blacks and Hispanics/Latinas remained at a significantly increased risk for type 2 diabetes across every poor sleep characteristic when compared with white women with recommended sleep. Among these comparisons, frequent napping was most strongly associated with type 2 diabetes risk for blacks and night awakenings for Hispanics/Latinas. When making within-race comparisons, only white women who reported napping frequently and Hispanic/Latina women who reported frequent night awakenings were at increased type 2 diabetes risk.

Although sleep duration may play a key role in the risk of developing diabetes, napping frequency, which is thought to be an indicator of sleep quality and/or disease severity, has been both cross-sectionally and longitudinally associated with diabetes.[33 34] Results from our study are consistent with prior studies that have reported significant longitudinal associations between both quantity and quality of sleep and incident type 2 diabetes.[4] In a study exclusively examining women in the Nurses' Health Study, Ayas *et al*[35] found that women with short sleep duration (defined as ≤5 hours) had a 57% increased risk of type 2 diabetes. Similar to our results, in multivariable models, the risk for women who reported short sleep was no longer significantly associated with type 2 diabetes after adjustment for BMI. In contrast, a previous study of a small sample of white women with a 32-year follow-up period reported no association between poor sleep (reporting sleep complaints or sleep duration <6 hours in a 24-hour period) and developing diabetes. However, these results may be explained by the small sample size in models investigating diabetes.[36]

Table 2 HRs (95% CI) for type 2 diabetes risk among 39 071 adult women in the Sister Study, with women with poor sleep compared with women with recommended sleep

	T2DM incidence rate	Model I	Model II	Model III	Model IV	Model V
	Per 1000 person-years	HR (95% CIs)	HR (95% CIs)	HR (95% CIs)	HR (95% CIs)	HR (95% CIs)
Sleep duration*						
Short sleep versus	6.6	**1.34 (1.22 to 1.48)**	**1.13 (1.02 to 1.26)**	1.11 (1.00 to 1.23)	1.03 (0.93 to 1.14)	1.09 (0.98 to 1.21)
Recommended (reference)	4.9					
Time to fall asleep						
>30 min versus	6.9	**1.36 (1.22 to 1.53)**	**1.18 (1.05 to 1.32)**	**1.14 (1.01 to 1.28)**	1.08 (0.96 to 1.22)	1.11 (0.99 to 1.25)
≤30 min (reference)	5.1					
Frequent night awakenings						
Yes† versus	6.5	**1.22 (1.07 to 1.39)**	**1.19 (1.04 to 1.35)**	**1.17 (1.03 to 1.34)**	1.09 (0.95 to 1.24)	1.14 (1.00 to 1.30)
No (reference)	5.2					
Napping frequency						
≥3 times/week versus	8.4	**1.65 (1.45 to 1.89)**	**1.50 (1.32 to 1.72)**	**1.36 (1.18 to 1.56)**	**1.19 (1.04 to 1.37)**	**1.30 (1.13 to 1.49)**
<3 times/week (reference)	5.1					
Cumulative sleep score‡						
1 versus	5.7	**1.27 (1.14 to 1.41)**	**1.14 (1.02 to 1.27)**	**1.09 (0.98 to 1.22)**	1.03 (0.93 to 1.15)	1.08 (0.96 to 1.20)
0 (reference)	4.5					
2–4 versus	7.8	**1.72 (1.52 to 1.94)**	**1.40 (1.24 to 1.59)**	**1.31 (1.15 to 1.49)**	**1.14 (1.00 to 1.30)**	**1.25 (1.10 to 1.42)**
0 (reference)	4.5					

Model I: adjusted for age (years) at baseline.
Model II: further adjusted for race/ethnicity, educational attainment (<HS, HS/GED, some college, and ≥bachelor's degree), marital status, and status of shift work/irregular hours (yes/no).
Model III: further adjusted for smoking status (current, past, andnever), alcohol consumption (current, past, and never), diet (daily glycemic load), and physical activity (MET hours).
Model IV: further adjusted for hypertension, waist-to-hip ratio (WHR), and BMI (kg/m^2) measured at baseline.
Model V: without WHR and BMI.
HRs are in bold.
*Long sleep duration (>9 hours) is not reported (n=356).
†Frequent night awakenings was classified as 'No' if participant reported waking up while sleeping less than three times per week and 'Yes' if participant reported waking up while sleeping more than three times per night/day for more than three nights/days per week.
‡Cumulative sleep score was calculated by summing each participant's 'yes' response for each sleep characteristic. Scores range from 0 to 4, with poor sleep characteristics being combined into the category '2 to 4'.
BMI, body mass index; GED, general education diploma;HS, high school; MET, metabolic equivalent of task.

