Title

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Journal

American Journal of Obstetrics and Gynecology, 217(4)

ISSN

0002-9378

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Publication Date

2017-10-01

DOI

10.1016/j.ajog.2017.05.066

Peer reviewed

Published in final edited form as:

Am J Obstet Gynecol. 2017 October ; 217(4): 447.e1–447.e13. doi:10.1016/j.ajog.2017.05.066.

Objectively measured short sleep duration and later sleep midpoint in pregnancy are associated with a higher risk of gestational diabetes

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The other authors did not report any potential conflicts of interest.

Comments and views of the author(s) do not necessarily represent the views of the NICHD.

Findings were presented at the Society for Maternal Fetal Medicine 2015 annual meeting, San Diego, February 02–07, 2015.

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Abstract

Background—Experimental and epidemiologic data suggest that among non-pregnant adults, sleep duration may be an important risk factor for chronic disease. Although pregnant women commonly complain of poor sleep, few studies have objectively evaluated the quality of sleep in pregnancy or have explored the relationship between sleep disturbances and maternal and perinatal outcomes.

Objectives—Our objective was to examine the relationship between objectively assessed sleep duration, timing and continuity (measured via wrist actigraphy) and maternal cardiovascular and metabolic morbidity specific to pregnancy.

Study Design—This was a prospective cohort study of nulliparous women. Women were recruited between 16 0/7 and 21 6/7 weeks' gestation. They were asked to wear a wrist actigraphy monitor and to complete a daily sleep log for a seven consecutive-day period. The primary sleep exposure variables were the averages of the following over the total valid nights (minimum 5, maximum 7 nights): short sleep duration during the primary sleep period (< 7 hours/night), late sleep midpoint (midpoint between sleep onset and sleep offset > 5 AM), and top quartile of minutes of wake time after sleep onset (WASO) and sleep fragmentation index. The primary outcomes of interest were a composite of hypertensive disorders of pregnancy (mild, severe, or superimposed preeclampsia; eclampsia; or antepartum gestational hypertension) and gestational diabetes (GDM). Chi-square tests were used to assess associations between sleep variables and categorical baseline characteristics. Crude odds ratios and 95% confidence intervals were estimated from univariate logistic regression models to characterize the magnitude of the relationship between sleep characteristics and hypertensive disorders of pregnancy and GDM. For associations that were significant in univariate analysis, multiple logistic regression was used to explore further the association of sleep characteristics with pregnancy outcomes.

Results—Nine-hundred and one eligible women consented to participate. Of these women 782 submitted valid actigraphy studies. Short sleep duration and a later sleep midpoint were associated with an increased risk of GDM (OR 2.24, 95% CI 1.11, 4.53; OR 2.58, 95% CI 1.24, 5.36, respectively) but not of hypertensive disorders. A model with both sleep duration and sleep midpoint as well as their interaction term revealed that while there was no significant interaction between these exposures, the main effects of both short sleep duration and later sleep midpoint

with GDM remained significant (aOR 2.06, 95% CI 1.01, 4.19; aOR 2.37, 95% CI 1.13, 4.97, respectively). Additionally, after adjusting separately for age, BMI and race/ethnicity, both short sleep duration and later sleep midpoint remained associated with GDM. No associations were demonstrated between the sleep quality measures (WASO, sleep fragmentation) and hypertensive disorders or GDM.

Conclusions—Our results demonstrate a relationship between short sleep duration and later sleep midpoint with GDM. Our data suggest independent contributions of these two sleep characteristics to the risk for GDM in nulliparous women.

Condensation

Both objectively measured short sleep duration and later sleep timing are associated with development of gestational diabetes.

Keywords

Gestational diabetes; hypertension; pregnancy outcomes; sleep duration; sleep midpoint; sleep quality; actigraphy

Introduction

Experimental and epidemiologic data suggest that among non-pregnant adults, sleep duration is an important risk factor for chronic disease.[1–3] For example, short sleep duration has been linked to a higher frequency of hypertension and cardiovascular disease.[4–7] There are particularly strong data suggesting that short sleep duration is associated with disordered metabolism and is linked to an increase in the risk of type 2 diabetes.[8–10] Long sleep duration has also been linked to cardiovascular and metabolic disease. [5, 8] While considerable research has focused on sleep duration, other aspects of sleep, including the timing of sleep and wake cycles and continuity of sleep, have been proposed as potential cardiometabolic risk factors.[11–14]

