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Sleep in Pregnancy and Maternal Hyperglycemia: a Narrative Review

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Abstract

Purpose of Review Prevalence of gestational diabetes is increasing globally and sleep may be a modifiable lifestyle factor associated with it. However, existing findings have been inconsistent.

Recent Findings Majority of studies reviewed found a link between extreme sleep durations and elevated risk of maternal hyperglycemia. The findings with sleep-disordered breathing are less consistent. Methodological differences across studies, in terms of sleep assessment methods (subjective vs. objective), study population (low vs. high risk), classification of gestational diabetes and sleep problems, may have contributed to the inconsistent findings. Some studies also suggest the possibility of trimester-specific association between sleep and maternal hyperglycemia.

Summary Large-scale prospective studies comprising objective measurements of sleep, preferably over three trimesters and preconception, are needed to better evaluate the relationship between sleep and maternal hyperglycemia.

Keywords Sleep duration · Sleep quality · Sleep-disordered breathing · Pregnancy · Gestational diabetes mellitus

Introduction

There is increasing global health burden of gestational diabetes mellitus (GDM), which is defined as glucose intolerance with onset or first recognition in pregnancy [1, 2]. Maternal hyperglycemia has been linked to adverse perinatal outcomes [3], and the women are at higher risk of developing type 2 diabetes later in life [4]. The offspring are also more likely to be obese and develop type 2 diabetes [5, 6] as well as suffer from other long-term health issues [7]. In order to alleviate the burden of GDM, it is important to identify behavioural risk factors that are modifiable.

Pregnant women often experience a decrease in sleep quantity and quality as a result of anxiety, physical and hormonal changes. As the pregnancy advances, nocturnal awakenings and insomnia tend to increase [8–10] coupled with decrease in sleep duration, quality and efficiency [9, 11, 12]. Pregnant women are also more susceptible to sleep-disordered breathing (SDB), which is a spectrum of abnormal respiratory events such as habitual snoring and obstructive sleep apnea (OSA), attributed to physiological and hormonal changes [13, 14]. Literature suggests that sleep insufficiency and sleep disturbances during pregnancy might be linked to hyperglycemia and GDM [15–20]. Exposure to poor sleep, sleep deprivation or SDB during pregnancy has been shown to associate with elevated markers of oxidative stress and pro-inflammatory cytokines in women with GDM compared with women with normal glucose levels [21–23]. During pregnancy, insufficient sleep or SDB can potentially result in increased sympathetic nervous system activity through changes in heart rate variability [24, 25], which promotes insulin resistance and gluconeogenesis. Another physiological pathway that has been described in some studies is the increase in cortisol secretion

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due to these sleep disturbances or lack of sleep [26, 27]. It has been proposed that chronically elevated cortisol may alter glucose metabolism by suppressing insulin secretion by pancreatic B-cells, subsequently impairing glucose uptake and downstream insulin signalling, thereby enhancing gluconeogenesis [28].

Current findings around the topic of sleep and GDM have been inconsistent, possibly due to the methodological differences across the studies, such as methods of sleep assessment (e.g. self-reported questionnaires, polysomnography, actigraphy) and study population (e.g. sample size, high-risk population). In this review, we aim to critically evaluate the recent literature in the past 5 years on sleep and gestational diabetes and/or maternal hyperglycemia.

Sleep Duration and Gestational Diabetes

Summary of Reviews

Recent reviews have covered studies that examined the relationship between sleep duration during pregnancy and GDM in the past decade (2010–2017). Reutrakul et al. performed a systematic review and meta-analysis to evaluate the relationship between sleep duration and hyperglycemia in pregnancy [29]. Results of the aggregate data analysis revealed that women with short sleep duration (< 6–7 h) during pregnancy, both self-reported and objectively measured, were more likely to have GDM than those without short sleep duration (odds ratio (OR) = 1.70, 95% CI 1.24, 2.33) [29]. In addition, Xu et al. pooled results from prospective and cross-sectional studies and reported that extreme sleep durations during early and middle pregnancy were closely associated with GDM (OR = 1.43, 95% CI 1.16, 1.75) [30]. When she narrowed down to findings from prospective studies, she found both long (≥ 9 h or ≥ 10 h) (OR = 1.28, 95% CI 1.10, 1.49) and short (≤ 4 h, 5–6 h or < 7 h) (OR = 1.58, 95% CI 0.99, 2.52) sleep durations (measured ≤ 22 weeks of gestation) to be associated with increased risk of GDM, although it is only statistically significant with long sleep duration. The wide confidence interval for the pooled OR with short sleep duration could be attributed to the wide confidence intervals for Facco et al. [17] (OR = 11.7, 95% CI 1.20, 114.50) and Qiu et al. [31] (OR = 5.56, 95% CI 1.31, 23.69) studies where the number of women with GDM [17, 31] and short sleep duration (≤ 4 h) [31] was small. A recent narrative review by Gooley et al. examined the association between sleep duration and risk for GDM and showed supportive evidence that both short (≤ 4 h, < 6 h, or < 7 h) and long (≥ 9 h or ≥ 10 h) sleep durations during pregnancy can increase the odds of GDM [32].

