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CLINICAL REVIEW

Maternal sleep during pregnancy and poor fetal outcomes: A scoping review of the literature with meta-analysis

Jane Warland [a, *], Jillian Dorrian [b], Janna L. Morrison [c], Louise M. O'Brien [d]

[a] Mother's Babies and Families Research Group, School of Nursing and Midwifery, University of South Australia, Adelaide, 5001, SA, Australia
[b] Behaviour-Brain-Body Research Centre, School of Psychology, Social Work and Social Policy, University of South Australia, Adelaide, SA, 5001, Australia
[c] Early Origins of Adult Health Research Group, School of Pharmacy & Medical Sciences, Sansom Institute for Health Research, University of South Australia, Adelaide, SA, 5001, Australia
[d] Sleep Disorders Center and Department of Obstetrics & Gynecology, University of Michigan, Ann Arbor, MI, USA

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SUMMARY

There is a wealth of evidence to say that sleep impacts maternal health during pregnancy, however, little has been published on fetal health and maternal sleep. This scoping review summarises current literature on maternal sleep including sleep disordered breathing, sleep quality, sleep duration and supine sleep position, as these relate to fetal outcomes specifically birth weight, growth, preterm birth and stillbirth.

An overall interpretation of the studies evaluated shows that events occurring during maternal sleep such as obstructive sleep apnea, sleep disruption and sleep position may have a negative effect on the fetus resulting in altered growth, gestational length and even death. These effects are biologically and physically plausible.

In conclusion, there is limited and often conflicting information on maternal sleep and fetal outcomes. However, existing evidence suggests that this is an important area for future research. This area is ripe for investigation if there is to be reduction in the physical, emotional, and financial burden of poor fetal outcomes related to maternal sleep.

Introduction

All adult humans sleep for approximately one third of their lives, thus the fetus is exposed to a mother who is asleep for one third of their gestation. In recent years, the relationship between sleep and pregnancy outcomes has become an area of intense research interest, which has thus far resulted in several recent systematic reviews/meta-analyses [1–5]. There has also been other informative work in the area of sleep and pregnancy that was either published after these reviews or was beyond their scope. Of note is, that none of the reviews have specifically focused on fetal outcomes, but rather have examined the associations between maternal sleep and maternal conditions such as pre-eclampsia, gestational diabetes, or outcomes such as length of labour or later development of maternal depression. Whilst each of these maternal conditions can have an effect on the fetus, and an increasing number of studies have reported fetal outcomes associated with maternal sleep practises, it is now an opportune time to review the current literature regarding the impact of maternal sleep on fetal outcomes, namely fetal growth, prematurity, and/or stillbirth.

Review objective

The objective of this scoping review was to collect, evaluate, and present the available research evidence that has investigated the impact of maternal sleep on fetal outcomes. The primary purpose of the review was to "map the field" [6] of current knowledge of association between maternal sleep and fetal outcomes with the view to identifying what is currently known about this important area.

Method

This scoping review follows the five-stage scoping review framework suggested by Arksey and O'Malley [6] namely to identify and review all relevant literature regardless of study design.

* Corresponding author.
E-mail address: jane.warland@unisa.edu.au (J. Warland).

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Abbreviations used

BMI	Body mass index
BQ	Berlin Questionnaire
FGR	Fetal growth restriction
IUGR	Intrauterine growth restriction
LGA	Large for gestational age
LBW	Low birth weight
OSA	Obstructive sleep apnea
PAR	Population attributable risk
PSQI	Pittsburgh sleep quality index
PSG	Polysomnography
PTB	Preterm birth
SB	Stillbirth
SDB	Sleep disordered breathing
SGA	Small for gestational age

Stage 1: Identifying the research question

The scoping review question was: What is known from the existing literature about maternal sleep in pregnancy and fetal outcomes?

Stage 2: Identifying relevant studies

Articles were chosen for inclusion in this review by searching the Medline and Embase databases. The fetal outcomes of interest were fetal weight, fetal growth, preterm birth (PTB), gestational age or stillbirth. We did not define any of these terms, as there is wide variation between definitions used in the literature, we therefore considered studies for inclusion if the authors used any of the following terms and word combinations in their publications: (sleep*) AND (pregnan*) AND ((prematur*, premmie, preterm) OR (growth restrict* OR low birth weight OR small for gestational age OR IUGR OR FGR) OR stillbirth OR stillborn OR fetal demise)) in all applicable combinations. Cross-references from articles found, conference proceedings, and bibliographies from review articles and book chapters were also examined for appropriate material. All studies published prior to the end of November 2017 were included.

Stage 3: Study selection

Our initial perusal of the identified citations indicated that the search strategy had picked up a large number of irrelevant studies. These were generally related to fetal sleep and/or infant sleep. Our primary focus was to comprehensively summarise the current literature on maternal sleep and fetal outcomes. Thus, we excluded results that were neonatal studies (nursery admission, Apgar scores), infant sleep, early pregnancy loss (prior to 28 weeks), animal studies, or secondary findings such as literature reviews. Studies were included in our review if they were (i) primary research (ii) published in English, and (iii) had a focus exploring an aspect of maternal sleep in pregnancy and fetal outcome. The fetal outcomes of interest were when the authors of the primary sleep study reviewed reported: fetal weight, fetal growth, preterm birth, gestational age or stillbirth.

Stage 4: Charting the data

Each article reviewed was organised by sleep parameter and tabulated by source (Author, year, country), study design/method, sample size, and summary of key fetal outcome findings (see Tables 1–5). Where it was possible, a meta-analysis was conducted and Forrest plots created to show these results alongside the relevant table.

