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# Objectively-measured sleep duration and hyperglycemia in pregnancy

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## Abstract

**Objective—**Our primary purpose was to assess the impact of objectively-measured nighttime sleep duration on gestational glucose tolerance. We additionally examined associations of objectively-measured daytime sleep duration and nap frequency on maternal glycemic control.

**Methods—**63 urban, low-income, pregnant women wore wrist actigraphs for an average of 6 full days in mid-pregnancy prior to screening for hyperglycemia using the 1-hour oral glucose tolerance test (OGTT). Correlations of nighttime and daytime sleep durations with 1-hour OGTT values were analyzed. Multivariable logistic regression was used to evaluate independent associations between sleep parameters and hyperglycemia, defined as 1-hour OGTT values ≥ 130 mg/dL.

**Results—**Mean nighttime sleep duration was 6.9 ± 0.9 hours which was inversely correlated with 1-hour OGTT values ($r = -0.28$, $p = 0.03$). Shorter nighttime sleep was associated with hyperglycemia, even after controlling for age and body mass index (adjusted OR: 0.2; 95% CI: 0.1, 0.8). There were no associations of daytime sleep duration and nap frequency with 1-hour OGTT values or hyperglycemia.

**Conclusions—**Using objective measures of maternal sleep time, we found that women with shorter nighttime sleep durations had an increased risk of gestational hyperglycemia. Larger prospective studies are needed to confirm our negative daytime sleep findings.



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The study was conducted in Philadelphia at Temple University.

### Keywords

Sleep duration; Actigraphy; Pregnancy; Hyperglycemia; Gestational diabetes

## Introduction

Abnormalities in glucose metabolism are common in pregnancy and linked to adverse perinatal outcomes as well as longer-term complications of obesity and type 2 diabetes in both mothers and their children [1, 2]. Other than maternal weight and physical activity, established risk factors for hyperglycemia in pregnancy, including multiparity, non-white race/ethnicity, older age, and family history of diabetes, are not modifiable [3–5]. Given the expected rise in diabetes incidence over the next several decades [6], identifying factors associated with glucose intolerance in pregnancy that are amenable to change is a public health priority.

Strong evidence from observational studies suggests that shorter nighttime sleep durations are associated with abnormalities in glucose metabolism among non-pregnant women and men [7]. However, limited and somewhat conflicting data exist regarding the relationship between sleep duration and glucose metabolism in pregnancy. Reutrakul *et al.* found an inverse association between self-reported nighttime sleep duration and glucose values from 1-hour oral glucose tolerance testing in mid-pregnancy (correlation coefficient [$r$] = −0.21, $p < 0.01$) [8], Similarly, Facco *et al.* and Qui *et al.* reported an increased risk of impaired glucose tolerance among women with shorter self-reported nighttime sleep [9, 10]. A recent study by Balserak *et al.*, on the other hand, did not find associations of hyperglycemia with self-reported nighttime sleep or sleep duration from a single night of polysomnography (PSG) [11]. These pregnancy studies are limited by: 1) lack of objective measures of habitual nighttime sleep duration; 2) little data about daytime sleep (napping); and 3) failure to include significant numbers of ethnically diverse, socially disadvantaged women. Given that sleep often becomes increasingly disturbed as pregnancy progresses, characterized by more restless sleep, frequent awakenings, near daily naps, and a decline in nocturnal sleep time [12–14], data using objectively-measured nighttime and daytime sleep durations in diverse samples of pregnant women are urgently needed to clarify the relationship between sleep and maternal hyperglycemia.

The primary purpose of this study was to assess the impact of objectively-measured nighttime sleep duration via actigraphy on glucose tolerance during pregnancy among a sample of urban, low-income mothers. We additionally sought to examine the association of actigraphy-measured daytime sleep duration and nap frequency on maternal glycemic control.