Hypertensive disease (e.g., preeclampsia) and metabolic disease (e.g., gestational diabetes mellitus) also can be acute complications during pregnancy. Hypertensive disorders of pregnancy and gestational diabetes are associated with maternal and perinatal morbidity and have long-term health consequences for both mothers and babies.[15, 16] Nevertheless, although pregnant women commonly complain of poor sleep,[17] few studies have objectively evaluated the duration, timing and quality of sleep in pregnancy and explored the relationship between objectively measured sleep and maternal and perinatal outcomes.[18–25] Such a relationship is clinically relevant, as its existence may elucidate a modifiable factor for adverse pregnancy outcomes.

Therefore, our objective was to examine the relationship between objectively assessed sleep duration, timing and continuity (measured via actigraphy) and maternal cardiovascular and metabolic morbidity specific to pregnancy.

Methods

This Sleep Duration and Continuity Study was conducted as a sub-study of the Nulliparous Pregnancy Outcome Study: Monitoring Mothers-to-be (nuMoM2b). NuMoM2b was an observational cohort study, conducted at 8 clinical sites and managed by a central data coordinating and analysis center.[26] The parent study protocol included nulliparous women of at least 13 years of age, although for this sub-study, those under the age of 18 were excluded given significant differences in adolescent versus adult sleep. In addition, while the parent study included women with chronic hypertension, these women were excluded from this sub-study given the difficulty in accurately diagnosing a new-onset hypertensive disorder in women with pre-existing hypertension. Women with pre-existing diabetes were also excluded as they cannot be diagnosed with gestational diabetes.

Women were recruited for this sleep sub-study at the parent study's second study visit, which was scheduled between 16 0/7 and 21 6/7 weeks' gestation. They were asked to wear a wrist actigraphy monitor (Spectrum, Philips Respironics, Figure 1) that records rest and activity periods and to complete a daily sleep log for the same seven consecutive-day period. Subjects were given up to 23 0/7 weeks to complete the 7 days of actigraphy and sleep log data collection. For the sleep log, participants were asked to note bedtime, wake-up time, total sleep time, sleep latency, wake after sleep onset, naps, any unusual events during the sleep period, and overall sleep quality. Actigraphy provides an objective measure of rest-activity patterns from which various measures of sleep duration, quality and timing can be determined daily. Studies have shown that there is good correlation between wrist actigraphy and polysomnographic (PSG) recorded sleep.[27] In addition, actigraphy provides the ability to collect sleep-wake information over many consecutive days, unlike PSG, in a simple, reliable and cost-effective manner. Obtaining an objective evaluation of sleep is important because data suggests that there is only a moderate correlation between self-reported and actigraphically recorded sleep, particularly among those with more disrupted sleep patterns. [28, 29]

An actigraphy recording was considered successful if there was a minimum of 5 days recorded and if within the five days there was less than 4 hours of off-wrist time per 24-hour period and no off-wrist signal during the time the subject indicated she was in bed. If a participant's recording did not meet these criteria, she was asked if she would be willing to wear the watch for another 7-day period if the total recording could be completed by 23 0/7 weeks.

All actigraph files and sleep log data were securely transmitted to a central actigraphy reading center. The complete sleep scoring and quality control protocol has been described previously.[30] The primary sleep exposure variables for this analysis were the averages of the following over the total valid nights (minimum 5, maximum 7 nights): sleep duration during the primary sleep period, sleep midpoint (the midpoint between sleep onset and sleep offset), minutes of wake time after sleep onset (WASO) and sleep fragmentation index. These variables are defined in greater detail in Table 1. Based on a review of available data detailing the relationship between sleep in non-pregnant populations and health outcomes, a cut-off of less than 7 hours for sleep duration was defined *a priori* as "short sleep duration"

[31], Similarly, a cutoff of later than 5 AM was defined *a priori* as a "late sleep midpoint". [32, 33] As there are no well-established, clinically-relevant cut-offs for actigraphy defined WASO and sleep fragmentation, these continuous variables were transformed into quartiles; women were categorized based on these quartiles, with women in the quartiles representing the longest WASO and the greatest sleep fragmentation considered to have the most disturbed, poorest quality, sleep.