Stage 5: Collating, summarising and reporting the results

Using the above search strategy 796 articles were identified from all sources with 418 non-duplicated. Of these, 153 were deemed relevant by reading the title and abstract and copies of the full article were obtained. Each article was reviewed by JW and LMO and consensus was reached for the 65 articles included in the review (Fig. 1)

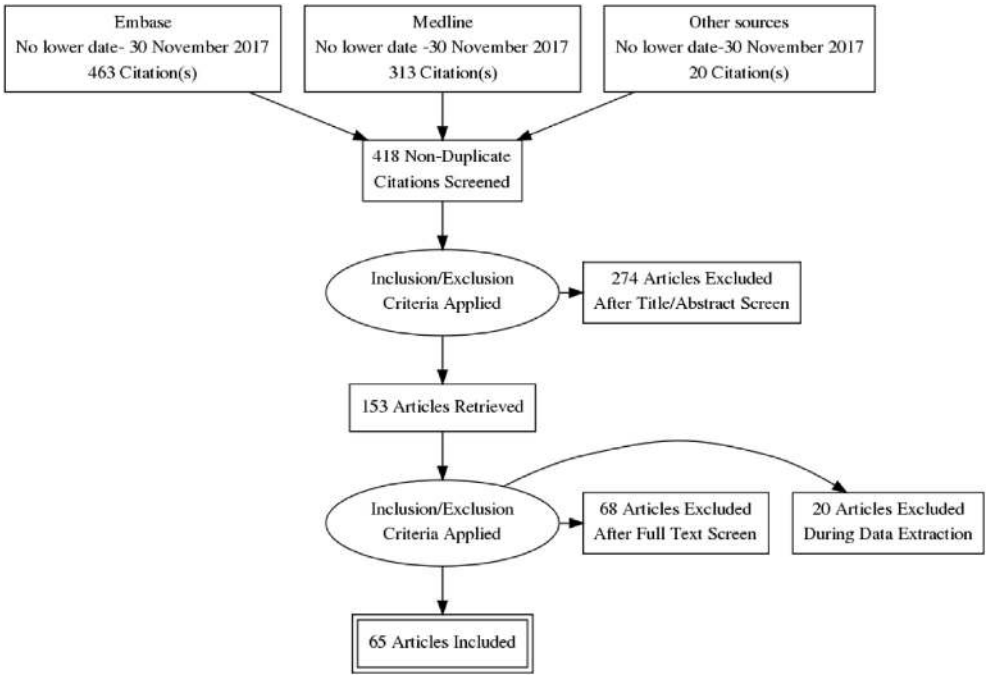

Fig. 1. Search strategy.

Table 1
Subjective measures of SDB and fetal outcomes.

Authors, reference number Country	Design/Methods	Sample size	Fetal outcomes		
			Weight/Growth	PTB	SB
Antony et al. 2014 [37] USA	Prospective cohort/BQ	n = 1153 (n = 178 BQ positive)	BW not reported. Positive BQ not associated with SGA but was associated with LGA>90th centile (aRR 2.19, 95%CI 1.3–3.6) and LGA>95th centile (aRR 2.56, 95%CI 1.4–4.8); frequent snoring associated with LGA>95th centile (aRR 2.2, 95%CI 1.1–4.3)	Positive BQ associated with PTB (18.5% vs. 11.7%, p = 0.01) unadjusted, no increased RR after adjustment	Not investigated
Ayrim et al. 2011 [15] Turkey	Cross sectional (recruited in labour)/Snoring Question	n = 200 (n = 42 snorers)	No difference in BW. Growth not investigated	Increase in PTB	Not investigated
Bourjeily et al. 2010 [35] USA	Cross sectional (recruited 24–48 h postpartum)/MAP Index	n = 1000 (35% frequent/always snoring)	BW not reported. Trend towards association with snoring and growth restriction but not enough data to perform adjustments	Gasping associated with increased PTB, (aOR 1.8, 95% CI 1.1–3.2)	Not investigated
Franklin et al. 2010 [13] Sweden	Cross sectional (recruited on day of delivery, only vaginal deliveries)/ Snoring question	n = 502 (n = 113 habitual snorers)	BW not reported. Snorers more likely to have IUGR (aOR 3.5, 95%CI 1.3–9.4). Witnessed apneas did not have an effect on infant outcome	No difference in gestational length; PTB not reported	SB excluded
Ge et al. 2016 [20] China	Prospective cohort (from a birth cohort study)/Snoring question	n = 3474 at 1st trimester (n = 361 pregnancy onset and n = 150 chronic snorers)	Mean BW not reported; no difference in LBW. Pregnancy onset snoring associated with macrosomia (RR 1.5, 95%CI 1.1–2.3) and LGA (RR 1.7, 95%CI 1.3–2.2).	No difference in PTB with snoring. Witnessed apneas associated with PTB (aRR 2.6, 95%CI 1.2–5.2)	Not investigated as excluded from analysis (n = 10)
Gordon et al. 2015 [45] Australia	Population-based matched case –control/BQ	n = 295 (n = 103 stillbirth and n = 192 live birth)	BW not investigated. Growth not investigated as regards snoring	Not investigated as regards snoring	No difference in snoring in SB vs. live births
Guilleminault et al. 2000 [25] Not stated.	Prospective cohort/Snoring question	n = 267 (n = 10 chronic, loud snorers)	No difference in BW although a trend was observed for infants of chronic snorers to be born with lower BW. Growth not investigated	NA as none were <37/40	Not investigated
Higgins et al. 2011 [26] USA	Prospective cohort/BQ	n = 4074 (n = 1343 BQ positive)	Mean BW higher in positive BQ. Growth not investigated	Not investigated	Not investigated
Howe et al. 2015 [33] New Zealand	Prospective cohort/Snoring question	n = 633 (n = 194 Maori women but no stratification regarding snoring)	BW not reported. No differences in growth for chronic or pregnancy onset snoring. Weak association between breathing pauses and SGA<10th (OR 2.8 95%CI 0.9–9.0). Association between LGA and pregnancy onset breathing pauses (aOR 3.4, 95%CI 1.3–9.6) after adjustment for BMI	Not investigated (PTB excluded)	Not investigated
Ko et al. 2013 [27] Korea	Prospective cohort/BQ	n = 276 (n = 89 BQ positive)	BW slightly higher in positive BQ (p = 0.05, unadjusted) and only in those with BMI<30). No differences in SGA<10th	No difference	Not investigated
Koken et al. 2007 [16] Turkey	Case-control/Snoring question	n = 83 (n = 40 snorers)	No differences in BW or growth	No difference	Not investigated
Leung et al. 2005 [28] China	Prospective cohort/Snoring question	n = 195 (outcome data available for n = 180 women,n = 81 of which were snorers)	No difference in BW. Growth not investigated	Mean gestational age not different but PTB not reported	Not investigated

(*continued on next page*)

Table 1 (*continued*)