Recent Studies

We reviewed studies that were published in the past 5 years that examined the relationship between sleep duration during pregnancy and GDM (Table 1). In line with previous studies, majority of the newer studies using self-reported and/or objective sleep measures also showed that short sleep duration (< 6 h or < 7 h) and/or long sleep duration (≥ 9 h) during pregnancy can influence glucose tolerance [33–35] and they are associated with increased odds of GDM [20, 36, 37]. Some studies however revealed mixed results, possibly due to methodological differences such as gestational age at which the sleep measurement is taken. For example, a study by Wang et al. found no association between self-reported short total sleep duration (< 7 h) (≤ 12 weeks of gestation) with GDM. Instead, they observed a positive association between longer total sleep duration and GDM risk, compared with women with 7 to < 9 h of sleep [37] (Fig. 1). Further, Xu et al. observed no significant associations between both short (< 7 h) and long (≥ 9 h) total sleep duration (reported anytime between enrolment and delivery) and GDM [38], which is consistent with a study by Facco et al. which also observed no significant association between self-reported short nighttime sleep and GDM [39] (Fig. 1). Unlike studies that have shown an influence of sleep duration on glucose tolerance [33–35], Ahmed et al. found no significant differences in blood glucose levels between groups of women who reported getting > 7 h vs. ≤ 7 h of sleep each night [40].

In addition to those studies that only included general population of pregnant women, recent studies have focused on high-risk pregnancies such as pregnant women who were obese or diagnosed with GDM [34, 35, 41••]. In a study that recruited only pregnant women with GDM, Twedt et al. found a significant negative association between nighttime sleep and lower fasting glucose (adjusted $\beta = -2.09$ mg/dL, 95% CI −3.98, −0.20) after adjusting for age, gestational age and BMI. Moreover, those women with extremely short sleep (< 5 h) were associated with significantly higher glucose values at fasting (adjusted $\beta = 15.87$ mg/dL, 95% CI 5.13, 26.61), as well as marked increases in postprandial glucose values (all $p < 0.005$) compared with those women who slept for ≥ 7 to < 9 h at night [35]. Rawal et al. observed in their study that all women (both non-obese and obese) that were exposed to short or long sleep durations showed no association with increased risk for GDM compared with those women who slept 8–9 h per night (Fig. 1). However, when stratified, non-obese women with short (5–6 h or < 7 h) (adjusted RR = 2.52, 95% CI 1.27, 4.99; adjusted RR = 2.01, 95% CI 1.09, 3.68 respectively) and long (≥ 10 h) (adjusted RR = 2.17, 95% CI 1.01, 4.67) sleep durations had increased GDM risk in the second trimester, after adjusting for relevant covariates [41••].

In summary, amongst the studies that looked at sleep duration and GDM risk, the findings were inconsistent. This is

Table 1 Sleep duration and maternal glycemia

Study	Study design	Country	Study population	Glycemia measurement		
				Period (weeks' gestation)	Outcome measured	GDM (*n*)/blood glucose
Herring et al. (2014) [33]	Prospective cohort	USA	*N* = 63 Healthy women	29 (26–32) weeks	GDM[a]	*N* = 7
Twedt et al. (2015) [35]	Prospective cohort	USA	*N* = 37 Women with GDM	28 (6–33) weeks	GDM[a], - GCT[c]	Median fasting glucose = 92 (34–199) mg/dL[a]
Rawal et al. (2017) [41••]	Prospective cohort	USA	*N* = 2581 Obese and non-obese women	After enrolment and before delivery	GDM[a], medical record	*N* = 107
Wang et al. (2017) [37]	Prospective cohort	China	*N* = 12,506 Healthy women	24–28 weeks	GDM[e]	*N* = 919
Cai et al. (2017) [36]	Cross-sectional	Singapore	*N* = 686 Healthy women	24–28 weeks	GDM[f]	*N* = 131
Facco et al. (2017) [20]	Prospective cohort	USA	*N* = 782 Healthy women	After enrolment and before delivery	GDM[b,e,g], medical record	*N* = 33
Xu et al. (2017) [38]	Cross-sectional	China	*N* = 2345 Obese and non-obese women	After enrolment and before delivery	Interview and self-reported questionnaire	*N* = 87
Facco et al. (2018) [39]	Prospective cohort	USA	*N* = 8610 Healthy women	≥ 30 days after delivery	GDM[b,e,h], medical record	Visit 1: *N* = 295 Visit 3: *N* = 304
Ahmed et al. (2018) [40]	Prospective cohort	USA	*N* = 43 Primiparous women	24–28 weeks	OGTT[i]	*N* = 5
Redfern et al. (2019) [34]	Cross-sectional	UK	*N* = 49 Obese women	28 weeks	OGTT[j]	*N* = 10

Table 1 (continued)