At least 30 days after delivery, a trained, certified chart abstractor assessed all participants' medical records to record birth outcomes and readmissions to the hospital. The primary outcomes of interest were 1) a composite of hypertensive disorders of pregnancy (mild or severe preeclampsia; eclampsia; or antepartum gestational hypertension) and 2) gestational diabetes (GDM). For any participant with documented hypertension or proteinuria a detailed chart abstraction was performed that included assessment of blood pressure severity, new-onset neurologic disturbances, epigastric pain or pulmonary edema, and blood and urine laboratory results. Supplement S1 (online) outlines study definitions for types of hypertensive disorders. Cases that presented atypically and were difficult to classify according to study criteria were adjudicated by review of clinical data by the principal investigators and final classification was reached by their consensus judgment.

All glucose tolerance testing (GTT) was performed as part of routine clinical care. A woman was considered to have GDM if she met one of the following GTT criteria: 1) fasting 3-hour 100 gram GTT with two of the following values: fasting ≥95 mg/dL, 1-hour ≥180 mg/dL, 2-hour ≥155 mg/dL, 3-hour ≥140 mg/dL; 2) fasting 2-hour 75 gram GTT with one of the following values: fasting ≥92 mg/dL, 1-hour ≥180 mg/dL, 2-hour ≥153 mg/dL; 3) non-fasting 50 gram GTT with a one-hour value ≥ 200 mg/dL if no fasting 3-hour or 2-hour GTT was performed. In addition to GTT data, chart abstractors recorded if a diagnosis of GDM was made during the course of clinical care. If no GTT data were available the diagnosis of GDM based on chart abstraction was used for GDM classification.

A detailed description of our sample size calculation has been previously published.[30] In summary, assuming, as described in non-pregnant US populations, a "short sleep duration" prevalence of 25%, an outcome prevalence of at least 8% for pregnancy related hypertension or GDM, and an alpha error of < 5%, a sample size of 760 women would provide at least 80% power to detect a 2-fold increase in the risk of the adverse pregnancy outcomes among women with short sleep durations.[34]

Descriptive statistics were used to characterize the study population by dichotomous sleep variables (sleep duration <7 hours, WASO ≥ 75th percentile, sleep fragmentation index ≥ 75th percentile, and sleep midpoint later than 5 AM). Chi-square tests were used to assess the associations between sleep variables and categorical baseline characteristics. Kruskal-Wallis tests were used to compare the distribution of sleep variables for categories of baseline characteristics. Spearman correlation coefficients were used to assess relationships between sleep variables. Crude odds ratios and 95% confidence intervals were estimated from univariate logistic regression models to characterize the magnitude of the relationship between sleep characteristics and hypertensive disorders of pregnancy and GDM. For associations that were significant in univariate analysis, multiple logistic regression was used

to explore further the association of sleep characteristics with pregnancy outcomes after adjustment for baseline characteristics. Adjustment covariates that were chosen a priori included maternal age and early pregnancy body mass index (BMI), treated as continuous variables and maternal race/ethnicity. Self-reported frequent snoring (a common symptom of sleep apnea), defined as pre-pregnancy snoring at least 3–4 times/week, and employment schedule were also chosen as covariates given their association with our sleep variables and pregnancy outcomes. Linear and quadratic terms were included for age and BMI to help ensure that their relationships with the outcome were fully accounted for in the adjustment. Multiple logistic regression was also used post hoc to consider interactions between two sleep characteristics on pregnancy outcomes, and to assess their independent association with GDM. Initial models included main effects and interaction, but were reduced to main effects in the absence of a significant interaction.

All tests were performed at a nominal significance level of α=0.05 with two-sided, single degree-of-freedom tests. No correction was made for multiple comparisons. Analyses were conducted using SAS 9.3/9.4 software. This study was approved the Institutional Review Board at each center, and all women provided informed written consent prior to enrollment.

Results

Nine-hundred and one eligible women consented to participate. Of these women, 782 submitted valid actigraphy studies and form the basis of this analysis. The median gestational age at recruitment (study visit 2) was 19 1/7 weeks' gestation (range 15 6/7 – 22 5/7 weeks of gestation). The median gestational age at delivery was 39 5/7 weeks' gestation (range 21 0/7 – 42 4/7 weeks of gestation). The rate of hypertensive disorders was 11.6%. Specifically, the rate of preeclampsia was 4.9% (38/782); the rate of antepartum gestational hypertension was 6.8% (53/782). The rate of gestational diabetes was 4.2%. The large majority of women (n= 699, 94.3%) completed their GTT more than 1 week after the actigraphy study. Forty-two women (5.7%) completed their GTT testing before the objective evaluation of sleep duration (only 16/42 were performed before 13 weeks); only 4 of these 42 women were diagnosed with GDM.

