

# Evening blue-light exposure, maternal glucose, and infant birthweight

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## Abstract

Maternal–fetal consequences of exposure to blue-wavelength light are poorly understood. This study tested the hypothesis that evening blue-light exposure is associated with maternal fasting glucose and infant birthweight. Forty-one pregnant women (body mass index =  $32.90 \pm 6.35$  kg/m<sup>2</sup>; 24–39 years old; 16 with gestational diabetes mellitus [GDM]) wore actigraphs for 7 days, underwent polysomnography, and completed study questionnaires during gestational week  $30 \pm 3.76$ . Infant birthweight ( $n = 41$ ) and maternal fasting glucose ( $n = 30$ ; range = 16–36 weeks) were recorded from the mothers' medical charts. Blue-light exposure was obtained from Actiwatch-Spectrum recordings. Adjusted and unadjusted linear regression analyses were performed to determine sleep characteristics associated with maternal fasting glucose and infant-birthweight. The mean fasting mid- to late-gestation glucose was  $95.73 \pm 24.68$  mg/dl and infant birthweight was  $3271 \pm 436$  g. In unadjusted analysis, maternal fasting glucose was associated with blue-light exposure ( $\beta = 3.82$ ,  $p = 0.03$ ). In the final model of multiple linear regression for fasting glucose, evening blue-light exposure ( $\beta = 4.00$ ,  $p = 0.01$ ) remained significant after controlling for gestational weight gain, parity, sleep duration, and GDM. Similarly, blue-light exposure was associated with infant birthweight ( $69.79$ ,  $p = 0.006$ ) in the unadjusted model, and remained significant ( $\beta = 70.38$ ,  $p = 0.01$ ) after adjusting for weight gain, wakefulness after sleep onset, gestational age at delivery, and GDM. Higher blue-light exposure in pregnancy is associated with higher fasting glucose and infant birthweight. Reduced use of electronic devices before bedtime is a modifiable behavior.

## KEYWORDS

adverse pregnancy outcomes, blue light, infant birthweight, maternal glucose, pregnancy

## INTRODUCTION

Pregnancy is associated with altered maternal sleep quality and duration due to pregnancy-related changes.<sup>1</sup> External factors, such as light exposure, also drive alterations in sleep parameters that affect a

number of endocrine and metabolic functions, such as glucose regulation and growth hormone release.<sup>2–4</sup> There is evidence that maternal sleep disturbances during pregnancy, such as short sleep duration, poor sleep quality, and sleep apnea, are poor prognostic factors for the mothers, who often have a greater risk of maternal hyperglycemia.

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Higher maternal glycemia has a known negative effect on the fetus, including altered growth and gestational length.<sup>5,6</sup> Accumulating evidence shows that subjective and objective short sleep duration and poor sleep quality due to internal and external factors are associated with increased risk of gestational diabetes, pregnancy-induced hypertension, preeclampsia, and small- or large-for-gestational age. Yet, the effect of light exposure on maternal and fetal outcomes is unknown.

Natural light is the most potent entraining signal for the circadian clock,<sup>7</sup> and it controls the circadian regulation of energy homeostasis. The entrainment of circadian rhythm leads to temporal coordination of physiology and behavior.<sup>8–13</sup> However, artificial light-at-night (ALAN) from normal ambient room lighting and computer, TV, smartphone, and tablet screens lit by light-emitting diodes (LEDs) suppresses melatonin release, increases alertness, shifts the timing of the circadian clock, and impairs the brain's restorative slow waves during deep sleep.<sup>8–13</sup> The disruption of clock function causes circadian misalignment, which can result in adverse health outcomes, such as mood disorders, impaired glucose metabolism, and obesity.<sup>14–20</sup> A recent large prospective cohort study of women reported that exposure to ALAN while sleeping was significantly associated with an increased risk of weight gain and the development of obesity.<sup>13</sup> In a national sleep survey, 90% of American adults reported using some type of LED device within the hour before bedtime.<sup>21</sup> Considering the near ubiquity of personal LED devices, high portions of women of reproductive age are voluntarily engaging in modifiable behaviors that may disturb their sleep and circadian rhythm, increasing the risk of obesity<sup>22</sup> and excessive weight gain during pregnancy and adverse maternal outcomes.<sup>23</sup>