Study	Sleep duration			Adjusted covariates	Main findings
	Period (weeks' gestation)	Sleep measure	Classification		
Herring et al. (2014) [33]	21 (18–25) weeks	Actigraphy (6 days)	Continuous night sleep[b]	Maternal age, race/ethnicity, parity, medical insurance (income proxy), education, history of GDM, smoking habits and BMI (first trimester)	Objectively measured sleep duration is inversely correlated with 1-h OGTT values
Twedt et al. (2015) [35]	28 (6–33) weeks	Actigraphy (7 days)	< 5 h ≥ 5–< 7 h ≥ 7–< 9 h[d] ≥ 9 h night sleep	Maternal age, pre-pregnancy BMI and gestational age at enrolment	Increase in sleep duration is associated with reduction in fasting and 1 h postprandial blood glucose
Rawal et al. (2017) [41••]	8–13; 16–22 weeks	Self-reported questionnaire	5–6 h 7 h 8–9 h[d] ≥ 10 h total sleep	Maternal age, gestational age, race/ethnicity, parity, education, pre-pregnancy BMI, marital status, family history of diabetes and napping frequency during corresponding weeks	U-shaped association between sleep duration and GDM. Nap and pre-pregnancy obesity may moderate this association
Wang et al. (2017) [37]	≤ 12 weeks	Self-reported questionnaire	< 7 h 7–9 h[d] ≥ 9 h total sleep	Maternal age, height, family history of diabetes, parity, Han ethnicity, education, BMI and systolic BP at first antenatal care visit, multiple pregnancies, weight gain from pre-pregnancy to glucose challenge test, habitual smoking and alcohol consumption before/during pregnancy	Poor sleep quality and longer duration of sleep are independently associated with an increased risk of GDM
Cai et al. (2017) [36]	26–28 weeks	Self-reported questionnaire	< 6 h ≥ 6 h[d] night sleep	Maternal age, ethnicity, education, BMI at < 14 weeks of gestation, previous history of GDM and STAI total score	Poor sleep quality and short nocturnal sleep duration are independently associated with increased risk of GDM
Facco et al. (2017) [20]	15–22 weeks	Actigraphy (7 days)	< 7 h ≥ 7 h[d] night sleep	Maternal age, BMI, race/ethnicity and employment schedule, self-reported frequent snoring	Short sleep duration and later sleep midpoint are associated with an increased risk of GDM in nulliparous women
Xu et al. (2017) [38]	After enrolment and before delivery	Interview and self-reported questionnaire	< 7 h 7–9 h[d] > 9 h total sleep	None	Sleep duration and sleep quality were not associated with GDM risk
Facco et al. (2018) [39]	Visit 1: 6–13 weeks Visit 3: 22–29 weeks	Self-reported questionnaire; actigraphy (≥ 5 days)	< 7 h ≥ 7 h[d] night sleep	Maternal age and pre-pregnancy BMI, race/ethnicity, employment status and insurance status	Self-reported sleep midpoint, but not sleep duration, in both early and late pregnancy, is associated GDM risk
Ahmed et al. (2018) [40]	22 and 32 weeks	Self-reported questionnaire	> 7 h vs. ≤ 7 h	None	No significant association between sleep duration and blood glucose
Redfern et al. (2019) [34]	End of 2nd trimester	Actigraphy (2–4 days)	Continuous night sleep[b]	Maternal age, BMI and WASO	Shorter night sleep duration is associated with higher 2-h plasma glucose values

BMI body mass index, *GDM* gestational diabetes mellitus, *OGTT* oral glucose tolerance test, *STAI* state-trait anxiety inventory, *WASO* wake after sleep onset

[a] 100-g OGTT; GDM diagnosed when two of four values were abnormal (fasting ≥ 5.3 mmol/L, 1-h ≥ 10.0 mmol/L, 2-h ≥ 8.6 mmol/L, 3-h ≥ 7.8 mmol/L)

[b] Variables reported as continuous values

[c] One-hour 50-g glucose challenge test (1-h ≥ 10.0 mmol/L)

[d] Indicates reference

[e] 75-g OGTT; GDM diagnosed when two of three values were abnormal (fasting ≥ 5.1 mmol/L, 1-h ≥ 10.0 mmol/L, 2-h ≥ 8.5 mmol/)

[f] 75-g OGTT; GDM diagnosed when one value was abnormal (fasting ≥ 7.0 mmol/L, 2-h ≥ 7.8 mmol/L)

[g] One-hour glucose values from non-fasting 50-g OGTT (1-h ≥ 11.1 mmol/L)

[h] One-hour glucose values from 50-g OGTT > 11.1 mmol/L

[i] 75-g OGTT; GDM diagnosed when one value was abnormal (fasting ≥ 92 mg/dL, 1-h ≥ 180 mg/dL, 2-h ≥ 153 mg/dL)

[j] 75-g 2-h OGTT; GDM diagnosed when 75-g glucose: plasma glucose level (1 or more time points need to elevated); fasting 7.0 mmol/L; 2-h 7.8 mmol/L