Authors, reference number Country	Design/Methods	Sample size	Fetal outcomes		
			Weight/Growth	PTB	SB
Loube et al. 1996 [29] USA	Prospective cohort (low risk women only)/Snoring question	n = 350 (n = 49 frequent snorers)	No differences in BW or growth	No difference in a composite of "complications" but calculations of raw data shows 7/49 PTB (14.3%) in snoring group vs. 5/296 (1.7%) controls (p < 0.001)	Not investigated
Micheli et al. 2011 [17] Greece	Prospective cohort/Snoring question	n = 1091 (n = 199 snorers of which n = 48 were severe snorers)	Increase in LBW, aRR 2.6 (1.2−5.4) in severe snorers compared to non-snorers. Increased FGR <10th centile RR 2.0 (95%CI 1.0−3.8) in severe snorers	No difference	Not investigated
Na-Rungsri et al. 2016 [42] Thailand	Prospective cohort/BQ	n = 1345 (n = 136 BQ positive)	BW not investigated; Growth not investigated	Positive BQ higher PTB (aOR 2.0, 95%CI 1.2 −3.3). BQ positive associated with increased risk of spontaneous PTB (aOR 2.5, 95%CI 1.2 −5.0). No clear evidence of association of BQ with preterm premature rupture of membranes or medically indicated PTB	Not investigated
O'Brien et al. 2013 [30] USA	Prospective cohort/Snoring question	n = 1673 (n = 151 chronic snoring and n = 435 pregnancy onset snoring)	No difference in BW. Chronic snorers more likely to have SGA<10th (aOR 1.7, 95%CI 1.0 −2.7); self reported apnea associated with larger birth centile and LGA >90th (unadjusted) but after adjustment LGA explained by diabetes	Not investigated	Not investigated
Olivarez et al. 2011 [31] USA	Prospective cohort/BQ	n = 220 (n = 56 BQ positive)	BW bigger in BQ positive group (p = 0.003) but when stratified by obesity this finding only held for those in the BMI≥30 group. In multivariate analysis BQ positive almost associated with increased BW in obese women (p = 0.06) but not in non-obese. No difference in SGA aOR 0.99 (95%CI 0.68 −1.46).	Not reported	Not investigated
Owusu et al. 2012 [18] Ghana	Cross sectional (recruited within 48 h of delivery)/Snoring question	n = 220 (n = 53 snorers)	No difference in BW. Growth not investigated	No difference	Not investigated
Pamidi et al. 2016 [36] Canada	Prospective cohort (part of a larger network study)/Snoring question	n = 182 in 1st trimester (n = 54 any snoring and/or witnessed apneas). There was a 35% incidence of new symptoms of SDB from the first trimester to the third trimester.	BW not investigated. No difference in SGA <5th between "any snoring" in 1st or 3rd trimester. Pregnancy onset snoring almost significant (OR 3. 8 95% CI 0.8 to 17.0, p = 0.08)	Not investigated	Not investigated
Perez-Chada et al. 2007 [19] Argentina	Cross sectional (recruited on day of delivery)/Snoring question	n = 447 (n = 32 habitual snorers)	No difference in BW between "any snoring" and non-snoring. No difference in growth between "any snoring" and non-snoring	PTB not investigated but GA almost shorter in "any snorers" vs. non-snorers (p = 0.069)	Not investigated
Salihu et al. 2015 [24] USA	Cross sectional (recruited on day of delivery)/BQ	n = 67 (n = 21 BQ positive)	No difference in BW. Growth not investigated	Not investigated (PTB excluded)	Not investigated
Sarberg et al. 2014 [34] Sweden	Prospective cohort/Snoring question	n = 340 (n = 27 chronic snorers and n = 45 pregnancy onset snorers)	BW not reported. Very few SGA (n = 3, 1.1% in non-snorers and n = 2, 4.4% in snorers, p = NS)	Very few PTB (n = 6, 2.2% in non-snorers and n = 3, 4.4%, in snorers, p = NS); no difference in gestational age although women had to reach 3rd trimester to be included.	Not investigated
Sharma et al. 2016 [23] India	Prospective cohort (high risk pregnant women)/modified BQ	n = 273 (n = 18 frequent snorers)	BW almost significant in those who snored at 3 time points compared to non-snorers (OR 2.7, 95% CI 1.0−7.2, p = 0.056). No	Not investigated	Not investigated

			differences in BW with total BQ. Growth not investigated		
Stacey et al. 2015 [44] New Zealand	Case-control/Snoring question	n = 465 (n = 155 stillbirth)	BW not investigated. Growth not investigated	Not investigated	No difference in snoring in the SB (cases) or live born (controls)
Tauman et al. 2011 [21] Israel	Cross sectional (recruited in labour)/Snoring question	n = 122 (n = 48 habitual snorers)	No difference in BW or birth centile. Growth not investigated	Not investigated (PTB excluded)	Not investigated
Tauman et al. 2012 [22] Israel	Cross sectional (recruited in labour)/Snoring question	n = 246 (n = 20 chronic snorers and n = 58 pregnancy onset snorers)	No difference in BW or birth centile (unadjusted). No difference in growth (but very few FGR)	Not investigated (PTB excluded)	Not investigated
Ugur et al. 2012 [32] Turkey	Prospective/BQ	n = 465 (n = 69 BQ positive)	Slightly lower BW in BQ positive group (p = 0.055). No difference in growth	Not investigated	Not investigated

BW: Birth weight. SGA: Small for gestational age. LGA: Large for gestational age. PTB: Preterm birth. FGR: Fetal growth restriction. SB Stillbirth.
BQ: Berlin Questionnaire. MAP index: Multivariable apnea risk index.
BMI: Body Mass Index.
Not Reported: data collected but not reported. Not Investigated: Outcome of interest not investigated.

The impact of maternal sleep on fetal outcome

The papers identified reported fetal outcomes associated with four main areas of maternal sleep, namely sleep disordered breathing (SDB), sleep duration, sleep quality, and sleep position. Literature from each of these areas is reviewed here.

Sleep disordered breathing and fetal outcomes

Sleep disordered breathing (SDB) is a spectrum of nocturnal breathing disorders that consists of primary snoring, upper airway resistance and obstructive sleep apnea (OSA) [7]. The latter is characterised by repeated occurrences of complete or partial upper airway collapse during sleep with recurrent episodes of gas exchange abnormalities and repeated arousals [7].

Potential mechanisms relating maternal SDB to fetal outcomes may include inflammatory cascades, maternal oxygen desaturation, placental dysfunction and intermittent bouts of fetal hypoxemia. In hypertensive pregnant women, SDB may worsen the already high peripheral vascular resistance and decrease cardiac output, which may then compromise uterine and placental blood flow leading to a higher risk of fetal compromise [8]. Inflammation, oxidative stress, and endothelial dysfunction have all been implicated not only in SDB [9] but also in adverse pregnancy outcomes [10,11].