Although evidence has suggested a potential impact of circadian rhythm disruptions on pregnancy outcomes,<sup>24,25</sup> this line of research so far has been limited to studies on the effect of night shift work on these outcomes, in which exposure to nighttime light is not measured (only inferred). Causal estimations of that impact are still lacking. Given the deleterious effect of light exposure on circadian system and health outcomes, understanding the effects of light exposure is important, particularly in vulnerable populations, such as pregnant women. However, there has been little investigation of the association of ALAN exposure during pregnancy with perinatal and infant outcomes. While significant occurrences of bleeding, miscarriage, and assisted delivery among night workers potentially exposed to evening blue light have been observed in previous studies<sup>24–26</sup> compared to day workers, the direct influence of evening blue-light exposure on mothers and, indirectly, their newborn infants has not been investigated previously.

Given the potential clinical impact of evening blue-light exposure on maternal–fetal well-being, it is crucial to evaluate independent associations between blue-light exposure and maternal–fetal well-being using objective and subjective sleep measures, conducting analyses in a way that controls for relevant covariates. In this study, our objective was to examine the relationship between mid- to late-gestation maternal blue-light exposure measured via actigraphy, fasting glucose measured in the mid-late pregnancy, and newborn birthweight.

## METHODS

Maternal blue-light exposure was examined as a substudy of a parent trial<sup>5</sup> in which sleep-related determinants of gestational diabetes were investigated. A total of 41 pregnant women with and without gestational diabetes, with available infant birthweight and blue-light exposure data, were included in this study.

### Procedures and measurements

This study was approved by the University of Illinois at Chicago (UIC) Institutional Review Board. We obtained medical, obstetric, and sleep histories at baseline evaluation. All participants completed questionnaires related to biometric and demographic features (e.g., maternal age, race/ethnicity, employment status and shift work [no vs. yes: rotating shift or night shift work], and sleep, including the Pittsburgh sleep quality index [PSQI]).<sup>27</sup> All participants underwent an overnight polysomnography (PSG) study at the UIC Sleep Science Center (30.38 ± 3.76 weeks gestation) and were asked to wear an actigraph for 7 days accompanied by sleep diaries. Of those participants, 30 pregnant women had available fasting glucose results during mid-late pregnancy. Gestational diabetes mellitus (GDM) cases were diagnosed<sup>28</sup> based on the following criteria: (1) glucose challenge test (GCT) ≥ 140 mg/dl with 2 or more abnormal values on a 3-h oral glucose tolerance test (OGTT: ≥95 mg/dl at baseline, ≥180 mg/dl at 1 h, ≥155 mg/dl at 2 h, or ≥140 mg/dl at 3 h) or (2) nonfasting 50-g GTT ≥200 mg/dl if no fasting 3-h GTT was performed. Seven women with GDM were treated with oral diabetes medication and two with insulin. Infant birthweight and fasting glucose values were extracted from medical records after delivery.

### Demographic and clinical measures

We obtained demographic and anthropometric measures using self-report questionnaires (e.g., maternal age, race/ethnicity, employment status, income, education, work schedule, and prepregnancy weight based on medical charts). The pregnancy body mass index (BMI) at the third trimester was calculated from weight and height measured during the visit to Sleep Science Center using an upright scale and wall-mounted stadiometer, respectively. We calculated total gestational weight gain as the difference between weight that was recorded at the admission to hospital for delivery and prepregnancy weight measured within 1 year obtained from the medical records.

### Actigraphy measures

The Actiwatch Spectrum (Philips Respironics, Andover, MA, USA) was used to assess the nocturnal sleep parameters. It has the capability to assess ambient light exposure in both pregnant and nonpregnant adults.<sup>29–33</sup> The device was configured to collect data in 30-s

epochs. All participants were instructed to wear the Actiwatch on the nondominant wrist on the PSG night and the following 7-day period and to complete daily sleep diaries for the same 7-consecutive-day period. Data were transferred to a computer to be analyzed by trained investigators. Actigraphy data were cross-referenced with daily sleep diaries. The first night recording performed at the UIC Sleep Science Center was excluded due to the women being away from their natural environment. For analysis, Actiware Software version 6.0.8 (Phillips Respironics) was used. Each epoch was scored as sleep or wake based on the algorithm, as determined by a combination of activity count and immobility threshold.<sup>30</sup> A separate score was assigned for *blue-wavelength light exposure*, scoring only evening data before sleep. Average light exposure in our study was 3 h 8 min  $\pm$  8 min (SD; maximum 3 h 16 min) before lights off indicated by women pressing the event button on the Actiwatch. Light exposure ( $\mu\text{W}/\text{cm}^2$ ) was recorded in 1-min bins via photodiode-based light sensors. Photopic light exposure was estimated by the Actiwatch software as a weighted function of the RGB signal. This study focused on only blue-light (400–500 nm) emission.<sup>34</sup> When the device was not worn, these periods were excluded from analysis on the assumption that the exposure to light was not being measured. Each participant was required to have a minimum of 3 days of valid data (maximum 7 days).