Sleep duration of < 7 hours was present in 27.9% of the participants. Only 3.5% and 2.6% of women had sleep durations of < 6 hours or ≥ 9 hours, respectively. 18.9% of women had a sleep midpoint later than 5 AM. Participant characteristics stratified by different sleep metrics are shown in Table 2. Short sleep duration was significantly associated with race/ethnicity and BMI. The upper quartiles of WASO and sleep fragmentation were associated with most baseline demographics, with the exception of self-reported frequent snoring and prior sleep disorder history (sleep apnea, insomnia, restless legs syndrome). Late sleep timing was associated with all characteristics with the exception of prior history of restless legs syndrome. Of note, we found that women who worked regular day shifts had significantly earlier sleep midpoints [N= 441, median (Q1: 25th percentile, Q3: 75th percentile) sleep midpoint 3:10 AM (2:40 AM, 3:51 AM)] compared to women who reported working some form of shift work [N=148 median (Q1, Q3) sleep midpoint 4:14 AM (3:22 AM, 5:39 AM)] or who were unemployed [N=152 median (Q1, Q3) sleep midpoint 4:34 AM (3:45 AM, 5:37AM)], Kruskal-Wallis test p<0.0001.

The relationship between sleep metrics and hypertensive disorders of pregnancy and GDM are shown in Table 3. Short sleep duration and a later sleep midpoint were associated with an increased risk of GDM (OR 2.24, 95% CI 1.11, 4.53; OR 2.58, 95% CI 1.24, 5.36, respectively) but not hypertensive disorders. When we repeated the sleep duration and midpoint analyses excluding women whose GDM status was ascertained only through diagnosis from chart abstraction but without a documented GTT (N =44), the odds ratios and p-values did not change appreciably (data not shown). No associations were demonstrated between the sleep quality measures (WASO, sleep fragmentation) and hypertensive disorders or GDM.

Given the significant findings between both sleep duration and sleep midpoint in relation to GDM, we examined the relationship of sleep duration with sleep midpoint, and found that they were not correlated in our cohort (Spearman correlation coefficient 0.02, P=0.67). A logistic regression model with both sleep duration and sleep midpoint as well as their interaction term revealed that while there was no significant interaction between these exposures, the main effects of both short sleep duration and later sleep midpoint with GDM were significant (aOR 2.06, 95% CI 1.01, 4.19; aOR 2.37, 95% CI 1.13, 4.97, respectively).

With 33 cases of GDM, multiple covariate adjustment would risk model overfitting. Therefore, to further examine the relationship of short sleep duration and late sleep midpoint with gestational diabetes, taking into account potential confounders, we performed a series of regression analyses with single variable adjustments. After adjusting separately for age, BMI, race/ethnicity and employment schedule, both short sleep duration and sleep midpoint remained associated with GDM (Table 4). After adjusting for self-reported frequent snoring, sleep midpoint remained significantly associated with GDM; the magnitude of the effect of short sleep duration with GDM was similar to that seen in the other unadjusted and adjusted analyses, although the p-value just exceeded 0.05 (aOR 2.29, 95% CI 0.97, 5.39; p-value = 0.059).

Conclusions

Our data demonstrate that, among nulliparous women, both sleep duration and timing of sleep in the second trimester are associated with the development of GDM. Specifically, mean sleep duration of less than 7 hours per night was associated with an approximate 2-fold increase in the odds of GDM, and this association was independent of age, BMI, race/ethnicity and self-reported frequent snoring. Similarly, a later sleep midpoint (after 5 AM) was associated with an increased risk of GDM, and our data suggest that this increase was independent of sleep duration. We did not detect any relationship between measures of sleep duration, timing or sleep quality and hypertensive disorders of pregnancy.

While laboratory studies demonstrate that experimentally fragmented sleep alters glucose metabolism, adrenocortical function and sympathovagal balance,[11] in our study, sleep quality, as measured by WASO and sleep fragmentation, was not associated with maternal increased cardiovascular and metabolic morbidity. However, our negative results should be considered with caution. The degree of WASO disturbance and sleep fragmentation were categorized by quartiles as there are no other well established, clinically relevant cut-offs for

these sleep characteristics in pregnancy, and it is possible our sample size did not allow for the examination of more severe, but less common, phenotypes.