Findings from a previous study showed ethnic differences in the associations of self-reported sleep duration and risk of diabetes.[12] Although this study included white, black, and Filipino women while our study included white, black, and Hispanic/Latina women, we also observed racial/ethnic differences in incident type 2 diabetes. When compared with white women with recommended sleep, women in all racial/ethnic groups who reported poor sleep had increased risk of developing type 2 diabetes, with blacks and Hispanics/Latinas experiencing the greatest risk across all sleep dimensions. A previous study of older US adults by Kaufmann *et al*[37] examined racial/ethnic differences in insomnia severity over time, which was characterized as having trouble falling asleep, having trouble waking up during the night, waking up too early and not being able to fall asleep again, and not feeling fully rested. The authors found that insomnia severity was disproportionately high in Hispanics compared with whites and blacks, even after controlling for all potential confounders, including BMI.[37] The consistent findings from our study and the Kaufmann *et al* study may be attributed to Hispanics/Latinos being more likely to report snoring and choking/gasping during sleep compared with other ethnic groups,[19] which are sleep characteristics that were not measured in our study, but may account for greater sleep disturbance among this ethnic group.[37 38] Determinants and consequences of racial/ethnic differences in physiological parameters such as adiposity and insulin resistance warrant further investigation as they likely serve as potential explanations for differential relationships between poor sleep and type 2 diabetes risk.

When making within-race comparisons for white women, napping frequency was the only sleep characteristic that remained significantly associated with increased type 2 diabetes risk in fully adjusted models. Our results are consistent with other studies that have also reported non-significant sleep–diabetes associations among white women.[20 32] There were non-significant relationships and even lower risks of type 2 diabetes for short sleep for blacks and Hispanic/Latinas in within-race comparisons. These results could be due to the high prevalence of recommended and short sleep within both groups of racial/ethnic minority women, making statistically significant differences harder to detect.

Table 3 HRs (95% CI) for type 2 diabetes risk among 39 071 adult women in the Sister Study, with white, black, and Hispanic women with poor sleep compared with white women with recommended sleep

Sleep characteristics	Model I HR (95% CIs)	Model II HR (95% CIs)	Model III HR (95% CIs)	Model IV HR (95% CIs)	Model V HR (95% CIs)
Sleep duration*, Short sleep versus recommended (reference)					
White	**1.22 (1.09 to 1.37)**	**1.17 (1.04 to 1.32)**	**1.15 (1.02 to 1.30)**	1.07 (0.95 to 1.21)	**1.13 (1.00 to 1.28)**
Black	**2.90 (2.44 to 3.46)**	**2.59 (2.16 to 3.10)**	**2.26 (1.86 to 2.73)**	**1.36 (1.12 to 1.65)**	**1.80 (1.49 to 2.18)**
Hispanic	**2.67 (2.02 to 3.52)**	**2.26 (1.70 to 3.00)**	**2.21 (1.65 to 2.96)**	**2.06 (1.54 to 2.76)**	**2.14 (1.59 to 2.86)**
Time to fall asleep, >30 min versus ≤30 min (reference)					
White	**1.32 (1.16 to 1.51)**	**1.26 (1.10 to 1.44)**	**1.20 (1.05 to 1.38)**	1.13 (0.98 to 1.29)	**1.16 (1.01 to 1.33)**
Black	**2.97 (2.35 to 3.77)**	**2.64 (2.07 to 3.35)**	**2.39 (1.87 to 3.06)**	**1.46 (1.13 to 1.87)**	**1.88 (1.46 to 2.40)**
Hispanic	**2.39 (1.71 to 3.32)**	**2.02 (1.44 to 2.82)**	**1.86 (1.31 to 2.64)**	**1.93 (1.36 to 2.75)**	**1.91 (1.34 to 2.71)**
Frequent night awakenings, Yes † versus no (reference)					
White	**1.17 (1.01 to 1.36)**	1.13 (0.97 to 1.31)	1.12 (0.96 to 1.30)	1.06 (0.92 to 1.24)	1.08 (0.93 to 1.26)
Black	**2.96 (2.10 to 4.19)**	**2.67 (1.88 to 3.78)**	**2.26 (1.56 to 3.26)**	1.26 (0.87 to 1.82)	**1.77 (1.23 to 2.55)**
Hispanic	**4.48 (3.20 to 6.27)**	**3.72 (2.64 to 5.24)**	**3.62 (2.55 to 5.12)**	**3.16 (2.23 to 4.49)**	**3.61 (2.54 to 5.13)**
Napping frequency, ≥3 times/week versus <3 times/ week (reference)					
White	**1.61 (1.37 to 1.88)**	**1.57 (1.35 to 1.84)**	**1.42 (1.22 to 1.67)**	**1.22 (1.04 to 1.43)**	**1.34 (1.15 to 1.58)**
Black	**3.90 (2.91 to 5.22)**	**3.43 (2.55 to 4.61)**	**2.64 (1.92 to 3.63)**	**1.61 (1.17 to 2.21)**	**2.12 (1.55 to 2.92)**
Hispanic	**3.00 (1.97 to 4.57)**	**2.56 (1.68 to 3.91)**	**2.47 (1.60 to 3.82)**	**2.25 (1.46 to 3.48)**	**2.29 (1.48 to 3.55)**