## Overnight polysomnography

All participants underwent an overnight sleep assessment by PSG at the UIC Sleep Science Center. PSGs (Respironics Alice5 at UIC) included electroencephalogram (EEG; central, frontal, occipital leads), electrooculogram, submental and tibialis electromyograms, single lead electrocardiogram, finger pulse oximetry, oro-nasal thermistor and nasal pressure, thoracoabdominal belts (piezo crystals), and snoring and body position sensors.<sup>35</sup> All sleep stages were scored according to standardized criteria.<sup>36</sup> Hypopnea events were scored using the accepted classification requiring a  $\geq 30\%$  drop in respiratory flow for  $\geq 10$  s with  $\geq 3\%$  oxygen desaturation or an arousal.<sup>37</sup> We defined apnea as a  $> 90\%$  decrease in airflow for  $\geq 10$  s. The apnea-hypopnea index (AHI) was defined as the number of apneas and hypopneas per hour of sleep. Based on the AHI (events/h), the diagnosis of obstructive sleep apnea (OSA) was made as no OSA (AHI  $< 5$ ) or yes OSA (AHI  $\geq 5$ ). The sleep studies were scored by two trained sleep technologists.

## Pittsburgh sleep quality index

The PSQI (completed before PSG on the same day) is a well-validated, 19-item, self-reported measure that assesses sleep quality and severity of specific sleep-related complaints over the previous month.<sup>27</sup> Additionally, five questions regarding snoring and apnea are rated by a bed partner/roommate. The PSQI provides a total score for sleep quality and has seven subscale scores, including sleep duration, latency, efficiency, and daytime functioning. The total PSQI score ranges from 0 to 21 and subscale scores range from 0 to 3, with higher scores reflecting

poorer sleep. Higher total score (referred to as global score) indicates worse sleep quality. The PSQI has demonstrated good validity and reliability (Cronbach's  $\alpha > 0.80$ ).<sup>27</sup> Jomeen and Martin also showed that the PSQI has good internal consistency and convergent and divergent reliability among pregnant women.<sup>38</sup>

## Statistical analysis

Unadjusted and multivariate linear regression analyses were performed to determine if sleep characteristics were significantly associated with maternal fasting glucose and infant birthweight. Using maternal fasting glucose and infant birthweight as outcome variables, confounding factors were evaluated in full linear regression models as independent variables.

In unadjusted regression analyses, the dependent variables were maternal fasting glucose and infant birthweight. Covariates included were parity (the number of pregnancies), gestational age at delivery, nocturnal sleep duration in hours, and wake after sleep onset (WASO) in minutes according to the Actigraphy recordings, self-reported sleep duration and quality based on PSQI, AHI, OSA (yes/no), and gestational diabetes diagnosis (yes/no).

Data analyses were conducted using Stata 17.0 (StataCorp LP, College Station, TX, USA) and SPSS 24.0 (SPSS Inc., Chicago, IL, USA). Prior to data analysis, missing data and outliers were checked. Descriptive statistics (mean  $\pm$  SD or percentage) were examined. Statistical significance was set at  $p < 0.05$  (two-tailed).

## RESULTS

### Sample characteristics

Characteristics of the pregnant women and newborns are presented in Table 1. Overall, these were healthy pregnant women with mild GDM who had a low incidence of hypertension, pre-eclampsia, and OSA. There were 22 (53.66%) women who gave birth to boys and 19 (46.34%) women who gave birth to girls. The participants were on average  $31.66 \pm 3.65$  years old. Sixteen women (39%) were African-American; 18 (~44%) were Hispanic women. The average gestational age was  $30.38 \pm 3.76$  weeks during data collection (PSG, actigraphy, and sleep diaries), and their mean BMI was  $32.90 \pm 6.35$  kg/m<sup>2</sup>. The mean prepregnancy BMI obtained from the medical record was  $30.27 \pm 8.16$  kg/m<sup>2</sup>. The mean weight gain was  $10.38 \pm 6.90$  kg. The average infant birthweight was  $3276.29 \pm 424.24$  g (gestational age at delivery: 33–41 weeks). Maternal fasting glucose ( $n = 30$ ; gestational weeks: 20–36) was  $95.73 \pm 24.68$  mg/dl.