The major strengths of this study are the use of actigraphy to obtain a weeklong objective recording of rest/activity in coordination with a central and blinded actigraphy reading center. The study population is diverse in terms of age, race, ethnicity, BMI and socio-economic measures, making our findings more generalizable. Nevertheless, given our onetime assessment of objectively measures sleep, we were not able to examine whether changes in sleep patterns and quality throughout pregnancy also may be associated with pregnancy outcomes. And while some studies suggest that long sleep duration (at least 9 hours) is also a risk factor for cardiovascular and metabolic disease, we were not able to examine the association between long sleep duration and hypertensive disorders and GDM due to the very small proportion of women who had sleep durations in this range (2.6%). Finally, given the observational design, there is always the possibility of residual confounding from unmeasured variables or a limit in the extent to which even measured confounders can be assessed.

In non-pregnant populations, several large, prospective cohort studies examined the association between sleep duration and incident type 2 diabetes, although the majority relied on self-reported sleep assessments. Most of these studies reported increased odds of diabetes associated with short sleep duration. In two independent pooled analyses of prospective studies, an association between short sleep and incident type 2 diabetes was documented, with odds ratio 1.33 (95% CI 1.20, 1.48) and relative risk 1.28 (95% CI 1.03, 1.60) reported. [8, 9] Data from pregnancy cohorts, both prospective and retrospective, using self-reported sleep duration have also suggested that short sleep is a risk factor for GDM.[35–37] In one study of 63 pregnant women who wore an actigraph in mid-pregnancy, objectively measured short sleep was associated with higher values on 1-hour GTT screening tests.[21] Our data are consistent with and extend the findings of these previous reports, confirming that short sleep duration, as objectively measured by actigraphy, is associated with incident GDM in a large population of nulliparous women. Our study is the largest to date in which objective measures of sleep duration in pregnancy were obtained. Furthermore, by excluding women with pre-gestational diabetes, rigorously defining GDM, and by measuring sleep duration prior to 24 weeks, we optimized case ascertainment and ensured that our exposure variable pre-dated the diagnosis in the vast majority of our subjects.

Compared to diabetes, the association between sleep duration and chronic hypertension is less consistent in non-pregnant individuals. Cross-sectional studies generally demonstrate a higher rate of hypertension among short sleepers, but data from longitudinal cohorts are conflicting.[38]. Limited data on the association of sleep duration measures with hypertension in pregnancy are similarly conflicting. [20, 39] In our study of nulliparous women that excluded women with chronic hypertension, we failed to demonstrate a relationship between sleep duration and pregnancy-related hypertension. We were powered to detect an approximately 2-fold increase in risk, but cannot rule out the possibility of a more modest risk relationship.

In addition to sleep duration and continuity, the timing of sleep as a marker of circadian timing is emerging as a risk factor for cardiometabolic health.[32] Later sleep timing is often associated with circadian misalignment. This misalignment can occur when sleep and wakefulness behaviors do not occur at an appropriate time relative to the timing of the central circadian clock (hypothalamus) and/or relative to the external environment (light-dark cycle). A large survey study demonstrated that, when free from occupation and social obligations, the most commonly reported sleep timing is approximately midnight to 8:00 AM.[32] However, work and social demands as well as environmental influences, can lead to alterations in sleep timing and circadian disruption. Experimental studies show that sleep misaligned with the timing of endogenous circadian rhythms is linked to alterations in leptin and glucose and increased mean arterial pressure. [40] In addition, being awake later at night has been linked to poorer health behaviors such as greater fast-food consumption and increased alcohol use.[12, 41] Epidemiologic data link shift-work to an increased risk of obesity, type 2 diabetes and cardiovascular disease.[42–44] Our study examined sleep timing in pregnancy (i.e., sleep midpoint) as a risk factor for adverse pregnancy outcomes. We found that both having a shift-work schedule and being unemployed while pregnant were associated with a later sleep midpoint (> 5 AM) and we demonstrated a strong association between later sleep timing and GDM that was independent of sleep duration and other confounders.