SDB may also impact on fetal wellbeing. For example, the association of maternal apneic episodes with fetal heart rate decelerations was first described in a case series of obese pregnant women in 1978 [12]. Furthermore, in 2000 the first study of maternal SDB demonstrated that habitual snoring was associated with maternal hypertension and infants born small for gestational age (SGA) [13]. Since then there has been a surge of interest in the role of maternal SDB, particularly as it relates to maternal pregnancy outcomes such as gestational hypertension, pre-eclampsia, and gestational diabetes. This has culminated in several recent systematic literature reviews and meta-analyses [1–5], none of which summarised fetal outcomes. Given the conflicting findings in the literature to date and the lack of focus on fetal outcomes, we identified and reviewed data from 42 studies that included fetal outcomes associated with SDB. We further divided these into studies where subjective measures of SDB were employed and where objective measures were used (Tables 1 and 2). We did this because in the non-pregnant literature self-reported snoring often predicts outcomes that polysomnography (PSG) fails to and standard PSG may be insensitive to physiological variables that nonetheless have substantial health consequences.

Subjective measures of SDB and fetal outcome

Birth weight

There was a mix of study designs defining SDB by the presence of snoring or by the Berlin Questionnaire (BQ), a well validated scale that incorporates questions about snoring, daytime somnolence, hypertension and body mass index (BMI) [14]. In the studies that reported birth weight (Table 1), the majority failed to find any relationship with snoring. There is only one report, from a large prospective study of over 1000 women, of an increased risk of low birth weight (LBW) in women with frequent/almost always snoring (aRR 2.6 95% CI 1.2–5.4) [17]. In a prospective study of n = 69 women with a positive BQ, compared to n = 396 with a negative questionnaire, Ugur et al. reported a, lower birth weight trending towards significance (3155.2 ± 650.8 vs 3280.3g ± 458.9g p = 0.055) [32]. However, Sharma et al. [23], in a smaller prospective study of n = 273 women, did not find a relationship with a positive BQ (n = 18) but did find a trend to a lower birth weight in infants of women who snored during each trimester, similar

Table 2
Objective measures of OSA and fetal outcomes.

Author, year, country	Design/Methods used	Sample size	Fetal Outcomes		
			Weight/Growth	PTB	SB
Bassan et al. 2016 Israel [46]	Prospective	n = 44 (n = 11 OSA)	No difference in BW or centile. No difference in growth	No difference in GA	Not investigated
Bin et al. 2016 Australia [56]	Population-based hospital discharge database	n = 636,227 (n = 519 OSA)	BW not investigated. No difference in SGA but increased LGA (aRR 1.3, 95%CI 1.0–1.6)	Increased PTB (aRR 1.5, 95%CI 1.2–1.8)	No difference in perinatal deaths (SB and neonatal death combined)
Chen et al. 2012 Taiwan [54]	Cross-sectional population database	n = 4746 (n = 791 OSA)	Increased LBW (aOR 1.8, 95% CI 1.3 –2.4). Increased SGA (aOR 1.3, 95% CI 1.1–1.7)	Increased PTB (aOR 2.3, 95%CI 1.8–3.0)	Not investigated
Facco et al. 2012 USA [63]	Retrospective chart review	n = 143 (n = 60 OSA)	BW and growth not investigated	PTB not presented but calculation using raw data found 10% PTB in OSA vs. 4.8% PTB in non-OSA (p = 0.4)	Not investigated
Facco et al. 2014 USA [47]	Prospective	n = 188 (n = 56 OSA)	BW not investigated. No difference in SGA<5th centile	No difference in PTB <34/40	Not investigated
Felder et al. *2017* [58] *USA*	Observational Cohort (Linked data)	n = 2,963,888 n = 2172 Sleep disorder 'propensity score'	Not Investigated	Nearly 15% of women with a recorded sleep disorder diagnosis delivered before 37 weeks of gestation compared with 10.9% of women without a sleep disorder OR 1.4, 95% CI 1.2–1.7).	Not Investigated
Fung et al. 2013 Australia [48]	Prospective	n = 41 (n = 14 OSA)	No differences in BW or birth centile. Evidence for slowing in fetal growth in late gestation	PTB not investigated; GA was slightly shorter in OSA (38.7 vs. 39.4 weeks, p = 0.06)	Not investigated
Louis et al. 2010 USA [49]	Case-control	n = 171 (n = 57 OSA)	BW lower in OSA compared to obese women but BW higher compared to non-obese controls. No differences in growth	Increased PTB <37/40 and < 32/40 compared to obese and to non-obese	Not investigated
Louis et al. 2012 USA [50]	Prospective	n = 175 obese women (n = 27 OSA)	No differences in BW. No differences with SGA or LGA	No differences with PTB <37/40 or <32/40	Noted that 2 SB occurred in control group, none in OSA
Louis et al. 2014 USA [55]	Retrospective cross sectional national inpatient discharge database	n = 55,781,965 (unclear how many had OSA)	BW not reported. No differences in growth	Increase in early onset delivery but definition not clear	No differences
Maasilta et al. 2001 Finland [51]	Case-control	n = 22 (n = 1 OSA)	No differences in BW between obese and non-obese. Only 1 IUGR in non-OSA	No differences in GA but PTB not reported	Not investigated
Olivarez et al. 2010 USA [61]	Prospective	n = 100 (n = 20 OSA)	BW and growth not investigated	No difference in GA but preterm labour trending towards significant (40% in OSA vs. 20% in non-OSA p = 0.06)	Not investigated
Pamidi et al. 2016 Canada [36]	Prospective	n = 230 (n = 153 OSA)	BW not different between AHI categories (<5, 5–10, 10–15, >15). Increased proportion of SGA with increasing OSA severity.	No differences in PTB <37/40 across AHI categories	Not investigated
Sahin et al. 2008 Turkey [52]	Prospective	n = 35 (n = 4 OSA)	No differences in BW or growth	Not investigated	Not investigated
Spence et al. *2017* USA [57]	Retrospective Cohort: database	n = 305,001 (n = 266 OSA diagnosis)	Not Investigated	Increased risk of PTB in OSA aOR 1.90 (1.09 –3.30)	Not Investigated
Yin et al. 2008 England [53]	Cross-sectional	n = 150 (n = 2 OSA)	BW not investigated. Women with IUGR fetuses not more likely to have OSA than those with normally growing fetuses	Not investigated	Not investigated

BW: Birth weight. SGA: Small for gestational age. LGA: Large for gestational age. PTB: Preterm birth. FGR: Fetal growth restriction. SB Stillbirth.
GA Gestational age OSA: Obstructive sleep apnea AHI: Apnea Hypopnea index.
BMI: Body Mass Index.
Not Reported: data collected but not reported Not Investigated: Outcome of interest not investigated.

Table 3
Sleep duration and fetal outcomes.