Model I: adjusted for age (years) at baseline.
Model II: further adjusted for race/ethnicity, educational attainment (<HS, HS/GED, some college, and ≥bachelor's degree), marital status, and status of shift work/irregular hours (yes/no).
Model III: further adjusted for smoking status (current, past, and never), alcohol consumption (current, past, and never), diet (daily glycemic load), and physical activity (MET hours).
Model IV: further adjusted for hypertension, waist-to-hip ratio (WHR), and BMI (kg/m^2) measured at baseline.
Model V: without WHR and BMI.
HRs are in bold.
**Long sleep duration (>9 hours) is not reported (n=356).
HS, high school; GED, general education diploma; BMI, body mass index; MET, metabolic equivalent of task.hazard ratios are bolded.

There have been previous epidemiologic studies that have examined the role of sleep loss in the pathophysiology of diabetes and the role of obesity in this relationship.[16 25] For example, sleep deprivation decreases insulin sensitivity without appropriate compensatory beta-cell release, resulting in reduced glucose tolerance and increased risk of type 2 diabetes.[16] The findings in our study indicate that napping frequency for women in the overall sample, along with every poor sleep characteristic for blacks and Hispanics/Latinas compared with whites

Table 4 HRs (95% CI) for type 2 diabetes risk among 39 071 adult women in the Sister Study, with women with poor sleep compared with women with recommended sleep, stratified by race/ethnicity