## Methods

### Study population and design

Research subjects were participants in an ancillary study of Project BABIES, a prospective cohort study of urban, low-income pregnant women designed to examine the relationship between bacterial vaginosis and spontaneous preterm birth. Recruitment began in July 2008 at five university-affiliated outpatient prenatal care clinics in Philadelphia, as summarized previously [15]. Eligibility criteria included: < 16 weeks' gestation at enrollment, English or Spanish fluency, and current residence in Philadelphia. Ninety percent of women seen at the clinics consented to participate in Project BABIES, with a 5% refusal rate and a 5% missed rate. All participants provided written informed consent, and all procedures were in

accordance with ethical standards for human experimentation. The institutional review board of Temple University approved the study protocol.

From July 2009 to September 2010, Project BABIES participants aged 18 or older were invited to take part in our ancillary sleep study; 101 of 139 (73%) agreed to do so. We excluded women from the current analysis who had pre-existing diabetes (n=1), were missing data on glucose tolerance testing (n=13), had less than two full weekdays and one weekend day of actigraphy data (n=21), were at high risk for sleep apnea using the Berlin Questionnaire (n=2), or delivered twins (n=1), leaving a final sample of 63 participants. Compared to the 63 women included, those not included were somewhat heavier (36% obese versus 27% obese) and less likely to be African-American (76% versus 86%), but did not differ in age (23.4 years versus 23.4 years) or mean glucose level at glycemic screening (97 mg/dL vs. 98 mg/dL).

## Assessment of exposure: sleep duration

Sleep data were collected in mid-pregnancy (mean 21 weeks' gestation, range 18 to 25 weeks) before women were screened for hyperglycemia. Participants were asked to wear an actigraph wristwatch (AW-64, Philips Respironics) on their non-dominant wrist continuously for seven days and nights. Actigraphs use highly sensitive accelerometers to measure gross motor activity, analyzed to identify sleep periods [16]. The wrist actigraph was set to record in 30-second epochs using the medium threshold set by the manufacturer (40 activity counts/min) for detection of wake and sleep periods [17]. The actigraph device had an event marker that could be pressed to indicate specific times; participants were asked to press this marker when they laid down for naps or got in and out of bed at night. Over this same seven day period, participants were also asked to keep a daily sleep log, recording their nighttime sleep onset time, morning wake time, and any naps ≥ 5 minutes during the day. Sleep log records were used to determine nap frequency and to aid in scoring the actigraph data when participants forgot to push their event markers. All participants were instructed in the use of the wrist actigraph and how to complete the sleep diary. Telephone support was available to participants throughout the monitoring period.

Actigraphy data were transferred via interface to a computer and analyzed by two trained investigators with the use of Actiware Software version 5.5 (Phillips Respironics). Sleep onset time and sleep end time (for both nighttime and daytime sleep) were set by the investigators based on review of the downloaded actogram and verified against the time recorded in the participants' sleep logs. If the actogram and sleep log data were discordant, and telephone clarification with the participant was not possible, the day's data were discarded.

Nighttime sleep duration was calculated as the sum of the sleep epochs within the interval between nocturnal sleep onset time and morning wake time; daytime sleep duration was calculated as the sum of the sleep epochs within the intervals between nap sleep onset times and nap wake times. Epochs over 40 activity counts within these intervals were scored as awakenings by Actiware software.

Main exposures derived from actigraphy included: 1) Total nocturnal sleep duration, obtained by averaging the total time in hours spent sleeping on all available nights; and 2) Total nap duration, obtained by averaging the total time in hours spent napping on the days participants napped. We additionally examined the frequency at which participants self-reported napping and categorized them into two groups based on prior literature in pregnant women [14]: 1) frequent nappers (4 or more days with naps); and 2) infrequent nappers (less than 4 days with naps).