Author, Year, Country	Design/Methods used	Sample size	Fetal Outcomes		
			Weight/Growth	PTB	SB
Abeysena et al. 2009 [67] Sri Lanka	Prospective/question about sleep duration	n = 690 (n = 194 ≤8hr sleep)	BW not reported. Sleeping ≤8hr associated with SGA <5th centile (aOR 2.2 95%CI 1.1−4.6, p = 0.03)	Not investigated	Not investigated
Abseysena et al. 2010 [68] Sri Lanka	Prospective/question about sleep duration	n = 739 (n = 204 ≤ 8 h sleep)	Sleeping ≤8hr associated with LBW (aOR 2.8, 95%CI 1.5−5.4, p = 0.002). Growth not reported	Not investigated	Not investigated
Guendelman et al. 2013 [79] USA	Nested population case control	n = 1042 (n = 305 < 7 h sleep and n = 120 > 8 h sleep)	BW and growth not investigated	Sleeping <7 or >8hr not associated with PTB. Not enough women sleeping <6 h to analyse although there appeared to be increased PTB	Not investigated
Heazell et al. *2017* [72] *UK*	Case-Control	N = 291 stillborn cases N = 733 ongoing pregnant	Not investigated	Not investigated	≤5.5 h night before associated with stillbirth (aOR 1.83, 95% CI 1.24−2.68)
Howe et al. 2015 [33] New Zealand	Prospective/Subjective questions (including naps)	n = 633 (n = 23 ≤6hr and n = 155 ≥ 9 h sleep)	BW not reported. No differences with short (≤6hr) or long (≥9hr) sleep and SGA or LGA	Not investigated	Not investigated
Kajeepeta et al. 2014 [78] Peru	Case control	n = 959 (n = 186 ≤ 6 h sleep, n = 164 ≥ 9 h sleep)	BW and growth not reported	Short sleep (≤6hr) associated with increased PTB (aOR 1.6, 95%CI 1.1−2.2).	Not investigated
Li et al. 2016 [76] China	Prospective/Subjective question from PSQI	n = 688 (n-22 ≤7hr sleep)	BW not reported. Growth not investigated	Short sleep (<7 h) had increased PTB (aOR 4.7, 95%CI 1.2−17.5)	Not investigated
McCowan et al. *2017* [73] *New Zealand*	Case Control	N = 164 stillborn cases N = 569 ongoing/liveborn	Not investigated	Not Investigated	≤ 6 h on the last night associated with Stillbirth (aOR 1.81 95%CI 1.14−2.88)
Micheli et al. 2011 [17] Greece	Prospective/Subjective questions on computer-assisted interview	n = 1091 (n = 73 ≤ 5 h sleep)	No association with LBW or SGA<10th centile	Short sleep (≤5hr) associated with PTB (aRR 1.7, 95% CI 1.1−2.8), with highest risk for medically indicated PTB (aOR 2.4, 95%CI 1.0−6.4)	Excluded n = 1 stillbirth
Okun et al. 201 [77] USA	Prospective/Subjective question from Hamilton Depression Scale (secondary analysis)	n = 217 (n = 57 < 7 h sleep, n = 29 > 9 h sleep)	BW and growth not investigated	No association between short sleepers (<7 h) or long sleepers (>9 h) at either 20/40 or 30/40 and PTB	Not investigated
Okun et al. 2013 [69] USA	Prospective/Subjective question from Hamilton Depression Scale (secondary analysis)	n = 168 (n = 32 < 7 h sleep, n = 26 > 9 h sleep)	BW lower in short sleepers (<7 h sleep) at 30 weeks (β = −424.3, p = 0.031) but only in depressed women. No differences in growth with short or long sleep. However, time in bed <7 h or >9 h at 30 weeks associated with babies >4 kg (p = 0.04 and 0.06 respectively)	No relationship between sleep and PTB. For depressed women at 30 weeks, higher inflammatory markers were associated with increased PTB (OR 1.2, p = 0.032)	Not investigated
Owusu et al. 2013 [18] Ghana	Cross-sectional	n = 220 (n = 37 ≤ 6 h sleep and n = 57 ≥ 10 h sleep)	BW not reported. No difference between sleep duration groups and LBW	No difference	No difference
Plancoulaine et al. *2017* [74] France	Cohort Self report/delivery data	N = 200 three sleep duration trajectories 'short-decreasing (<6.5h/night, 10.8% of the sample), medium-decreasing	Birth-weight-z-score was lower in the long-increasing trajectory group.	Those in the 'short-decreasing' sleep duration trajectory group was	Not Investigated

(*continued on next page*)

Table 3 (*continued*)

Author, Year, Country	Design/Methods used	Sample size	Fetal Outcomes		
			Weight/Growth	PTB	SB
		(6.5–8h/night, 57.6%), and long-increasing (>8h/night, 31.6%)	NB cannot easily convert this into an OR effect size	more at risk for preterm birth,	
Rabkin et al. *1990* [70] England	Prospective	n = 1507 (n = 145 < 7 h sleep; n = 199 ≥ 9 h sleep at 17 weeks)	Long sleepers (>9 h sleep) before the 17 and 28 weeks had mean birth weights 74g and 60g higher, respectively; a linear trend evident at 17 weeks (p = 0.04). Growth not reported	Not reported	Excluded (n = 14 macerated stillbirths)
Stacey et al. *2011* [44] New Zealand	Population-based matched case–control/self report sleep duration in last month	n = 465 Cases (n = 155) Controls (n = 310)	Not Investigated	Not Investigated	More (n = 43 28%) cases than controls (n = 59 19%) self reported >8 h average sleep per night in last month of pregnancy (OR 1.83 (1.14–2.94)
Wang et al. *2017* [75] *China*	Prospective Cohort. PSQI and hospital records	N = 3567	No significant association with birth weight. However, shorter sleep duration in early pregnancy was associated with shorter birth length. 2.42 mm (95% CI: −4.27 to −0.58, p = 0.010)	No Association	SBs excluded
Zafarghandi et al. 2012 [71] *Iran*	Cross-sectional	n = 457 (n = 154 <8hr sleep)	BW reported as 3 groups (<2500g, 2500 −3500g, >3500g). Calculations from raw data show higher proportion of infants >3500g had mothers sleeping <8 h (22% vs. 12.5%, p = 0.01). No association between sleep duration and SGA.	Excluded	Not investigated

BW: Birth weight. SGA: Small for gestational age. LGA: Large for gestational age. PTB: Preterm birth. FGR: Fetal growth restriction. SB Stillbirth.
BQ: Berlin Questionnaire. MAP index: Multivariable apnea risk index: PSQI: Pittsburgh Sleep Quality Index.
BMI: Body Mass Index.
Not Reported: data collected but not reported. Not Investigated: Outcome of interest not investigated.