	Model I	Interaction	Model II	Interaction	Model III	Interaction	Model IV	Interaction	Model V	Interaction
Sleep characteristics	**HR (95% CIs)**	**P value**	**HR (95% CIs)**	**P value**	**HR (95% CIs)**	**P value**	**HR (95% CIs)**	**P value**	**HR (95% CIs)**	**P value**
Sleep duration*										
Short sleep versus recommended (reference)		0.62		0.51		0.40		0.32		0.49
White	**1.22 (1.09 to 1.37)**		**1.17 (1.04 to 1.32)**		**1.15 (1.02 to 1.30)**		1.05 (0.93 to 1.19)		1.13 (1.00 to 1.27)	
Black	1.09 (0.86 to 1.39)		1.01 (0.79 to 1.30)		0.96 (0.74 to 1.24)		0.95 (0.73 to 1.22)		0.95 (0.74 to 1.23)	
Hispanic/Latina	1.09 (0.77 to 1.54)		1.05 (0.75 to 1.49)		1.10 (0.77 to 1.57)		1.02 (0.71 to 1.47)		1.06 (0.74 to 1.52)	
Time to fall asleep										
>30 min versus ≤30 min (reference)		0.19		0.18		0.24		0.57		0.45
White	**1.32 (1.15 to 1.51)**		**1.24 (1.09 to 1.42)**		**1.19 (1.04 to 1.37)**		1.11 (0.97 to 1.28)		1.15 (1.00 to 1.32)	
Black	1.09 (0.83 to 1.42)		1.04 (0.79 to 1.36)		1.06 (0.80 to 1.41)		1.00 (0.75 to 1.32)		1.04 (0.78 to 1.37)	
Hispanic/Latina	0.96 (0.66 to 1.41)		0.94 (0.64 to 1.38)		0.86 (0.58 to 1.29)		0.90 (0.60 to 1.35)		0.89 (0.60 to 1.34)	
Frequent night awakenings										
Yes† versus no (reference)		**0.01**		**0.02**		**0.01**		**0.02**		**0.01**
White	**1.16 (1.00 to 1.35)**		1.11 (0.96 to 1.29)		1.11 (0.95 to 1.29)		1.05 (0.90 to 1.22)		1.07 (0.92 to 1.24)	
Black	1.13 (0.78 to 1.63)		1.09 (0.76 to 1.58)		1.03 (0.70 to 1.51)		0.98 (0.66 to 1.44)		1.00 (0.68 to 1.48)	
Hispanic/Latina	**2.18 (1.49 to 3.21)**		**2.19 (1.48 to 3.23)**		**2.19 (1.46 to 3.29)**		**1.97 (1.30 to 2.99)**		**2.21 (1.47 to 3.33)**	
Napping frequency										
≥3 times/week versus <3 times/week (reference)		0.60		0.50		0.55		0.87		0.68
White	**1.60 (1.37 to 1.87)**		**1.56 (1.34 to 1.82)**		**1.42 (1.21 to 1.67)**		**1.20 (1.02 to 1.41)**		**1.33 (1.14 to 1.56)**	
Black	**1.52 (1.11 to 2.09)**		1.45 (1.05 to 2.00)		1.26 (0.89 to 1.78)		1.19 (0.84 to 1.69)		1.25 (0.89 to 1.76)	
Hispanic/Latina	1.26 (0.80 to 1.99)		1.23 (0.79 to 1.95)		1.26 (0.79 to 2.02)		1.13 (0.70 to 1.81)		1.18 (0.73 to 1.89)	

Continued

Table 4 Continued

Sleep characteristics	Model I HR (95% CIs)	Interaction P value	Model II HR (95% CIs)	Interaction P value	Model III HR (95% CIs)	Interaction P value	Model IV HR (95% CIs)	Interaction P value	Model V HR (95% CIs)	Interaction P value
Cumulative sleep score ‡		0.11		0.07		0.06		**0.05**		0.05
White										
1 versus 0 (reference)	**1.25 (1.11 to 1.41)**		**1.22 (1.08 to 1.38)**		**1.18 (1.04 to 1.33)**		1.11 (0.99 to 1.25)		**1.17 (1.03 to 1.32)**	
2–4 versus 0 (reference)	**1.55 (1.34 to 1.78)**		**1.43 (1.23 to 1.65)**		**1.35 (1.16 to 1.56)**		1.15 (0.99 to 1.33)		**1.25 (1.08 to 1.45)**	
Black										
1 versus 0 (reference)	1.00 (0.74 to 1.34)		0.91 (0.68 to 1.22)		0.83 (0.61 to 1.13)		0.80 (0.59 to 1.09)		0.82 (0.60 to 1.12)	
2–4 versus 0 (reference)	1.33 (0.98 to 1.79)		1.20 (0.88 to 1.62)		1.09 (0.80 to 1.50)		1.02 (0.74 to 1.41)		1.07 (0.78 to 1.47)	
Hispanic/Latina										
1 versus 0 (reference)	0.74 (0.49 to 1.11)		0.73 (0.48 to 1.10)		0.74 (0.48 to 1.13)		0.69 (0.44 to 1.07)		0.70 (0.46 to 1.08)	
2–4 versus 0 (reference)	1.30 (0.87 to 1.95)		1.26 (0.84 to 1.88)		1.21 (0.79 to 1.85)		1.10 (0.72 to 1.70)		1.19 (0.78 to 1.82)	