In conclusion, we found a relationship between short sleep duration and later sleep timing with GDM in nulliparous women. Our data suggest independent contributions of these two sleep characteristics to the risk for GDM. Identifying and addressing modifiable risks factors for GDM is important as GDM is associated with an increased risk of preeclampsia, fetal macrosomia, birth trauma, and neonatal metabolic complications.[45] Based on our data, sleep, like diet and exercise, should be considered a modifiable behavior that has potential health implications for pregnancy. Although this is an observational study, it is biologically plausible that the relationship may be causal. Yet, the mechanisms by which sleep duration and timing may impact metabolism in pregnancy are likely multifactorial and further research is needed to better understand the biology of sleep in pregnancy and whether interventions in early pregnancy to address sleep duration and schedule can modify the risk of GDM.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Supported by grant funding from the National Heart Lung and Blood Institute R01HL105549 and *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD): U10 HD063036, Research Triangle Institute; U10 HD063072, Case Western Reserve University; U10 HD063047, Columbia University; U10 HD063037, Indiana University; U10 HD063041, Magee-Women's Hospital; U10 HD063020, Northwestern University; U10 HD063046, University of California Irvine; U10 HD063048, University of Pennsylvania; and U10 HD063053, University of Utah.

Dr. Zee reports receiving grant support from Jazz and Technogel and is a consultant for Merck, Eisai, Philips and Teva none of these activities are related to this manuscript.

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Figure 1.
Actiwatch Spectrum (Philips Respironics)

Table 1

Sleep variable definitions

Variable name	Variable description
Sleep duration (hours)	• Sleep duration was defined as the total amount of time scored as sleep during the main designated rest period each day. • The sleep duration for each available day (5–7 days) were then averaged. • The cut off of an average sleep duration of < 7 hours was chosen based on prior data in pregnant women from the authors and on recommendations for adequate sleep duration recently published by the American Academy of Sleep Medicine.
Sleep midpoint (hh:mm)	• Sleep midpoint is the clock time that represents the midpoint between the clock time of sleep onset and the clock time of sleep offset. • The sleep midpoint was calculated for each valid day then averaged for each available day (5–7 days). • The cut off of an average sleep midpoint later than 5am was chosen a *priori* based on large samples of population based self-reported data.
Wake after sleep onset (minutes)	• Wake after sleep onset (WASO) is the total amount of minutes spent awake between sleep onset and the end of the rest interval. • The minutes of WASO were calculated for each valid day then averaged for each available day (5–7 days). • Given that there are no well-established actigraphy based cutoffs for WASO, the data were divided into quartiles and those with an average WASO ≥ 75th percentile were considered to have poor sleep.
Sleep fragmentation (%)	• The sleep fragmentation index (FI) is calculated as the proportion of all epochs from sleep onset to sleep offset with an activity count of 2 or greater plus the proportion of all bouts of immobility (activity count less than 2 in every epoch) that were 1 minute or less in duration. • The FI was determined for each valid day then averaged for each available day (5–7 days). • Given that there are no well-established actigraphy based cutoffs for FI, the data were divided into quartiles and those with an average FI ≥ 75th percentile were considered to have poor sleep.

Table 2

Percentage of women in worst category of sleep measures according to participant characteristics