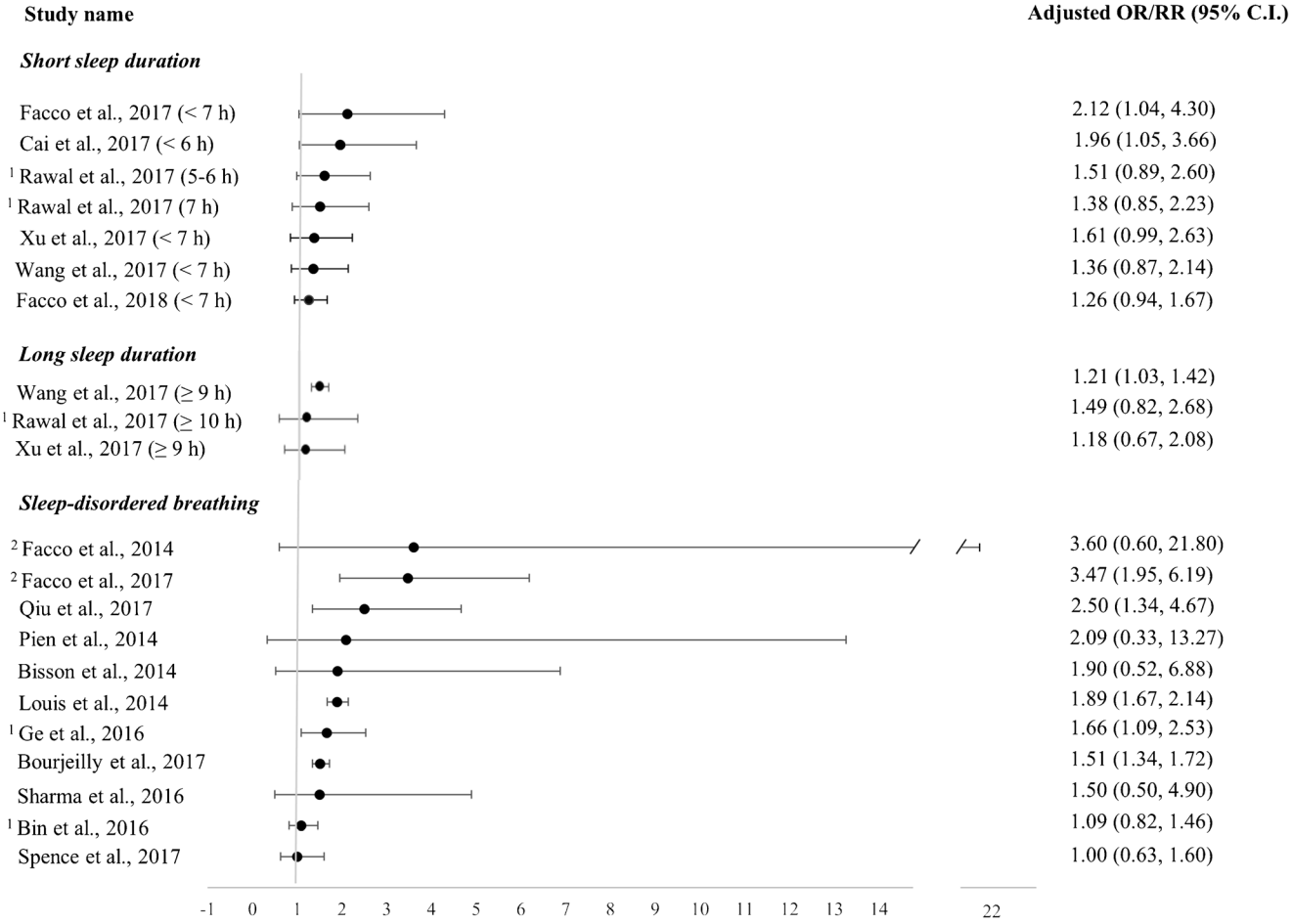

Fig. 1 Forest plot of the associations between GDM and extreme sleep duration as well as sleep-disordered breathing. OR, odds ratio; RR, relative risk; CI, confidence interval; GDM, gestational diabetes mellitus. Results are expressed as ORs with 95% CI, unless otherwise stated. [1]Results expressed as adjusted RR. [2]Results reported for early pregnancy

possibly due to the differences in study populations and the methods of measuring sleep duration. We reviewed six recent papers that studied short sleep duration and GDM risk, of which half observed that short sleep duration is associated with increased GDM risk [20, 36, 41••]. One of these studies only saw an increased GDM risk in non-obese women but not when obese women were included [41••]. The only study to use objectively assessed sleep parameter was the Sleep Duration and Continuity Study [20], while the others used self-reported data [36–39, 41••]. Participants were asked to report total sleep duration for some studies [37, 38, 41••] while others asked specifically about night sleep duration [20, 36, 39]. The definition of short sleep duration was similar (i.e. < 7 h) except for Cai et al. which defined short sleep duration as < 6 h [36] and Rawal et al. which used < 8 h [41••]. Five studies also looked at long sleep duration and risk of GDM [36–39, 41••], and three of them found long sleep duration to be linked to higher GDM risk [37, 39, 41••], although for Rawal et al. this was again only observed amongst the non-obese women [41••]. Cai et al. acknowledged that no association was observed with long sleep duration possibly due to the small number of women ($n = 32$) who reported long night sleep (> 9 h) [36].

It is noteworthy that half of the studies were done in the USA [20, 39, 41••] and the other half in Asian countries (i.e. China and Singapore) [36–38]. Asians have been reported to have higher risk for GDM compared with their Caucasian counterparts [42, 43]. Even amongst the Asian populations studied, there is also heterogeneity. While some studies included a largely homogenous Chinese population [37, 38], Cai et al. included a multiethnic population consisting of Chinese, Malay and Indian. The gestational age at which the sleep duration is assessed is quite varied across the studies (ranging from 6 to 29 weeks), and this may have contributed to the inconsistent findings. It has been reported that it is the sleep duration in the 2nd but not the1st trimester that is significantly associated with GDM risk [41••], suggesting that sleep duration closer to when the GDM diagnosis is made (usually late second trimester) tends to be more predictive.

Four studies only looked at sleep duration and blood glucose levels in pregnant women, either because they had a study population with only participants who had GDM [35]

or the number of GDM cases was too few to make meaningful conclusion on the effect on GDM risk [33, 34, 40]. All but one study showed a negative association between night sleep duration and blood glucose levels, in both the fasting [35] as well as postprandial/postoral glucose tolerance test blood concentrations [33–35]. No similar association was observed with day sleep duration [33]. Ahmed et al. were the only group that did not observe association between night sleep duration and blood glucose levels. In their study, night sleep duration was stratified to < 7 and ≥ 7 h per night [40] and it was not clear how many women were in each category. Given that only n = 43 women were retained in the study, there might be insufficient power, especially given that sleep duration is dichotomized instead of analysed as a continuous variable. It is important to note that most of these studies were done in high-risk study populations, with pregnant women who are obese [34], had GDM [35] or are largely of (over 60%) African American ethnicity [40]. As such, the findings may not be generalizable to other low-risk pregnant population.