### Assessment of outcome: maternal hyperglycemia

Participating women underwent routine glycemic screening for gestational diabetes (GDM) early in the third trimester (mean 29 ± 3 weeks' gestation) with a non-fasting oral glucose tolerance test (OGTT), in which venous blood was sampled 1 hour after a 50-gram oral glucose load. If the 1-hour glucose result was ≥ 130 mg/dL, the participant was referred for a 100-gram fasting glucose 3-hour OGTT. Normal 3-hour OGTT results were a blood glucose < 95 mg/dL at baseline, < 180 mg/dL at 1 hour, < 155 mg/dL at 2 hours, and < 140mg/dL at 3 hours [18]. GDM was diagnosed if two or more values were abnormal on the 3-hour OGTT.

In this study, outcomes examined included mean 1-hour OGTT values and 1-hour OGTT values ≥ 130 mg/dL. We chose the cut-point of 130 mg/dL to define hyperglycemia to be consistent with prior literature [9] and because glucose values at this level or higher are associated with adverse pregnancy outcomes [19, 20]. The small number of GDM cases in our sample (n=1) precluded our ability to look at this outcome alone.

### Assessment of covariates

At the time of their sleep measurement, participants completed questionnaires assessing sociodemographics and medical factors, including maternal age, race/ethnicity, parity, medical insurance (income proxy), education, history of GDM in a prior pregnancy, and smoking habits. We calculated body mass index (BMI) from measured first trimester height and weight, and categorized women as either obese (BMI ≥ 30 kg/$m^2$) or not obese (BMI < 30 kg/$m^2$). Sixty (95%) participants had a first trimester weight available for analysis; 58% of who had a measured weight before the ninth week of gestation, and 73% before the eleventh week. For the remaining three women, whose initial measured weight ranged from 14 to 21 weeks' gestation, we created a regression model to predict first trimester prenatal weight (details of which have been reported previously) [15].

### Statistical analysis

Univariate distribution of the outcomes, exposures, and covariates were examined for normality. Square root transformation was used to normalize the skewed distribution of daytime sleep duration; log transformation was used to normalize 1-hour OGTT values. Given that analyses of the transformed data derived identical patterns of results, here we report results on the analyses of the raw data to aid in interpretation. To identify differences in distribution for each covariate by sleep exposure status, Fisher's exact tests were used for categorical variables and two-sided t-tests were used for continuous variables relaxing the assumption of equal variances when indicated. Pearson's correlations and two-sided t-tests were used to assess relationships of sleep duration parameters and nap frequency with 1-hour OGTT values. All parametric comparisons were confirmed with Mann–Whitney U-tests and Spearman's rho to ensure that violations of normality did not affect results. Multivariable logistic regression was used to evaluate the extent to which nighttime sleep duration, daytime sleep duration, and nap frequency were each independently associated with hyperglycemia, adjusting for BMI and age. Adjustment for other characteristics, including parity, race/ethnicity, and gestational age at sleep assessment, did not affect results and thus, we did not include these variables in the final models. We used SAS version 9.3 (SAS Institute, Cary, NC) to carry out all analyses.

## Results

Table 1 presents selected baseline characteristics by nighttime sleep duration, daytime sleep duration, and nap frequency. The majority of participants were Black or African-American (86%), unemployed (71%), Medicaid recipients (100%), and multiparous (51%). Mean age

was 23.4 ± 4.8 years and mean early pregnancy BMI was 26.9 ± 5.6 kg/m$^2$. Only one mother (2%) had a history of GDM in a prior pregnancy. Participant characteristics did not vary by nighttime sleep duration or nap frequency. However, nap duration was longer among leaner mothers and those self-identifying as Black or African-American.

On average, participants wore the actigraph wristwatch for 6 full days (range 4 to 7 days), with the vast majority (70%) wearing the device for 6 or 7 full days. Mean measured nighttime sleep duration was 6.9 ± 0.9 hours. While the majority of participants' bedtimes were after midnight (54%), mean wake time was early in the morning (8:35 am), and nighttime sleep was often disrupted (mean time awake after sleep onset was 1.5 ± 0.6 hours). Napping was common; 95% napped at least once during the week-long study period, with a quarter (25%) defined as frequent nappers. Those who napped at least once per week took an average of 2.7 ± 1.4 naps each week that lasted 1.7 ± 0.8 hours. There was no difference in daytime sleep duration among frequent nappers and infrequent nappers (1.6 hours vs. 1.7 hours respectively, p = 1.0).