### Associations between sleep variables and blue light

None of the sleep parameters, including WASO, objective and subjective sleep duration, sleep quality, and AHI, were significantly associated with evening blue-light exposure ( $p$  values: 0.53, 0.33, 0.48, 0.92, and 0.67, respectively).

**TABLE 1** Characteristics of the 41 participants and their infants

Variable	Mean $\pm$ SD	No. (%)
Age (years)	31.66 $\pm$ 3.65	
Gestational age (weeks)	30.38 $\pm$ 3.76	
Race/ethnicity		
White/others		7 (17.10)
African American		16 (39.00)
Hispanic		18 (43.90)
Pregnancy BMI (kg/m <sup>2</sup> )	32.90 $\pm$ 6.35	
Prepregnancy BMI (kg/m <sup>2</sup> )	29.32 $\pm$ 6.65	
Gestational weight gain (kg)	10.38 $\pm$ 6.90	
Education		
College/higher education		24 (58)
High school or lower		17 (42)
Income		
\$24,999 or less		12 (29)
\$25,000–\$49,999		1 (3)
\$50,000–\$74,999		12 (29)
\$75,000–\$99,999		2 (5)
\$100,000 or more		5 (12)
Unknown		9 (22)
Parity (nulliparous)		29 (71)
Objective sleep duration (h)	7.36 $\pm$ 1.03	
WASO (min)	69.14 $\pm$ 48.12	
Subjective sleep duration (h)	6.65 $\pm$ 1.75	
Fasting glucose, mg/dl (n = 30)	95.73 $\pm$ 24.68	
Infant weight (g)	3271.29 $\pm$ 435.57	
Gestational age at delivery (weeks)	38.81 $\pm$ 1.67	
GDM diagnosis, yes		25 (60.98)
GDM diagnosis with nonfasting GTT		9 (36.00)
OGTT diagnosis with OGTT		16 (64.00)
OGTT–0 h (mg/dl)	83.73 $\pm$ 7.45	
OGTT–1 h (mg/dl)	195.53 $\pm$ 24.05	
OGTT–2 h (mg/dl)	170.67 $\pm$ 31.75	
OGTT–3 h (mg/dl)	120.2 $\pm$ 37.53	
Oral med. treatment of GDM		7 (23.23)
Insulin treatment of GDM		2 (6.67)
OSA diagnosis, yes		5 (12.20)
AHI (events/h)	2.12 $\pm$ 4.36	
Delivery method		
Vaginal delivery		28 (68.29)
Planned C-section		4 (9.76)
Emergency C-section		9 (21.95)
Preterm >37 weeks		35 (85.37)
<37 weeks		6 (14.63)
Preeclampsia, yes		4 (9.76)

(Continues)

**TABLE 1** (Continued)

Variable	Mean $\pm$ SD	No. (%)
Pregnancy-induced hypertension, yes		4 (9.76)

Abbreviations: AHI, apnea-hypopnea index; BMI, body mass index; GDM, gestational diabetes mellitus; OGTT, oral glucose tolerance test; OSA, obstructive sleep apnea; WASO, wake after sleep onset.

### Associations between maternal fasting glucose during pregnancy, blue-light exposure, and sleep variables

We examined demographic and sleep characteristics associated with fasting glucose level ( $n = 30$ , Table 2). Fasting glucose based on medical charts was measured in the second half of pregnancy (mean gestational age = 26.05  $\pm$  5.45 weeks). In unadjusted analysis, fasting glucose was significantly associated with blue-light exposure ( $\beta = 3.82$ ,  $p = 0.03$ ). Fasting glucose was also associated with parity ( $\beta = -18.85$ ,  $p = 0.05$ ) and actigraphy-measured sleep duration ( $\beta = 11.08$ ,  $p = 0.01$ ). Nevertheless, fasting glucose was not associated with self-reported nocturnal sleep duration, sleep quality, GDM diagnosis, PSG-diagnosed OSA (AHI > 5, yes/no), AHI, WASO, or shift work. Additionally, the association of fasting glucose with other known confounding variables was not significant (Table 2).