Table 4
Sleep quality and fetal outcomes.

Author, Year, Country	Design/Methods	Sample size	Fetal Outcomes		
			Weight/Growth	PTB	SB
Blair et al. 2015 [83] USA	Observational PSQI (secondary analysis)	n = 138 (n = 78 with clinically disturbed sleep)	BW and growth not investigated	Poor quality sleep associated with PTB (aOR 4.1, 95% CI 1.0−16.3).Using sleep as a continuous variable (total PSQI), the odds of PTB increased by 1.4 (95% CI 1.2−1.6) with each unit increase in PSQI. African American women with poor sleep quality had 10.2 times the odds of PTB compared to those with good sleep quality.	Not investigated
Dolation et al. 2014 [92] Iran	Prospective ISI	n = 231 (n = 119 with "Sleep disorders")	BW categorised as <2500g, 2600−3000g, 3001−4000g, and >400g. BW lower in "sleep disorder" group (p = 0.07) but no mean values provided. Calculations from raw data show no differences in SGA or LGA in women with and without "Sleep disorders"	Difficulty falling asleep was much less in the preterm group (3% vs. 45% p = 0.06)	Not investigated
Hernandez-Diaz et al. 2014 [93] USA	Case control "disturbed sleep"	n = 258 (n = 54 disturbed sleep)	BW and growth not investigated	Disturbed sleep associated with PTB (OR 4.5, 95%CI 1.5, 13.3)	Not investigated
Howe et al. 2015 [33] New Zealand	Prospective GSDS	n = 633 (n = 527 poor sleep quality)	BW not reported. No relationship between short sleep (≤6hr) or long sleep (≥9hr) and SGA or LGA.	Not investigated (recruited late pregnancy)	Not investigated
Hung et al. 2014 [84] Taiwan	Prospective PSQI	n = 248 (n = 153 poor sleepers)	No association with BW and poor sleep quality. Growth not reported	No association with poor sleep quality	Not investigated
Li et al. 2016 [76] China	Prospective PSQI	n = 688 (n = 53 poor sleep quality at 2nd trimester and n = 127 at 3rd trimester)	BW not reported. Growth not investigated	Poor sleep quality in the second and third trimester associated with increased PTB (aOR 5.4, 95%CI 2.1−13.6 and aOR 3.0 95% CI 1.3−7.2 respectively)	Not investigated
Naghi et al. 2011 [85] Iran	Prospective PSQI	n = 488 (n = 214 poor sleep quality)	No difference in BW. Growth not reported	Not investigated as excluded <36 weeks gestation but mean GA at birth not different	Not investigated
Okun et al. 2011 [86] *USA*	Prospective PSQI	n = 166 (n = 48 with poor sleep quality)	BW and growth not investigated	Poor sleep quality in early pregnancy associated with PTB (aOR: 1.3, 95% CI 1.0−1.5). With every one-point increase in PSQI, the odds of preterm birth increased 25% in early pregnancy and 18% in later pregnancy	Not investigated
Owusu et al. 2013 [18] Ghana	Cross-sectional GSDS	n = 220 (n = 190 poor sleep quality)	No difference in LBW. Growth not investigated	No difference	No difference
Rajendiran et al. 2015 [87] India	Prospective PSQI	n = 68 (n = 30 ″sleep deprived")	No difference in BW. When deprived group further subdivided into scores <18 (n = 14) and ≥18 (n = 16), the ≥18 group had smaller BW (p = 0.02). Growth not reported	Not investigated	Not investigated
Sharma et al. 2016 [23] India	Prospective PSQI	n = 209 (n = 55 poor sleep quality)	No association with LBW. Growth not investigated	Not investigated	Not investigated
Stinson et al. 2003 [90] USA	Prospective GSDS	n = 359 (number with poor sleep quality not specified)	BW and growth not investigated	Perception of *Good* sleep associated with preterm labour (p = 0.01) and PTB (p = 0.05). *Good* sleep quality predicted preterm labour (OR 2.4, 95%CI 1.1−5.1, p = 0,03) but not PTB	Not investigated
Strange et al. 2009 [88] USA	Prospective PSQI	n = 220 (number with poor sleep quality not specified)	BW and growth not investigated	No association with PSQI score but longer self-report sleep latency in women with PTB (26.1 min vs. 18.5min, p = 0.03).	Not investigated
Zafarghandi et al. 2012 [71] Iran	Cross-sectional " refreshing sleep"	n = 457 (n = 66 with un-refreshing sleep)	BW reported as 3 groups (<2500g, 2500−3500g, >3500g). Calculations from raw data show 26% of un-refreshed sleepers had baby >3500g vs. 14% of refreshed/somewhat refreshed sleepers (p = 0.03). No association with un-refreshed sleep and LBW. Growth not investigated	Excluded	Not investigated

BW: Birth weight. SGA: Small for gestational age. LGA: Large for gestational age. PTB: Preterm birth. FGR: Fetal growth restriction. SB Stillbirth.
BQ: Berlin Questionnaire. MAP index: Multivariable apnea risk index. GSDS: General sleep disturbance scale.
BMI: Body Mass Index.
Not Reported: data collected but not reported. Not Investigated: Outcome of interest not investigated.

Table 5
Sleep position and fetal outcomes.