There was an inverse correlation between nighttime sleep duration and 1-hour OGTT values ($r = -0.28$, p = 0.03) such that each hour of shorter nighttime sleep was associated with an 8.2 mg/dL increase in glucose. Neither daytime sleep duration nor nap frequency were associated with higher glucose values.

A total of 7 women (11%) were classified with hyperglycemia using the 1-hour OGTT (≥ 130 mg/dL). Mean nighttime sleep duration was 1 hour shorter among participants with hyperglycemia (6.0 ± 1.0 hours/night) than those without hyperglycemia (7.0 ± 0.8 hours/night, p = 0.007; Table 2). Even after controlling for age and early pregnancy BMI, shorter nighttime sleep was associated with hyperglycemia, while longer sleep duration was protective against hyperglycemia (adjusted odds ratio [OR]: 0.2; 95% confidence interval [CI]: 0.1, 0.8). There were no associations of daytime sleep duration or nap frequency with hyperglycemia in unadjusted or adjusted models (Table 2).

## Discussion

In this first prospective study using objective measures of habitual sleep duration to explore the relationship between sleep time and hyperglycemia in pregnancy, we found that women with shorter nighttime sleep durations in mid-pregnancy had an increased risk of gestational hyperglycemia measured early in the third trimester. Adjustment for important covariates, namely age and early pregnancy BMI category, did not change our results. Our findings are consistent with associations between sleep duration and diabetes risk in non-pregnant populations [21] and support data linking self-reported nighttime sleep durations during pregnancy with abnormal glucose tolerance and GDM [8–10]. We did not find links among daytime sleep duration and nap frequency with gestational hyperglycemia.

The use of objective measures to evaluate habitual sleep duration and outcomes in pregnancy is critical, given recent investigations reporting little agreement between objective and subjective assessments of gestational sleep time [22, 23]. Questions have been raised about whether significant associations between self-reported pregnancy-related changes in sleep time and health are due to actual sleep duration. Our findings confirm a relationship between shorter nighttime sleep and gestational hyperglycemia using actigraphy, a measure well-correlated with PSG for assessing sleep duration (even in pregnancy) [24, 25] and often preferable because actigraphy is unobtrusive, ambulatory, and can record for multiple days and nights at a much lower cost than PSG. Actigraphy eliminates the "first night effect" seen with PSG [26], which may explain why our results differed from Balserak *et al.*, who did not find a relationship between hyperglycemia and

sleep duration from a single night of PSG [11]. Actigraphy also offered the opportunity to objectively measure nap time. While longer self-reported nap times and greater nap frequency have been linked to increased risk of diabetes in the literature [11, 27, 28], we did not find associations of objectively-measured daytime sleep duration or nap frequency with hyperglycemia in this study. Given our small sample size, however, larger prospective studies with diverse samples are needed to confirm these findings, so to clarify whether daytime naps could be used to compensate for shorter sleep duration at night.

Experimental studies in non-pregnant persons provide some insight into the biologic basis of our results. Exposing 17 healthy volunteers to 24 hours of sleep deprivation resulted in significant decreases in glucose utilization in several cortical and subcortical regions of the brain compared to a baseline rested state, as observed with positron emission tomography [29]. Diminished brain glucose uptake results in increased exposure of peripheral tissues to higher glucose concentrations, which may facilitate the development of insulin resistance. Additionally, restricting 11 healthy men to 4 hours of nightly sleep over 6 days resulted in reductions in acute insulin response and glucose disposal rate along with elevations of both evening growth hormone and cortisol levels [30, 31]. Increased concentrations of growth hormone and cortisol can lead to reduced insulin sensitivity (particularly in muscle cells), resulting in decreased glucose uptake and elevated blood glucose levels. Men exposed to 6 nights of sleep debt also had evidence of increased sympathetic tone, which at the level of the pancreas, may result in a reduction of insulin secretion from beta-cells [31]. These findings support the hypothesis that nighttime sleep curtailment affects pathways involved in glycemic control and perhaps leads to diabetes. In pregnancy, short sleep duration has been shown to alter the inflammatory state [32], which can affect insulin signaling, as well as insulin sensitivity, suggesting another pathway by which sleep may impair glucose control [33].