Baseline Characteristic	Sample Size	Sleep Duration < 7 hours		WASO ≥ 75th percentile[a]		Sleep Fragmentation Index ≥ 75th percentile[a]		Sleep Midpoint > 5 AM	
		n (%)	p-value[b]	n (%)	p-value[b]	n (%)	p-value[b]	n (%)	p-value[b]
Overall	782	218 (27.9)		196 (25.1)		196 (25.1)		148 (18.9)	
Maternal age, in years									
<22	148	41 (27.7)	0.9571	60 (40.5)	<.0001	56 (37.8)	0.0001	57 (38.5)	<.0001
22 to 35	573	159 (27.7)		121 (21.1)		122 (21.3)		86 (15.0)	
>35	61	18 (29.5)		15 (24.6)		18 (29.5)		5 (8.2)	
Race/ethnicity									
White Non-Hispanic	496	110 (22.2)	<.0001	89 (17.9)	<.0001	90 (18.1)	<.0001	64 (12.9)	<.0001
Black Non-Hispanic	92	40 (43.5)		42 (45.7)		47 (51.1)		28 (30.4)	
Hispanic	122	39 (32.0)		42 (34.4)		37 (30.3)		40 (32.8)	
Asian	28	14 (50.0)		9 (32.1)		8 (28.6)		4 (14.3)	
Other	44	15 (34.1)		14 (31.8)		14 (31.8)		12 (27.3)	
BMI, in kg/m^2									
<25	412	109 (26.5)	0.0209	89 (21.6)	0.0086	92 (22.3)	0.0241	68 (16.5)	0.0110
25 to <30	194	46 (23.7)		46 (23.7)		47 (24.2)		34 (17.5)	
≥30	166	60 (36.1)		56 (33.7)		55 (33.1)		45 (27.1)	
Employment/school status									
Employed and in school	116	30 (25.9)	0.9466	28 (24.1)	<.0001	25 (21.6)	0.0382	14 (12.1)	<.0001
Employed and not in school	474	132 (27.8)		99 (20.9)		109 (23.0)		57 (12.0)	
Unemployed and in school	48	14 (29.2)		20 (41.7)		14 (29.2)		14 (29.2)	
Unemployed and not in school	104	27 (26.0)		42 (40.4)		37 (35.6)		47 (45.2)	
Below Poverty Level									
Yes	179	49 (27.4)	0.6457	72 (40.2)	<.0001	61 (34.1)	<.0001	55 (30.7)	<.0001
No	461	118 (25.6)		71 (15.4)		85 (18.4)		42 (9.1)	
Education status									
Less than high school	28	8 (28.6)	0.3623	16 (57.1)	<.0001	12 (42.9)	<.0001	12 (42.9)	<.0001
Completed high school or GED	89	29 (32.6)		40 (44.9)		42 (47.2)		43 (48.3)	

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Baseline Characteristic	Sample Size	Sleep Duration < 7 hours		WASO ≥ 75th percentile[a]		Sleep Fragmentation Index ≥ 75th percentile[a]		Sleep Midpoint > 5 AM	
		n (%)	p-value[b]	n (%)	p-value[b]	n (%)	p-value[b]	n (%)	p-value[b]
Some college	176	55 (31.3)		53 (30.1)		45 (25.6)		44 (25.0)	
Associate or technical degree	87	25 (28.7)		26 (29.9)		22 (25.3)		14 (16.1)	
Completed college	210	59 (28.1)		39 (18.6)		45 (21.4)		26 (12.4)	
Degree work beyond college	192	42 (21.9)		22 (11.5)		30 (15.6)		9 (4.7)	
Smoked during 3 months prior to pregnancy:									
Yes	113	38 (33.6)	0.1405	38 (33.6)	0.0231	39 (34.5)	0.0122	46 (40.7)	<.0001
No	669	180 (26.9)		158 (23.6)		157 (23.5)		102 (15.2)	
Frequent snoring before pregnancy[c]:									
Yes	96	29 (30.2)	0.4749	29 (30.2)	0.1386	28 (29.2)	0.1925	25 (26.0)	0.0130
No	573	153 (26.7)		133 (23.2)		132 (23.0)		90 (15.7)	
Employment schedule[c]:									
Regular day shift	441	121 (27.4)	0.9895	80 (18.1)	<.0001	92 (20.9)	0.0044	21 (4.8)	<.0001
Some form of shift work[d]	148	41 (27.7)		47 (31.8)		42 (28.4)		50 (33.8)	
Unemployed	152	41 (27.0)		62 (40.8)		51 (33.6)		61 (40.1)	
Ever diagnosed with sleep apnea[c]:									
Yes	12	4 (33.3)	0.7447	5 (41.7)	0.1955	6 (50.0)	0.0837	5 (41.7)	0.0441
No/Don't know	724	198 (27.3)		183 (25.3)		178 (24.6)		125 (17.3)	
Ever diagnosed with insomnia[c]:									
Yes	28	6 (21.4)	0.6657	9 (32.1)	0.5074	8 (28.6)	0.6594	10 (35.7)	0.0194
No/Don't know	709	196 (27.6)		180 (25.4)		177 (25.0)		120 (16.9)	
Ever diagnosed with restless legs syndrome[c]:									
Yes	10	1 (10.0)	0.2999	4 (40.0)	0.2871	2 (20.0)	1.0000	2 (20.0)	0.6923
No/Don't know	726	201 (27.7)		184 (25.3)		182 (25.1)		128 (17.6)	

[a] A cutoff of 56.0 minutes for WASO is the upper 25th percentile. A cutoff of 21.4% for sleep fragmentation is the upper 25th percentile.