### Associations between blue-light exposure, sleep variables, fasting glucose, and infant birthweight

In unadjusted analysis (Table 2), blue-light exposure during pregnancy was significantly associated with infant weight ( $\beta = 69.79$ ,  $p = 0.006$ ). Actigraphy-measured sleep duration, self-reported sleep duration, sleep quality, PSG-diagnosed OSA (AHI > 5), and other known confounding variables were not associated with infant birthweight (Table 2).

### Multivariate models investigating associations between blue-light exposure and sleep variables and maternal fasting glucose

In the final multiple linear model where maternal fasting glucose was the dependent variable, evening blue-light exposure ( $\beta = 4.00$ ,  $p = 0.01$ ) remained significant after controlling for parity, actigraphy-measured sleep duration, weight gain during pregnancy, and GDM diagnosis. The coefficient demonstrates that for each unit increase in evening blue-light exposure, there was a 4 mg/dl increase in fasting glucose concentration (Table 3). In this model, nocturnal sleep duration by actigraph was also associated with fasting glucose ( $\beta = 9.32$ ,  $p = 0.02$ ). In separate models, we replaced GDM diagnosis with OSA diagnosis or AHI. None were significantly associated with fasting glucose (OSA diagnosis:  $\beta = -4.33$ ,  $p = 0.42$ ; AHI:  $\beta = -0.16$ ,  $p = 0.84$ ).

**TABLE 2** Unadjusted relationships between sleep, anthropometric, and other independent variables with maternal fasting glucose and infant birthweight as dependent variables

Independent variables	Fasting coefficients	Glucose std. error	p value	Birthweight coefficients	Weighted std. error	p value
Evening blue light ( $\mu\text{W}/\text{cm}^2$ )	3.82	1.66	0.03	69.79	24.11	0.006
Age (years)	-0.11	1.19	0.93	5.50	18.96	0.77
Gestational age (weeks)	0.64	0.95	0.51	-9.35	13	0.
Parity	-18.85	9.05	0.05	102.19	149.57	0.50
Race and ethnicity						
White/others	reference			reference		
African American	-6.33	12.54	0.62	-0.03	0.21	0.88
Hispanic	-12.83	12.54	0.32	0.06	0.21	0.79
Education (higher education vs. high school)	8.91	9.37	0.35	-12.14	138.94	0.93
Pregnancy BMI ( $\text{kg}/\text{m}^2$ )	-0.61	0.67	0.37	1.12	10.92	0.92
Prepregnancy BMI ( $\text{kg}/\text{m}^2$ )	-0.76	0.64	0.24	-0.004	0.01	0.72
Gestational weight gain (kg)	1.15	0.64	0.08	12.04	10.03	0.24
Actigraphic sleep duration (h)	11.08	4.02	0.01	36.22	68.25	0.60
Self-reported sleep duration (h)	-0.14	2.55	0.96	-20.47	39.36	0.61
Sleep quality	0.63	0.92	0.50	1.18	14.56	0.94
OSA diagnosis	-7.60	12.22	0.54	-162.37	207.58	0.44
AHI (event/h)	-0.36	0.93	0.70	-0.02	.016	0.22
GDM diagnosis	-15.76	11.94	0.20	-4.63	140.34	0.97
Family history of diabetes	0.79	9.51	0.93	0.05	0.14	0.74
WASO (min)	0.003	0.09	0.97	-1.84	1.40	0.20
Shift work	6.89	9.92	0.49	-74.57	155.58	0.63
Maternal fasting glucose (mg/dl)				3.87	3.72	3.01

Abbreviations: BMI, body mass index; WASO, wake after sleep onset.