While the small number of GDM cases precluded our ability to examine the relationship between sleep duration and GDM, our findings among women with a more modest degree of glucose intolerance still may be clinically significant, given strong evidence of adverse pregnancy outcomes among this group [34]. Several studies have found that mothers who failed the 1-hour OGTT but passed the 3-hour OGTT were at increased risk of pregnancy induced hypertension, cesarean section due to fetal distress, and 3-month postpartum reductions in insulin sensitivity compared to those with normal glucose tolerance in pregnancy [35–37]. Because a much larger proportion of pregnant women have less pronounced hyperglycemia than GDM [38], interventions to promote longer gestational nighttime sleep may have substantial population impact. Experimental studies examining sleep extension among mothers with varying degrees of glucose intolerance that have longer-term maternal and infant outcomes are needed to determine if clinically significant benefits, particularly among those mothers with more modest degrees of glucose intolerance, can be derived.

Our study has a number of strengths including the use of objective measures of habitual sleep time collected prior to glycemic screening, our focus on urban, low-income women, and the examination of both daytime and nighttime sleep durations on hyperglycemia in pregnancy. Nevertheless, our findings must be interpreted within the context of the study design. Due to our sample size, we lacked the statistical power to stratify analyses based on BMI and age or to look at our nocturnal sleep exposure variable in categories. Larger studies among samples of women from a variety of incomes and racial/ethnic groups are needed to provide more generalizable, precise estimates of effect. We did not assess family history of diabetes, and thus, adjustment for this variable in our final models was not possible. We measured sleep duration at only one time period in pregnancy, which may not be reflective of sleep time before or after our measurement. We also lacked a comprehensive assessment

of sleep duration before pregnancy and cannot be certain whether shorter sleep duration during pregnancy alone may increase the risk of hyperglycemia.

In summary, we found that shorter objectively-measured nighttime sleep durations were associated with hyperglycemia during pregnancy. As glucose intolerance less severe than that used to define overt diabetes is related to clinically important perinatal disorders [34–37], strategies to prevent maternal hyperglycemia are critical to the design of interventions to improve outcomes in both mothers and their infants. Our results suggest that nighttime sleep duration may be a promising target, yet more work is needed to test whether extending sleep duration affects not only gestational hyperglycemia, but also health outcomes in mothers and their children. While we did not find associations of daytime sleep duration and nap frequency with glucose intolerance in pregnancy, larger prospective studies are needed to confirm these negative findings.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

## Acknowledgments

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## Table 1

Participant baseline characteristics by objectively-measured nighttime sleep duration, by objectively-measured daytime sleep duration, and by self-reported nap frequency in mid-pregnancy (18–25 weeks' gestation)