Model I: adjusted for age (years) at baseline.
Model II: further adjusted for race/ethnicity, educational attainment (<HS, HS/GED, some college, and ≥bachelor's degree), marital status, and status of shift work/irregular hours (yes/no).
Model III: further adjusted for smoking status (current, past, and never), alcohol consumption (current, past, and never), diet (daily glycemic load), and physical activity (MET hours).
Model IV: further adjusted for hypertension, waist-to-hip ratio (WHR), and BMI (kg/m^2) measured at baseline.
Model V: without WHR and BMI.
HRs are in bold.
*Long sleep duration (>9 hours) is not reported (n=356).
†Frequent night awakenings was classified as 'No' if participant reported waking up while sleeping less than three times per week and 'Yes' if participant reported waking up while sleeping more than three times per night/day for more than three nights/days per week.
‡Cumulative sleep score was calculated by summing each participant's 'yes' response for each sleep characteristic. Scores range from 0 to 4, with poor sleep characteristics being combined into the category '2 to 4'.
HS, high school; GED, general education diploma; BMI, body mass index; MET, metabolic equivalent of task.hazard ratios are bolded.

may directly affect risk of type 2 diabetes, independent of body weight. Beihl *et al*[25] found that adjustment for BMI did not attenuate the odds of type 2 diabetes risk for Hispanics and blacks. In contrast to our study that examined the impact of both poor sleep and race/ethnicity on type 2 diabetes risk, the findings in this previous study resulted from stratified models.

This study has limitations. For instance, all sleep data were self-reported. A recent study found that all racial/ethnic groups overestimated their sleep duration in a non-differential manner when comparing self-report to both polysomnographic-measured and wrist actigraphy-measured sleep.[39] We recognize this as a limitation, and the results should be interpreted with caution. Type 2 diabetes was determined by self-report of a physician diagnosis, but validated positive predictive values were found for self-reported prevalent diabetes (91.8%) and incident diabetes (82.2%). A high negative predictive value (94.5%) for diabetes was also reported.[40] Furthermore, we only used one timepoint at baseline for characterizing sleep in assessing type 2 diabetes risk. Using a single measure to determine exposure may not fully capture long-term sleep disruption patterns. This approach, however, is consistent with most prospective epidemiologic studies examining the sleep–diabetes relationship using self-reported data. Participants were also asked if they have ever been diagnosed with diabetes without respect to type. Despite various approaches excluding type 1 diabetes, there still might be misclassification error due to its derived status. We did not include family history of type 2 diabetes in our analysis. We did not include Asians as a racial/ethnic minority in our analysis due to the sample size being too small to provide robust estimates explicitly among this population. Generalizability of these results may be reduced. Furthermore, sleep apnea could explain, at least in part, why patients with sleep disturbances or frequent napping are more prone to develop incident diabetes. Obstructive sleep apnea syndrome diagnosis or continuous positive airway pressure (CPAP) use data were not available in this study. Lastly, we acknowledge that high napping frequency could serve as compensation for sleep deficiency (eg, short sleep and longer latency) or poor sleep quality due to sleep disturbance (eg, frequent night awakenings). When modeling the napping frequency–diabetes relationship, we did not adjust for sleep duration, latency or night awakenings, which increased the likelihood that these sleep dimensions may confound this relationship. Despite some weaknesses, there are several strengths to our study, which include its prospective design with a high follow-up response rate of >92%, our analysis of multiple sleep dimensions, a fair representation of racial/ethnic minorities and standardized data collection methods. To our knowledge, this is the first study to examine race-specific associations and if race/ethnicity modified the relationship between multiple sleep dimensions and type 2 diabetes risk in a large sample of women across the USA.

CONCLUSIONS

This paper extends the findings from previous studies regarding the sleep–diabetes relationship by examining if race/ethnicity modifies the relationship between poor sleep and type 2 diabetes risk. Racial/ethnic minority women who experienced poor sleep had an approximately threefold increased risk for type 2 diabetes compared with white women with recommended sleep after adjustments, including adjustment for adiposity measures. More population-based research is needed to investigate potential social and environmental pathways as well as individual-level factors that can help identify reasons for racial/ethnic differences in the relationship between various sleep dimensions (eg, napping frequency and night awakenings) and diabetes risk. Intervention strategies for improving sleep health (especially particular dimensions) may reduce type 2 diabetes risk in the general population and racial/ethnic disparities in metabolic conditions like type 2 diabetes.