Sleep Quality and Gestational Diabetes

A meta-analysis review of previous studies by Sedov and colleagues concluded that some reduction in sleep quality happens during pregnancy [44]. Past reviews of studies that looked at sleep parameters during pregnancy and their association with glucose tolerance did not report significant differences in sleep quality during pregnancy between healthy women and women with hyperglycemia [15, 45]. For example, a review of studies that investigated the influence of sleep disturbances on maternal hyperglycemia, particularly GDM [15], cited two studies that showed no significant differences in sleep quality between those women with GDM, compared with non-GDM women [19, 46].

The studies reviewed all used self-reported information to determine sleep quality, either with the Pittsburgh Sleep Quality Index (PSQI) [36, 40] or a single question to the women to self-rate the sleep quality [37, 38] or whether they suffered from insomnia [47•]. Out of the 4 studies, 3 of them (2 prospective [37, 47•] and 1 cross-sectional [36]) reported association between poor sleep quality and increased risk for GDM. The only study that reported no association between sleep quality and risk of GDM was a cross-sectional study conducted by Xu et al. in China [38]. The study recruited and examined pregnant women from all three trimesters, which might have biased the findings towards the null because the gestational age at which sleep quality is assessed may be important. Zhong et al. observed that poor sleep quality was associated with increased GDM risk in early but not mid-pregnancy [47•]. Similarly, Wang et al. observed a significant effect of first-trimester (≤ 12 weeks) sleep quality on GDM risk. Another study observed an association between mid-pregnancy (26–28 weeks) sleep quality and GDM risk [36]; however, it is a cross-sectional study unlike the two studies mentioned above [37, 47•]. It is also noteworthy that all 4 studies were done in Asians, consisting of majority if not all Chinese women. As such, the findings may not be generalizable to other populations.

Aside from GDM, two other studies also looked at sleep quality and measures of glycemia. Chirwa et al. reported that poorer sleep quality is correlated with higher haemoglobin A1c (HbA1c) [48] while Ahmed et al. did not observe any difference in blood glucose levels between pregnant women with good and poor sleep quality [40]. However, the sample size in the latter is quite small (n = 43 retained in the study).

Sleep-Disordered Breathing and Gestational Diabetes

Summary of Reviews

In the past 5 years, there have been several reviews that support the association of SDB with GDM. In a meta-analysis by Li et al. [49] consisting of 26 studies, they reported that women with SDB had a significant increased risk of GDM (OR = 1.95, 95% CI = 1.60–2.37) compared with women without SDB. Further stratification according to the type of SDB showed that snoring (OR = 2.14, 95% CI = 1.63–2.81) and OSA (OR = 1.71, 95% CI = 1.23–2.38) in pregnant women were both independently associated with an increased risk of GDM. These findings are consistent with a meta-analysis by Liu et al. [50•], who found that pregnant women with OSA were at increased risk of GDM based on the adjusted pooled results (OR = 1.55, 95% CI = 1.26–1.90) from seven studies that objectively measured OSA. Ding et al. classified SDB based on severity and pooled analyses revealed that moderate-severe SDB was significantly associated with GDM (OR = 1.78, 95% CI = 1.29–2.46) [51]. However, after further stratification by country, study design and presence of data adjustment, it was found that the association was not significant in studies with Asian populations (OR = 1.17, 95% CI = 0.42–3.27), prospective study design (OR = 1.20, 95% CI = 0.93–1.53) and unadjusted data (OR = 1.38, 95% CI = 0.89–2.14). The authors cautioned they were unable to address the lack of precision by which important confounders such as smoking and body mass index were measured in most of the studies included in the review. This was a similar limitation faced by Pamidi and colleagues [52] in their own meta-analysis, where obesity (a strong risk factor for SDB [53]) was controlled for in varying degrees across the five observational studies that were pooled. While Pamidi et al. found SDB to be

significantly associated with GDM (OR = 1.86; 95% CI = 1.30–2.42), they believed that the overall effect estimates may have been affected by the variability in definitions and assessments of SDB as well as the different study populations across the pooled studies. Another meta-analysis, which only included cohort studies ($n = 5$) (4 prospective and 1 retrospective), provided contrasting findings from those discussed above, with no significant difference in risk of GDM between pregnant women with and without OSA (RR = 1.40, 95% CI = 0.62–3.19) [54].

An overarching view of the literature thus far has produced mixed findings on the relationship between SDB and GDM, as corroborated in a recent review by Gooley et al. [32]. Several of the meta-analyses reviewed by Gooley and colleagues have already been described above [51, 52, 54], although they also included an earlier meta-analysis by Luque-Fernandez et al. [55] suggesting that SDB during pregnancy augments GDM risk. All in all, they noted that of the studies included that controlled for BMI or obesity, only about half suggested an association between SDB and GDM, possibly attributed to variability in the way SDB was assessed.

Recent Studies

Although reviews in the last 5 years have generally pointed to an association between SDB and GDM, this relation has not always been incontrovertible. Indeed, 12 individual studies published over the last 5 years have provided mixed findings (Table 2, Fig. 1). There are four population-based studies that looked at the association of OSA and GDM in large numbers of pregnant women [56–59], but the findings were not consistent. Two of the studies found significant association between OSA and increased likelihood of GDM [57, 58] while the other two did not [56, 59]. The study by Spence et al. involved women who delivered in the Department of Defense hospitals under the Military Health System [59]. Their study was on a military population made up of a significant proportion of women that are in active duty, with reasonable level of physical fitness and body weight. The other women are spouses of active duty members. As such, the findings may not be generalizable to the civilian population. The diagnoses of GDM and OSA in these population studies were based on medical records, classified by the International Classification of Diseases (ICD) 9th or 10th edition or a modified version of the 9th edition (ICD-9-CM). There is a potential of variability in diagnosis, especially for OSA where we cannot be sure if all the diagnoses were based on standard diagnostic criteria.