**TABLE 3** The final model of multiple linear regression analysis for fasting glucose (dependent variable)

Independent variables	Coefficients	Std. error	p value
Evening blue light ( $\mu\text{W}/\text{cm}^2$ )	4.00	1.46	0.01
Gestational weight gain (kg)	0.63	0.55	0.26
Parity	-10.15	8.13	0.22
Actigraphic sleep duration (h)	8.43	3.60	0.03
GDM diagnosis	-11.70	9.70	0.24

**TABLE 4** Final model of multiple linear regression analysis for infant birthweight (dependent variable)

Variables	Coefficients	Std. error	p value
Evening blue light ( $\mu\text{W}/\text{cm}^2$ )	51.80	23.93	0.04
Gestational age at delivery (weeks)	108.63	37.39	0.007
Gestational weight gain (kg)	13.88	10.24	0.18
WASO (min)	-0.96	1.24	0.44
GDM diagnosis	-73.49	120.88	0.55

### Multivariate models investigating associations between blue-light exposure and sleep variables and birthweight

In the multivariate models represented in Table 4, blue-light exposure ( $\beta = 70.38$ ,  $p = 0.01$ ) remained significantly associated with infant birthweight after adjustment for gestational weight gain, gestational age at delivery, GDM diagnosis, and WASO. The coefficient demonstrates that for each unit increase in evening blue-light exposure, there was a 51.80 g increase in infant birthweight (Table 4). In separate

models, we replaced GDM diagnosis with OSA diagnosis or AHI. None of them were significantly associated with infant birthweight (OSA diagnosis:  $\beta = -158.12$ ,  $p = 0.40$ ; AHI:  $\beta = -13.23$ ,  $p = 0.36$ ).

### DISCUSSION

The current study is the first to observe that higher maternal evening blue-light exposure during pregnancy is associated with increased infant birthweight, independent of weight gain during pregnancy, GDM



diagnosis, PSG-diagnosed OSA, and WASO. To our knowledge, this is also the first study to observe the relationship between blue-light exposure and mid- to late-gestation maternal fasting glucose during pregnancy after controlling for weight gain, parity, sleep duration, and GDM diagnosis. In this study, infant birthweight was not associated with other sleep metrics, including maternal objective and self-reported sleep duration, sleep quality, OSA, and WASO. Similarly, these metrics, except for objective sleep duration, were not associated with maternal fasting glucose in linear regression models.

Our findings concerning blue-light exposure and glucose level are compatible with the findings of prior studies in nonpregnant populations in which the association of light exposure, hunger, metabolic function, and physiological arousal was investigated.<sup>8-12</sup> An experimental study of healthy adults aged 20–39 years found that blue-enriched light exposure in the morning and evening, relative to dim light, led to higher insulin resistance. In the evening group of the study, blue-enriched light also resulted in higher peak postprandial glucose compared to dim light.<sup>8</sup> Higher ambient light exposure has also been found to associate with increased prevalence (51.2%) of diabetes.<sup>39</sup> Several factors in relation to light exposure may be involved in glucose metabolism. First, light exposure during the dark cycle can modulate circadian gene expression, shift the timing of food consumption, increase body mass, and reduce glucose tolerance.<sup>10</sup> Second, melatonin may be one of the mediators between blue-light exposure during the nighttime and abnormal metabolism.<sup>40</sup> Light can significantly suppress melatonin production, adding to sleep disturbance. Low urine melatonin levels have been shown to be associated with increased diabetes risk.<sup>41</sup> Evidence in nonpregnant populations suggests that irregularity in melatonin secretion due to personal light exposure may cause circadian misalignment and irregularities in the sleep/wake profile. Third, light exposure impacts cortisol levels,<sup>42</sup> which influence hepatic and peripheral tissue insulin sensitivity and, consequently, circulating glucose levels.<sup>43,44</sup> Fourth, light exposure may stimulate the sympathetic nervous system, which favors higher plasma glucose.<sup>45</sup> Next, poor sleep quality and short sleep duration as seen in shift workers<sup>46</sup> have been found to be associated with insulin resistance and poor glycemic control in patients with type 2 diabetes.<sup>47</sup> Finally, it has been observed in clinical trials that circadian misalignment leads to increased glucose, insulin, and triglyceride levels<sup>16-18</sup> and lowered energy expenditure.<sup>19</sup> This possible mechanism may be involved in the effects of light exposure on glucose metabolism.<sup>8</sup> Future studies with light exposure for successive days and light exposure at different times of day should determine effects on melatonin phase and its possible contribution to any metabolic alterations.