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REFERENCES

1. Rowley WR, Bezold C, Arikan Y, *et al*. Diabetes 2030: insights from yesterday, today, and future trends. *Popul Health Manag* 2017;20:6–12.
2. Trikkalinou A, Papazafiropoulou AK, Melidonis A. Type 2 diabetes and quality of life. *World J Diabetes* 2017;8:120–9.
3. Narayan KMV, Boyle JP, Geiss LS, *et al*. Impact of recent increase in incidence on future diabetes burden: U.S., 2005-2050. *Diabetes Care* 2006;29:2114–6.
4. Cappuccio FP, D'Elia L, Strazzullo P, *et al*. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. *Diabetes Care* 2010;33:414–20.
5. Gangwisch JE, Heymsfield SB, Boden-Albala B, *et al*. Sleep duration as a risk factor for diabetes incidence in a large US sample. *Sleep* 2007;30:1667–73.

6. Shan Z, Ma H, Xie M, *et al*. Sleep duration and risk of type 2 diabetes: a meta-analysis of prospective studies. *Diabetes Care* 2015;38:529–37.
7. Rudnicka AR, Nightingale CM, Donin AS, *et al*. Sleep duration and risk of type 2 diabetes. *Pediatrics* 2017;140:e20170338.
8. Knutson KL, Spiegel K, Penev P, *et al*. The metabolic consequences of sleep deprivation. *Sleep Med Rev* 2007;11:163–78.
9. Buxton OM, Marcelli E. Short and long sleep are positively associated with obesity, diabetes, hypertension, and cardiovascular disease among adults in the United States. *Soc Sci Med* 2010;71:1027–36.
10. Jackson CL, Redline S, Kawachi I, *et al*. Association between sleep duration and diabetes in black and white adults. *Diabetes Care* 2013;36:3557–65.
11. Jackson CL. Determinants of racial/ethnic disparities in disordered sleep and obesity. *Sleep Health* 2017;3:401–15.
12. Shadyab AH, Kritz-Silverstein D, Laughlin GA, *et al*. Ethnic-Specific associations of sleep duration and daytime napping with prevalent type 2 diabetes in postmenopausal women. *Sleep Med* 2015;16:243–9.
13. Liu Y, Wheaton AG, Chapman DP, *et al*. Prevalence of healthy sleep duration among adults — United States, 2014. *MMWR Morb Mortal Wkly Rep* 2016;65:137–41.
14. Stamatakis KA, Kaplan GA, Roberts RE. Short sleep duration across income, education, and Race/Ethnic groups: population prevalence and growing disparities during 34 years of follow-up. *Ann Epidemiol* 2007;17:948–55.
15. Morselli LL, Guyon A, Spiegel K. Sleep and metabolic function. *Pflugers Arch - Eur J Physiol* 2012;463:139–60.
16. Morselli L, Leproult R, Balbo M, *et al*. Role of sleep duration in the regulation of glucose metabolism and appetite. *Best Pract Res Clin Endocrinol Metab* 2010;24:687–702.
17. Buxton OM, Pavlova M, Reid EW, *et al*. Sleep restriction for 1 week reduces insulin sensitivity in healthy men. *Diabetes* 2010;59:2126–33.
18. St-Onge M-P, McReynolds A, Trivedi ZB, *et al*. Sleep restriction leads to increased activation of brain regions sensitive to food stimuli. *Am J Clin Nutr* 2012;95:818–24.
19. Grandner MA, Petrov MER, Rattanaumpawan P, *et al*. Race/Ethnicity, and socioeconomic position. *Journal of Clinical Sleep Medicine : JCSM : Official Publication of the American Academy of Sleep Medicine* 2013;9:897–905.
20. Whinnery J, Jackson N, Rattanaumpawan P, *et al*. Short and long sleep duration associated with Race/Ethnicity, Sociodemographics, and socioeconomic position. *Sleep* 2014;37:601–11.
21. Hall MH, Matthews KA, Kravitz HM, *et al*. Race and financial strain are independent correlates of sleep in midlife women: the Swan sleep study. *Sleep* 2009;32:73–82.