Overnight polysomnography (PSG) has been the gold standard for the diagnosis of OSA [60]. Five studies used some form of objective measurement to diagnose OSA. Three of them used PSG [61, 62•, 63] while the other two used the Watch-PAT [64, 65•] which has been validated against PSG in pregnancy [66]. Both studies by Facco et al. suggest that severity of SDB may influence the association with GDM [62•, 64]. In the earlier study, she demonstrated in a group of women at high risk for pre-eclampsia (e.g. obese, twin gestation, with pre-gestational diabetes) that moderate to severe SDB was associated with increased risk of GDM, although it did not reach statistical significance due to the relatively modest sample size [64]. She went on to show in the Nulliparous Pregnancy Outcomes Study: Monitoring Mothers-to-Be parent study a dose response between apnea-hypopnea index (AHI) and GDM risk [62•]. This may explain why Bisson et al. had null findings with SDB and GDM, as there is only one subject with AHI ≥ 15 in the GDM group [61]. Meanwhile, Pien et al. acknowledged that the small number of participants who developed GDM ($n = 5$) may have resulted in lack of statistical power to observe any association with OSA [63]. Separately, Wanitcharoenkul et al. studied 82 obese women with GDM and found that severity of OSA to be positively correlated with fasting blood glucose levels, but not haemoglobin A1c (HbA1c) [65•]. Amongst the women with OSA, increasing severity of OSA, marked by hypoxia-related parameters such as oxygen desaturation index (ODI) and minimum oxygen saturation level (Min O_2), was shown to correlate positively with higher insulin resistance (ODI, $p = 0.037$; Min O_2, $p = 0.040$) and severe B-cell dysfunction (ODI, $p = 0.021$; Min O_2, $p = 0.009$), thereby supporting the detrimental metabolic effects of OSA in pregnant women with GDM [65•].

The other studies reviewed used only questionnaires to self-report on SDB [67–69], which may be subjected to recall bias and inaccuracy in reporting, especially since some of the symptoms such as daytime sleepiness and fatigue are also common pregnancy complaints [70]. One advantage of self-reported data is that it is cost-effective and relatively easy to collect and can be collected repeatedly over the course of the pregnancy. Interestingly, Ge et al. found that it is chronic snoring (i.e. habitual snoring before and during pregnancy) and not pregnancy onset snoring that significantly increased GDM risk [67]. Unlike sleep duration and sleep quality, the gestational age when SDB is assessed seems to make little difference on the link between SDB and GDM risk [62•, 64, 67]. Sharma et al. collected information on SDB and other sleep disorders before pregnancy (based on recall) and for every trimester; however, the information was collated as the number of times each sleep disorder occurred before delivery and unable

Table 2 Sleep-disordered breathing and maternal glycemia

Study	Study design	Country	Study population	Glycemia measurement		
				Period (weeks' gestation)	Outcome measured	GDM (*n*)
Pien et al. (2014) [63]	Prospective cohort study	USA	*N* = 105 Healthy women	Third trimester	GDM	*N* = 5
Bisson et al. (2014) [61]	Case-control study	Canada	*N* = 52 (26 matched pairs)	24–28 weeks	GDM (OGTT[b])	*N* = 26
Louis et al. (2014) [58]	Retrospective cross-sectional study	USA	*N* = 55,781,965 General population	Not specified	GDM (Database: Nationwide Inpatient Sample, 1998–2009[c])	*N* = 2,520,604
Facco et al. (2014) [64]	Prospective cohort study	USA	*N* = 182 Women at high risk of pre-eclampsia	Not specified	GDM (Medical records)	*N* = 27
Ge et al. (2016) [67]	Prospective cohort study	China	*N* = 3079 Healthy women	≥ 20 weeks	GDM (Medical records)	*N* = 386
Sharma et al. (2016) [69]	Prospective observational study	India	*N* = 209 Women with symptom-diagnosed OSA and other sleep disorders	Delivery	GDM (OGTT[d])	*N* = 40
Bin et al. (2016) [56]	Population-based cohort study	Australia	*N* = 636,227 Healthy women	Not specified	GDM (Medical records, 2002–2012[e])	*N* = 39,601
Facco et al. (2017) [62•]	Prospective cohort study	USA	*N* = 3245 Women without pre-gestational diabetes	27 weeks	GDM (GTT[f] and chart abstraction)	*N* = 134
Bourjeilly et al. (2017) [57]	Cohort study	USA	N = 1,577,632 General population	Not specified	GDM (Delivery discharge record[g])	*N* = 410,184
Qiu et al. (2017) [68]	Prospective cohort study	USA	*N* = 1579 Healthy women	24–28 weeks	GDM (OGTT[h])	*N* = 100
Wanitcharoenkul et al. (2017) [65•]	Prospective cohort study	Thailand	*N* = 82 Obese women with diet-controlled GDM	24–34 weeks	HOMA-IR[a] Insulinogenic index[a] Matsuda index[a] Disposition index[a]	Diagnosis before 24 weeks by FPG = 49 Diagnosis after 24 weeks by OGTT = 33
Spence et al. (2017) [59]	Retrospective cohort study	USA	*N* = 305,001 Women	Not specified	GDM (Military Health System database[g])	*N* = 28,676