Although low and high birthweight is a risk factor for obesity and chronic metabolic disorders later in life,<sup>48,49</sup> no studies have investigated the relationship between maternal blue-light exposure and infant birthweight. Most studies were performed in nonpregnant subjects. In an experimental study performed on healthy young women, ambient light exposure, such as higher late evening light exposure, reduced melatonin levels and subjective sleepiness and resulted in larger skin temperature gradients as compared to dim.<sup>50</sup> There are indications that circadian misalignment has adverse

cardiometabolic consequences. Likewise, in an animal study, mice housed in either bright or dim light at night had significantly higher body weight and glucose tolerance than those housed in standard light-dark environment.<sup>51</sup> The precise mechanism(s) by which changes in circadian rhythm could affect fetal metabolism remain unknown, but maternal blue-light exposure may result in alterations in maternal glucose tolerance.<sup>8</sup> This may lead to alterations in placental nutrient and glucose transport, which have detrimental outcomes on fetal growth.<sup>45,49</sup> The associations of maternal blue-light exposure with infant birthweight may also be explained by endocrinological events caused by blue-light exposure that may re-entrain the uterus' circadian rhythm and lead to a disturbed rhythm *in utero*. Thus, disturbed maternal circadian rhythm possibly changes the developing environment *in utero*.<sup>52,53</sup> Concerning the lack of association between sleep parameters and fetal growth, our results support those in a large Japanese cohort study in which the relationship between the amount and quality of mothers' sleep and infant birthweight was investigated. Similar to our findings, investigators in this study did not find significant relationships between sleep duration, quality, and infant birthweight.<sup>54</sup>

The current study has strengths and limitations of note. A strength of this study is the evaluation of multiple factors (maternal fasting glucose, sleep, and light) in pregnant women and infant birthweight, and provides new information related to maternal glucose and infant birthweight. Participants were observed for 7 days using subjective (e.g., sleep diaries) and objective (e.g., actigraphs) sleep assessments. The blue-light exposure was recorded objectively prior to infant birthweight measurement. Thus, our finding suggests temporal sequence and a potential association between maternal blue-light exposure and infant birthweight. This can be investigated in future adequately by powered randomized controlled trials. The diagnosis of OSA was also based on PSG, which remains the gold standard for assessing OSA. Clinical data were systematically extracted from medical charts, although maternal fasting glucose was not available for all women. There was a wide variation in the week of fasting glucose measurement. A woman delivered at 33 weeks of pregnancy, but gestation age at delivery was adjusted in the model. The studied population was diverse in terms of ethnicity/race and socioeconomic status, including traditionally underrepresented populations in research studies, such as African Americans and Hispanics. The substantial diversity of the study participants further supports the generalizability of our findings. Finally, the methodology and statistical methods reduced, but could not eliminate, bias. Some limitations should also be taken into account when interpreting the results. The study had a relatively small sample size in this secondary analysis from a larger trial. Seven women with GDM were treated with oral medication and two with insulin. Their fasting glucose values may be an underestimation of their true values. Participants wore an Actiwatch Spectrum on their wrist. Light sensors are often worn on the wrist, which may not directly measure the amount of light entering the eye. Future studies should consider employing wearable sensors measuring light at eye level. Habitual evening light levels in participants' home environment should be also measured. Additionally, actigraphy devices have a tendency to underestimate light intensity.<sup>55</sup> However, currently these devices are only tools that

measure and synchronize light and sleep parameters. Light exposure data were obtained before their bedtime. Thus, it is less likely that their wrist was covered by their blanket.

In conclusion, the results from the current study indicate that evening blue-light exposure is associated with maternal glucose metabolism and the pattern of fetal growth. In this preliminary study, we have found that evening maternal blue-light exposure in the home environment was associated with increased mid- to late-gestation fasting maternal glucose and infant birthweight. The use of LED devices that increase maternal exposure to blue light represents a modifiable behavior that stands to improve maternal sleep patterns. Evening blue-light exposure during mid- and later pregnancy may alter maternal glucose regulation and placental nutrient transport to the fetus, leading to fetal under- or overgrowth patterns. These should be investigated in larger studies. The data reported here may inform future research on the effect of evening light exposure on pregnancy outcomes.

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### AUTHOR CONTRIBUTIONS

B.I.B. contributed to the conception of work, acquisition of data, analysis, and interpretation of data; participated in drafting the manuscript. B.I.B. accepts responsibility for the integrity of the data analyzed. R.H. contributed to scored and analyzed raw actigraphic data; participated in drafting and revising the manuscript. T.L.H. contributed to the analysis and interpretation of data; participated in drafting and revising the manuscript. C.P. and C.B. contributed to interpretation of data; participated in revising the manuscript. All authors approved the final version of the submitted manuscript.

### COMPETING INTERESTS

The authors declare no competing interests.

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