22. Mezick EJ, Matthews KA, Hall M, *et al*. Influence of race and socioeconomic status on sleep: Pittsburgh SleepSCORE project. *Psychosom Med* 2008;70:410–6.
23. Grandner MA, Williams NJ, Knutson KL, *et al*. race/ethnicity, and socioeconomic position. *Sleep medicine* 2016;18:7–18.
24. Zizi F, Pandey A, Murrray-Bachmann R, *et al*. Race/Ethnicity, sleep duration, and diabetes mellitus: analysis of the National health interview survey. *Am J Med* 2012;125:162–7.
25. Beihl DA, Liese AD, Haffner SM. Sleep Duration as a Risk Factor for Incident Type 2 Diabetes in a Multiethnic Cohort. *Ann Epidemiol* 2009;19:351–7.
26. Broussard JL, Chapotot F, Abraham V, *et al*. Sleep restriction increases free fatty acids in healthy men. *Diabetologia* 2015;58:791–8.
27. Nilsson PM, Rööst M, Engström G, *et al*. Incidence of diabetes in middle-aged men is related to sleep disturbances. *Diabetes Care* 2004;27:2464–9.
28. Sandler DP, Hodgson ME, Deming-Halverson SL, *et al*. The sister study cohort: baseline methods and participant characteristics. *Environ Health Perspect* 2017;125:127003.
29. Hirshkowitz M, Whiton K, Albert SM, *et al*. National Sleep Foundation's updated sleep duration recommendations: final report. *Sleep Health* 2015;1:233–43.
30. Register F. The daily Journal of the United States government. standards for maintaining, collecting, and presenting federal data on race and ethnicity. A notice by the management and budget office on 09/30/2016.. Available: https://www.federalregister.gov/documents/2016/09/30/2016-23672/standards-for-maintaining-collecting-and-presenting-federal-data-on-race-and-ethnicity
31. Block G, Hartman AM, Dresser CM, *et al*. A data-based approach to diet questionnaire design and testing. *Am J Epidemiol* 1986;124:453–69.
32. Whelton PK, Carey RM, Aronow WS, *et al*. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: Executive summary: a report of the American College of Cardiology/American heart association Task force on clinical practice guidelines. *Hypertension* 2018;71:1269–324.
33. Arora T, Chen MZ, Omar OM, *et al*. An investigation of the associations among sleep duration and quality, body mass index and insulin resistance in newly diagnosed type 2 diabetes mellitus patients. *Ther Adv Endocrinol Metab* 2016;7:3–11.
34. Chen GC, Liu MM, Chen LH, *et al*. Daytime napping and risk of type 2 diabetes: a meta-analysis of prospective studies. *Sleep Breath* 2017.
35. Ayas NT, White DP, Al-Delaimy WK, *et al*. A prospective study of self-reported sleep duration and incident diabetes in women. *Diabetes Care* 2003;26:380–4.
36. Björkelund C, Bondyr-Carlsson D, Lapidus L, *et al*. Sleep disturbances in midlife unrelated to 32-year diabetes incidence. *The prospective Population Study of Women in Gothenburg* 2005;28:2739–44.
37. Kaufmann CN, Mojtabai R, Hock RS, *et al*. Racial/Ethnic differences in insomnia trajectories among U.S. older adults. *The American Journal of Geriatric Psychiatry* 2016;24:575–84.
38. Petrov ME, Lichstein KL. Differences in sleep between black and white adults: an update and future directions. *Sleep Med* 2016;18:74–81.
39. Jackson CL, Patel SR, Jackson WB, *et al*. Agreement between self-reported and objectively measured sleep duration among white, black, Hispanic, and Chinese adults in the United States: multi-ethnic study of atherosclerosis. *Sleep* 2018;41.
40. Jackson JM, DeFor TA, Crain AL, *et al*. Validity of diabetes self-reports in the Women's Health Initiative. *Menopause* 2014;21:861–8.