[b] P-values are shown from Chi-square tests.

[c] Data collected by survey done as part of the nuMoM2b parent study, administered between 6^0–15^0 weeks of pregnancy

[d] Self-reported usual work schedule described as one of the following: night shift, split shift, afternoon shift, irregular shift/on call or rotating shift

Table 3

Association of sleep duration, continuity and timing with hypertensive disease of pregnancy and gestational diabetes

	Hypertensive disease of pregnancy		Gestational Diabetes	
Sleep characteristic	**N(%)**	**Crude OR**	**N(%)**	**Crude OR**
Sleep Duration				
<7 hours	27/218 (12.4)	1.10 (0.68, 1.78)	15/218 (6.9)	2.24 (1.11, 4.53)
≥7 hours	64/564 (11.3)	1.00	18/564 (3.2)	1.00
		p-value = 0.6850		*p-value = 0.0246*
Sleep Midpoint				
>5 AM	17/148 (11.5)	0.98 (0.56, 1.72)	12/148 (8.1)	2.58 (1.24, 5.36)
≤5 AM	74/634 (11.7)	1.00	21/634 (3.3)	1.00
		p-value = 0.9497		*p-value = 0.0114*
WASO				
≥75th percentile[a]	27/196 (13.8)	1.30 (0.80, 2.11)	10/196 (5.1)	1.32 (0.62, 2.82)
<75th percentile	64/586 (10.9)	1.00	23/586 (3.9)	1.00
		p-value = 0.2816		*p-value = 0.4789*
Sleep Fragmentation Index				
≥75th percentile[a]	25/196 (12.8)	1.15 (0.70, 1.88)	10/196 (5.1)	1.32 (0.62, 2.82)
<75th percentile	66/586 (11.3)	1.00	23/586 (3.9)	1.00
		p-value = 0.5730		*p-value = 0.4789*

[a]A cutoff of 56.0 minutes for WASO is the upper 25th percentile. A cutoff of 21.4% for sleep fragmentation is the upper 25th percentile.

Table 4

Association of sleep duration and timing with gestational diabetes[a]

Sleep Characteristic Categories	Gestational Diabetes n/N (%)	Crude OR, Point Estimate (95% CI) N = 782	Adjusted OR, Point Estimate (95% CI), After Adjustment for:					
			Age, linear & quadratic N = 782	BMI, linear & quadratic N = 772	Race/ethnicity (4 categories) N = 782	White, non-Hispanic (yes/no) N = 782	Frequent snoring (yes/no) N = 669	Employment Schedule (3 categories) N = 741
Sleep Duration <7 hours	15/218 (6.9)	2.24 (1.11, 4.53)	2.26 (1.12, 4.58)	2.12 (1.04, 4.30)	2.31 (1.13, 4.73)	2.25 (1.10, 4.60)	2.29 (0.97, 5.39)	2.42 (1.16, 5.06)
≥7 hours	18/564 (3.2)	1.00	1.00	1.00	1.00	1.00	1.00	1.00
		p-value = 0.0246	*p-value = 0.0232*	*p-value = 0.0380*	*p-value = 0.0220*	*p-value = 0.0266*	*p-value = 0.0586*	*p-value = 0.0190*
Sleep Midpoint >5 AM	12/148 (8.1)	2.58 (1.24, 5.36)	3.87 (1.74, 8.59)	2.41 (1.15, 5.07)	2.61 (1.22, 5.57)	2.62 (1.23, 5.58)	2.84 (1.16, 6.99)	3.71 (1.50, 9.21)
≤5 AM	21/634 (3.3)	1.00	1.00	1.00	1.00	1.00	1.00	1.00
		p-value = 0.0114	*p-value = 0.0009*	*p-value = 0.0202*	*p-value = 0.0132*	*p-value = 0.0124*	*p-value = 0.0229*	*p-value = 0.0047*

[a]Odds ratios are given to show the association between gestational diabetes and the sleep characteristic, without consideration of covariates and with separate adjustment for: age; BMI; race/ethnicity categories; white, non-Hispanic race/ethnicity; frequent snoring noted before pregnancy; and employment schedule (regular day shift, some form of shift work, unemployed). For race/ethnicity, Asian and other are collapsed.