Table 2 (continued)

Study	Sleep-disordered breathing			Adjusted covariates	Main findings
	Period (weeks' gestation)	Sleep measure	Classification		
Pien et al. (2014) [63]	First trimester (median, 12.1 weeks) Third trimester (median, 33.6 weeks)	AHI measured by PSG[a]	Presence of OSA (AHI ≥ 5) Difference in AHI between first and third trimesters	BMI, maternal age	No significant association between SDB and GDM risk
Bisson et al. (2014) [61]	24–32 weeks	Measurements by PSG: - AHI - ODI[a] - Arousal index[a] - Percentage of sleep time spent with inspiratory flow limitation[a] Self- and partner/roommate-reported questionnaire	Presence of OSA based on two different criteria (AHI ≥ 15, AHI ≥ 5) Snoring before pregnancy Presence of loud snoring	Participants in case and control conditions were matched for gestational age at PSG, age and BMI.	Amongst pregnant women with pre-pregnancy BMI < 35 and no medical comorbidities; there is no significant association between SDB and GDM
Louis et al. (2014) [58]	Not specified	Database: Nationwide Inpatient Sample, 1998–2009[c]	OSA diagnosis[c]	Maternal age, ethnicity, household income, multiple gestation, tobacco, alcohol and drug use, primary payer, rural/urban status, obesity, composite variable coding for coronary heart disease, chronic renal disease, anaemia, lipid metabolism, hypothyroidism and disorders of the adrenal glands	Pregnant women with OSA are at increased risk for GDM.
Facco et al. (2014) [64]	6–20 weeks 28–37 weeks	AHI measured by Watch-PAT100	SDB: AHI ≥ 5 None: AHI = 0 Minimal: AHI < 5 Mild: 5 ≤ AHI < 15 Moderate: 15 ≤ AHI ≤ 29.9 Severe: AHI ≥ 30	Pre-pregnancy BMI, maternal age, race, parity, chronic hypertension, twin gestation	Moderate to severe SDB are associated with non-statistically significant increase in risk of GDM
Ge et al. (2016) [67]	First trimester Third trimester	Snoring measured by self-report	Habitual snoring (snoring at least 3–4 times per week) Pregnancy onset habitual snoring Chronic snoring (snored before and during pregnancy)	Pre-pregnancy BMI, maternal age, educational level, gravidity, maternal smoking, only child or not	Chronic snoring is independently associated with GDM risk
Sharma et al. (2016) [69]	Before pregnancy At all three trimesters	Self-reported questionnaires	Snoring frequency High risk of OSA as screened by MBQ	Not specified	No significant association between GDM and snoring in pregnant women with OSA and other sleep disorders
Bin et al. (2016) [56]	Presence of sleep apnea in year before or during pregnancy	Medical records	OSA diagnosis [e]	Maternal age, country of birth, socioeconomic disadvantage, smoking, obesity	Women with sleep apnea were not more likely to have GDM than women without sleep apnea
Facco et al. (2017) [62•]	Early pregnancy (6–15 weeks) Mid-pregnancy (22–31 weeks)	AHI measured by PSG	SDB: AHI ≥ 5 None: AHI = 0 Minimal: AHI < 5 Mild: 5 ≤ AHI < 15 Moderate-severe: AHI ≥ 15	Age, BMI, chronic hypertension, rate of weight gain per week between early and mid-pregnancy assessments, race, smoking	SDB in early and mid-pregnancy is associated with increased risk of GDM. Exposure-response relationship observed with AHI and GDM

Table 2 (continued)

Study	Sleep-disordered breathing			Adjusted covariates	Main findings
	Period (weeks' gestation)	Sleep measure	Classification		
Bourjeilly et al. (2017) [57]	Not specified	Delivery discharge record	OSA diagnosis [g]	Maternal obesity, pre-pregnancy hypertension, pre-pregnancy diabetes, maternal age, ethnicity, multiple birth, tobacco use, alcohol/drug use, rural/urban status, coronary heart disease, anaemia, hyperlipidemia, hypothyroidism and adrenal gland disorders	Elevated risk of GDM amongst women with OSA compared with those without OSA
Qiu et al. (2017) [68]	Median, 15.1 weeks	Self-reported questionnaire	Snoring frequency	Maternal age, race, smoking during pregnancy, family history of diabetes mellitus, pre-pregnancy BMI	Women who reported snoring most of the time are at higher risk of IGT and GDM, especially in overweight women
Wanitcharoenkul et al. (2017) [65•]	Median, 29 weeks	Measurements by Watch-PAT 200: - AHI - ODI[a] - T90[a] - Min O_2[a]	Presence of OSA (AHI ≥ 5)	Pre-pregnancy BMI	OSA severity, particularly the degree of oxygen desaturation, correlated with FPG, insulin resistance and B-cell function.
Spence et al. (2017) [59]	Not specified	Military Health System database[g]	OSA diagnosis[g]	Maternal age, ethnicity, hospital characteristics, obesity and clinical conditions or comorbidities associated with OSA (coronary heart disease, chronic hypertension, adrenal disorders, hypothyroidism, lipid metabolism disorder, anaemia). Women with pre-existing diabetes were excluded from analysis	No significant association between OSA and GDM