Author, Year, Country	Design/Methods	Sample size	Fetal Outcomes		
			Weight/Growth	PTB	SB
Gordon et al. *2015* [45] Australia	Matched case–control/ self report sleep position	Stillborn Cases (n = 103) Liveborn Controls (n = 192)	Cases more likely to be followed during pregnancy for suspected FGR, 11.7% v 1.6% (aOR 5.5, 95%CI 1.4 –22.5)	PTB not reported. Mean GA 36 weeks	aOR 6.26 (95%CI 1.2–34.0) for SB with supine sleep in the previous month
Heazell et al. *2017* [72] UK	Prospective case–control/self report sleep position	Stillborn Cases (n = 291) Controls (n = 733) women with an ongoing pregnancy at the time of interview.	No interaction	No Interaction	aOR 2.31 (95% CI 1.04–5.11) for SB with supine going-to-sleep position the night before stillbirth.
Lakshmi et al. *2017*,[102] India	Prospective case control study/ interview	Cases (n = 100) Control (n = 200)	The mean birth weight in the case group was 1.478 kg and the control group was 2.723 kg (a 22% increase)	PTB Not reported. The incidence of premature rupture of membranes, was comparable in both groups.	aOR 2.95 (95% CI 1.5–5.8) for SB with "non left lateral sleep pattern"
McCowan et al., 2017 [73] New Zealand	Case-control Self reported sleep position	Stillborn Cases (n = 164) Control (n = 569) women with an ongoing pregnancy at the time of interview	Cases more likely to be SGA aOR 2.76, 95% CI 1.59 to 4.80	The risk of supine going to sleep position was greater for term (aOR 10.26, 3.00–35.04) than preterm stillbirths (aOR 3.12, 0.97–10.05)	aOR 3.67, (95% CI 1.74–7.78) for SB with supine going-to-sleep position on the last night
Owusu et al. *2013* [18] Ghana	Cross-sectional/self report sleep position	n = 220	Mean birth weight not significant. Women who reported supine sleep during pregnancy were ar increased risk of LBW (OR 5.0, 95%CI, 1.2–20.2: p = 0.03	No Difference	aOR 8.0 (95% CI 1.5–43.2) for SB in supine/supine and side sleep the week before delivery
Stacey et al. *2011* [44] New Zealand	Population-based matched case –control/self report sleep position	Case (n = 155) Control (n = 310)	Not reported	Not Investigated	aOR 2.54 (95% CI 1.04–6.18) for SB with supine sleep the night before the demise. aOR 1.88 (95%CI 1.14–3.10) for SB with right-sided sleep the night before the demise

BW: Birth weight. SGA: Small for gestational age. LGA: Large for gestational age. PTB: Preterm birth. FGR: Fetal growth restriction. SB Stillbirth.
Not Reported: data collected but not reported. Not Investigated: Outcome of interest not investigated.

findings to those of Guilleminault et al. [25] from their prospective study of n = 267 women with n = 10 loud and chronic snorers. Despite not finding a relationship with LBW, Tauman et al. [21], in a cross-sectional study of n = 122 women recruited during labour, found that nucleated red blood cells, an indicator of fetal hypoxemia, were higher in the cord blood of infants born from snoring women (n = 48). A few studies have reported that snoring women delivered babies with larger birth weights compared to non-snoring women [20,26,27,31], possibly related to maternal obesity, as all but one study used the BQ, which includes maternal weight. Olivarez et al. stratified their cohort by BMI and found that the association between high birth weight in snoring women was only present in those with a body mass index (BMI) > 30 [31]. Ge et al. examined macrosomia (defined as birth weight >4000g) in a Chinese population and found that pregnancy-onset snorers had an increased risk for large babies (RR 1.54, 95% CI 1.05–2.27). In the same study, when compared to lean non-snorers (BMI<24 kg/m^2), both lean snorers and overweight/obese snorers (BMI≥24 kg/m^2), had an increased relative risk of macrosomia (aRR 1.61, 95% CI 1.09–2.37 and aRR 2.27, 95% CI 1.25–4.11, respectively) [20].

In summary, the data regarding subjective measures of SDB suggest that there is no clear association with birth weight as most failed to demonstrate a relationship. Even the three large studies (≥1000 participants) [17,26,30] that reported birth weight, each reported a different finding of higher birth weight, lower birth weight, and no difference in birth weight. Nonetheless, birth weight *per se* is perhaps not the best measure since it is dependent on gestational age. A better outcome measure is fetal growth, which is discussed below.

Fetal growth

Several studies examined fetal growth rather than only birth weight as their outcome measure [13,16,17,19,20,22,27,29–37]. Associations were observed in cohort studies between habitual snoring and SGA/FGR [13,17,30] after adjusting for covariates. One cross-sectional study did not find a relationship with snoring and growth restriction on the day of delivery [35]. However, the number of infants with growth restriction in the latter study, as well as others [22,34], was not sufficient to perform a multivariable regression model. Of note, O'Brien et al., demonstrated that the timing of onset of maternal SDB symptoms may be relevant to fetal outcome in a study that provided enough power to detect the difference in SGA frequencies [30]. In this study only chronic habitual snoring (aOR 1.7 95% CI 1.0–2.7), but not pregnancy onset habitual snoring, was associated with SGA <10th centile (as defined by customised birth centiles [38] rather than population norms). McGillick points out that, the use of population based or customised growth charts to determine fetal growth restriction may, in part, explain some of the conflicting reports with regards to maternal SDB and fetal growth [39].

Similar to studies on birth weight alone, some studies have also associated SDB symptoms with excessive fetal growth i.e., weight adjusted for gestational age. For example, Antony et al. found that maternal snoring was associated with large for gestational age (LGA) infants, but not SGA, in a predominantly Hispanic cohort [37], as did Ge et al. in a Chinese population [20]. Some support for these findings is also provided by O'Brien et al. [30] and Howe et al. [33] who both reported that women with witnessed apneas were more likely to have infants born LGA. One possible mechanism for larger fetuses could be that SDB and maternal obesity are somewhat interrelated and the impact of maternal obesity and/or diabetes resulting in increased transfer of nutrients to the fetus [40] causing fetal hyperglycemia, hyperinsulinemia, and/or dyslipidemia [41]. This seems to be supported when pre-pregnancy BMI is accounted for in multivariate regression and the apparent association with abnormal glucose levels disappears [30].

In summary, most of the studies that reported on birth centile as an outcome measure suggest that there is no difference in fetal growth between women with and without subjective measures of SDB. However, of five large studies (≥1000 participants) [17,20,30,35,37] three that used only a question about snoring found that SGA was more common in snoring women [17,30,35] whereas the one study that used the BQ [20] found an association with LGA. The BQ takes account of maternal weight as well as snoring and sleepiness so it is not possible to tease out the role of snoring alone in studies that use it and it is plausible that obesity plays a role in LGA. Indeed, in the study by Ge [20] the RR of macrosomia was highest in overweight/obese snorers.