| | Nighttime sleep duration (hours/night) Mean (SD) | *P value* | Daytime sleep duration (hours/day) Mean (SD) | *P value* | Nap Frequency[a] | | |
|---|---|---|---|---|---|---|---|
| | | | | | Frequent nappers n (%) | Infrequent nappers n (%) | *P value* |
| **Age (years)** | | | | | | | |
| < 25 | 6.9 (0.9) | 0.4 | 1.7 (0.9) | 0.4 | 10 (63%) | 37 (79%) | 0.3 |
| ≥ 25 | 6.7 (0.8) | | 1.5 (0.9) | | 6 (37%) | 10 (21%) | |
| **Race/ethnicity** | | | | | | | |
| Black or African-American | 6.8 (0.9) | 0.2 | 1.7 (0.9) | 0.03 | 14 (88%) | 40 (85%) | 1.0 |
| Other | 7.1 (0.5) | | 1.1 (0.8) | | 2 (12%) | 7 (15%) | |
| **Education** | | | | | | | |
| Did not complete high school | 7.0 (0.9) | 0.4 | 1.4 (1.0) | 0.1 | 6 (37%) | 14 (30%) | 0.8 |
| High school degree or more | 6.8 (0.9) | | 1.8 (0.8) | | 10 (63%) | 33 (70%) | |
| **Employment** | | | | | | | |
| Unemployed | 6.9 (0.9) | 0.6 | 1.7 (0.9) | 0.6 | 10 (63%) | 35 (74%) | 0.4 |
| Employed | 6.8 (0.9) | | 1.6 (0.9) | | 6 (37%) | 12 (26%) | |
| **Early pregnancy BMI category** | | | | | | | |
| < 30 kg/m$^2$ | 6.8 (0.9) | 0.8 | 1.8 (1.5) | 0.04 | 11 (69%) | 35 (74%) | 0.7 |
| ≥ 30 kg/m$^2$ | 6.9 (1.0) | | 1.3 (0.8) | | 5 (31%) | 12 (26%) | |
| **GDM in a previous pregnancy** | | | | | | | |
| Previous GDM | 7.2 (−) | 0.9 | 1.7 (−) | 0.4 | 0 (0%) | 1 (2%) | 0.7 |
| No previous GDM | 6.8 (0.7) | | 1.5 (1.0) | | 7 (44%) | 24 (51%) | |
| Nulliparous | 6.9 (1.1) | | 1.8 (0.8) | | 9 (56%) | 22 (47%) | |

To identify differences in distribution for each covariate by sleep exposure status, Fisher's exact tests were used for categorical variables and two-sided t-tests were used for continuous variables relaxing the assumption of equal variances when indicated. All parametric comparisons were confirmed with Mann–Whitney U-tests to ensure that violations of normality did not affect results.

[a]Frequent nappers included participants who had 4 or more days with naps; infrequent nappers included participants who had less than 4 days with naps.

**Table 2**

Associations of objectively-measured nighttime sleep duration, objectively-measured daytime sleep duration, and self-reported nap frequency with hyperglycemia in pregnancy

| | 1-hour oral glucose tolerance test results | | | |
|---|---|---|---|---|
| | **Hyperglycemia, ≥ 130mg/dL Mean (SD) or n (%)** | **No hyperglycemia, < 130mg/dL Mean (SD) or n (%)** | **Unadjusted odds of hyperglycemia OR (95% CI)** | **Adjusted odds of Hyperglycemia[a] OR (95% CI)** |
| Nighttime sleep duration (hours) | 6.0 (1.0) | 7.0 (0.8)[b] | 0.3 (0.1, 0.8) | 0.2 (0.1, 0.8) |
| Daytime sleep duration (hours) | 2.1 (0.7) | 1.7 (0.8)[c] | 1.7 (0.6, 4.8) | 2.8 (0.8, 9.8) |
| Nap frequency | | | | |
| Frequent nappers (≥ 4 naps/week) | 2 (29%) | 14 (25%)[d] | 1.2 (0.2, 6.9) | 0.9 (0.1, 5.9) |
| Infrequent nappers (< 4 naps/week) | 5 (71%) | 42 (75%) | Referent | Referent |

Mean timing of hyperglycemia assessment = 29 weeks' gestation; mean timing of sleep assessment = 21 weeks' gestation.

[a] Adjusted for age and body mass index.

[b] $t$ (61, n = 63) = 2.77, p = 0.007, participants with hyperglycemia versus no hyperglycemia.

[c] $t$ (58, n = 60) = −1.06. p = 0.3, participants with hyperglycemia versus no hyperglycemia.

[d] Fisher's exact test (n = 63), p = 1.0, participants with hyperglycemia versus no hyperglycemia.