BMI body mass index, *GDM* gestational diabetes mellitus, *OGTT* oral glucose tolerance test, *AHI* apnea-hypopnea index, *PSG* polysomnography, *OSA* obstructive sleep apnea, *SDB* sleep-disordered breathing, *FPG* fasting plasma, *IGT* impaired glucose tolerance, glucose, *MBQ* Modified Berlin Questionnaire, *NSW* New South Wales, *HOMA-IR* Homeostatic Model Assessment of Insulin Resistance, *ODI* oxygen desaturation index, *T90* percentage of total sleep time in which oxygen saturation remains < 90%, *Min* O_2 lowest oxygen saturation value over the recording period

[a] Variables reported as continuous values

[b] 75 g OGTT: GDM diagnosed when two or more glucose levels were abnormal (fasting ≥ 95.5 mg/dL, 1-h ≥ 191.0 mg/dL, 2-h ≥ 160.4 mg/dL)

[c] International Classification of Diseases, 9th edition, Clinical Modification (ICD-9-CM)

[d] 75 g OGTT: GDM diagnosed when one or more glucose levels are exceeded (fasting ≥ 92 mg/dL; 1-h ≥ 180 mg/dL; 2-h ≥ 153 mg/dL)

[e] International Statistical Classification of Diseases and Related Health Problems, 10th Revision (ICD-10-AM)

[f] GDM defined by one of the following glucose tolerance testing (GTT) criteria: (1) fasting 3-h 100 g GTT with two abnormal values: fasting ≥ 95 mg/dL, 1-h ≥ 180 mg/dL, 2-h ≥ 155 mg/dL, 3-h ≥ 140 mg/dL; (2) fasting 2-h 75 g GTT with one abnormal value: fasting ≥ 92 mg/dL, 1-h ≥ 180 mg/dL, 2-h ≥ 153 mg/dL; (3) non-fasting 50 g GTT ≥ 200 mg/dL if no fasting 3-h or 2-h GTT was performed

[g] International Classification of Diseases, 9th edition (ICD-9)

[h] 100 g OGTT: GDM diagnosed when two or more glucose values were abnormal (fasting ≥ 95 mg/dL; 1-h ≥ 180 mg/dL; 2-h ≥ 155 mg/dL; 3-h ≥ 140 mg/dL)

[i] FPG: GDM diagnosed when glucose levels ≥ 92 mg/dL

to give more granularity as to which period was most critical for SDB to exude an effect on GDM [69].

Other Sleep Parameters

Recent studies have also explored other estimates of sleep parameters such as the midpoint of nocturnal sleep, wake after sleep onset (WASO), sleepiness, restless leg syndrome and frequency of napping. For example, studies by Facco et al. have observed a positive association between self-reported late sleep midpoint (> 5:00 am) and increased odds of GDM, in both early and late pregnancy (visit 1 adjusted OR = 1.67, 95% CI 1.17, 2.38; visit 2 adjusted OR = 1.73, 95% CI 1.23, 2.43) [39], even after adjusting separately for BMI, maternal age, ethnicity and snoring (all $p < 0.05$) [20]. The authors agreed based on concordance between daily diary and actigraphy data that self-reported sleep midpoint ascertainment may be more accurate than self-reported sleep duration when assessing the relationship between sleep during pregnancy and GDM [39]. Meanwhile, two studies showed no association of WASO during pregnancy in relation to plasma glucose values ($\beta = -0.04$, 95% CI −0.131, −0.103, $p = 0.809$) [34] or GDM (OR = 1.32, 95% CI 0.62, 2.62, $p = 0.479$) [20]. Using objective measures of sleep, Herring et al. did not observe any association of nap frequency with glucose intolerance in pregnancy [33]. Separately, a case-control study looked into the association between self-reported daytime sleepiness (by the Epworth Sleepiness Scale) and restless leg syndrome with GDM risk [61]. The authors found that daytime sleepiness was greater in women with GDM than in those without GDM (9.8 ± 3.6 vs. 7.2 ± 3.6, $p = 0.05$), and prevalence of restless leg syndrome in women with GDM was significantly greater than those without GDM (46% vs. 19%, $p = 0.07$). Along this line, future research should explore new reliable sleep measures to assess both quantity and quality of sleep in pregnant women.

Conclusion

Most studies support that extreme sleep duration is associated with elevated risk for maternal hyperglycemia. The findings with sleep-disordered breathing are less consistent. From our review, we noticed that gestational age of sleep assessment may have contributed to the inconsistency, on top of the common methodological differences like subjective vs. objective sleep assessment and type of study population (i.e. low vs. high risk). Sleep duration closer to when the GDM diagnosis is made (usually late second trimester) tends to be more predictive, while sleep quality in early, rather than middle/late pregnancy, seems to be more predictive. Presence of SDB preconception (e.g. chronic snoring) may also be more predictive than pregnancy onset SDB (e.g. pregnancy onset snoring). Severity of SDB is also a consideration, with moderate/severe SDB more likely to be associated to GDM compared with mild cases. Hence, stratification of SDB severity may be necessary for a clearer picture of the link between SDB and GDM, instead of lumping mild cases together with moderate/severe cases. Large-scale prospective studies comprising objective measurements of sleep problems, preferably over three trimesters and preconception if possible, are needed to better evaluate the relationship between sleep and GDM. If a causal relationship is confirmed, behavioural therapies for improving sleep can be tested out in randomised controlled trials to potentially reduce risk and burden of GDM.

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Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

Human and Animal Rights and Informed Consent All reported studies/experiments with human or animal subjects performed by the authors have been previously published and complied with all applicable ethical standards (including the Helsinki declaration and its amendments, institutional/national research committee standards and international/national/institutional guidelines).

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- • Of importance
- •• Of major importance

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