Preterm delivery

A total of 11 studies made an assessment of the association between SDB and preterm birth (PTB) [15–18,20,27,29,34,35,37,42] and another three reported gestational length [13,19,28]. In one of the first studies in this area, Loube et al. [29] in a prospective study of n = 350 women, reported outcomes as a composite score and found no difference between snoring (n = 49) and non-snoring groups. However, inspection of their raw (non-composite) data suggests that there was an increase in PTB in the snoring group (14% of snoring women vs 1.7% of non-snoring, $p < 0.001$). In a prospective cohort of n = 1153 women, Antony et al. [37] found an association between a positive Berlin screen and PTB in an unadjusted model but this became statistically non-significant after adjustment for other variables known to be associated with preterm birth. The majority of the prospective cohort studies using subjective measures either failed to find a relationship between habitual snoring and PTB [17,20,27], despite two of the latter studies having cohorts of >1000 women [17,20]), or did not report PTB or gestational age as an outcome [23,25,26,28,30–33,36]. Similarly, both a small cross-sectional study (n = 220) and a small case–control study (n = 83) failed to find any relationship [16,18]. Perez-Chada et al., in a cross-sectional study of women on the day of birth, found a trend towards a statistically significant shortening of gestation in n = 32 snorers compared to n = 415 non-snorers (38.8 ± 1.8wks vs 39.2 ± 1.9 wks p = 0.069) [19] but this difference in gestational length has limited clinical significance. One large cross-sectional study of n = 1000 women within 48 h postpartum reported that 'gasping for air' was associated with an increased odds of PTB after accounting for maternal age, smoking, and multifetal pregnancies (aOR 1.8, 95% CI 1.1–3.2) [35] but no data were provided after adjustment for other factors known to be related to PTB such as pre-eclampsia. The other cross-sectional studies did not specifically investigate PTB, probably due to study design [21,22,24].

In summary, only a small number of studies utilising subjective measures of SDB have investigated preterm birth. While findings appear inconsistent, it is important to note that in four [20,35,37,42] of the five largest studies (≥1000 women) [17,20,35,37,42] that have reported preterm birth, an association between either snoring or witnessed apnea with preterm birth has been reported. This suggests that power to detect an association may be lacking in smaller studies.

Stillbirth

Few studies investigating subjective reports of SDB reported stillbirth as an outcome. However, the prevalence of stillbirth in most high income countries is between 2 and 5 per 1000 [43] and thus most studies are not powered to determine statistically significant differences, even if they were to include this variable. Indeed in the study by Franklin et al. [13], stillbirths were excluded

for this reason. Neither the case control studies [44,45] nor the cross-sectional study [18] reported an association between snoring and stillbirth, although the former were retrospective case–control design and thus may have been subject to recall bias and the latter was not specifically powered for stillbirth as an outcome.

Objective measures of OSA

Birth weight

We identified 16 studies utilising objective measures of OSA that reported fetal outcomes of interest (Table 2). Nine studies examined birth weight or growth as an outcome either by recruitment of women to undergo PSG or by medical record review of women who had undergone PSG [36,46–53] and an additional five studies used population-based datasets [54–58]. Of those that investigated birth weight, the vast majority reported no difference in birth weight [36,46,48,50–52], although one study of n = 171 women found lower birth weight in n = 57 women with OSA when compared to obese women, but higher birth weight when compared to infants of non-obese controls [49]. Cross-sectional data from a large (n = 4700) population-based database from Taiwan [54] showed an increased frequency of LBW in women who received a diagnosis of OSA within one year prior to birth.

In summary, the literature regarding objective measures of OSA and birth weight is very limited. All studies, except retrospective population studies utilising discharge data, comprise less than n = 230 women. Of the single population-based study that has reported birth weight, an association was found between LBW and a diagnosis of OSA. However, these findings remain to be demonstrated in a large prospective study.

Fetal growth

Most of the non-epidemiological studies (with sample sizes less than n = 230 women) that reported FGR did not find a relationship with OSA [46–53]. Although in the largest published prospective study to date, Pamidi et al. [36] have recently reported that there is 2–3 fold increase in the odds of SGA at various thresholds of OSA severity (OR 2.65; 95% CI 1.15–6.10; p = 0.02). In the five studies utilising the large epidemiological databases [54–58] only Chen et al. [54] reported that infants of women with OSA were more likely to have SGA, whereas two reported no differences in SGA between women with and without a diagnosis of OSA [55,56] and two did not report SGA [57,58]. However, one of the latter studies of hospital discharges showed an increase in LGA infants [56]. Nevertheless these population-based studies, by virtue of their design, cannot know how many women assigned to the control group actually had undiagnosed OSA, nor how many received treatment and how that may have impacted the findings.

A single measure of birth weight/centile after delivery does not describe the pattern of fetal growth across gestation, as highlighted by Fung et al. who showed that fetuses of women with OSA demonstrate a fall across growth centiles, even if the resulting birth centile is not considered in the FGR range [48]. Recent data using serial fetal growth measures supports the suggestion that maternal OSA is associated with faltering of fetal growth in the third trimester regardless of the presence of FGR [59]. Furthermore, there are case reports demonstrating fetal heart rate decelerations in response to maternal apneas and oxygen desaturations [12,60]. Although, there are currently few observational studies, Sahin et al. [52] found that three of four women with OSA had fetal heart rate decelerations that were associated with maternal oxygen desaturations. Nonetheless, Olivarez et al. [61] were unable to support this with their study, which only included women who had been admitted to hospital for clinical reasons. However, emerging data from our group suggests there is a relationship between maternal oxygen desaturations and fetal heart rate decelerations when the mother is asleep [62].

In summary, few studies of maternal OSA have investigated fetal growth as an outcome measure. Most of those that have, do not find a relationship with birth centile. Of note, the largest prospective study (n = 230) did find that increasing severity of OSA was associated with increasing frequency of SGA [36]. Taken together with a recent report of fetal growth slowing across the third trimester [48,59], this is suggestive that maternal OSA may impact fetal growth regardless of whether birth centile crosses the threshold to SGA and could explain, in part, these inconsistent findings.

Preterm birth

Thirteen studies utilising objective measures of OSA reported PTB or gestational age as an outcome (Table 2). Of these, the four larger studies identified a clear association between OSA and PTB [54,56–58], while seven studies (all n < 230) showed no evidence of an association between OSA and PTB/gestational age [36,46,47,50,51,61,63]. However, definitions of PTB were not consistent across studies and included <37 weeks, <34 weeks, and <32 weeks, making it difficult to compare data, as concluded in other studies [39]. Indeed, the largest population-based dataset of over 55 million women reported an increase in "early onset delivery" but it is unclear what this meant because gestational age was not reported [55]. Two recent reports from large cohort studies (n = 305,001 and 2,963,888) that both used data from population databases indicated that PTB was associated with sleep disorders including diagnosis of OSA with aOR of 1.9 (1.4–2.6) and 1.9 (1.09–3.30), respectively [57,58]. While a small prospective study of n = 100 women (n = 20 with OSA) did not report PTB, there was a small increase in PTB in women with OSA compared to those without (40% vs. 20%; p = 0.06) [61]. In the study by Fung et al. [48], despite exclusion of PTB as an outcome criteria, they noted that gestational length of infants born > 37 weeks was slightly shorter in n = 14 women with OSA compared to n = 27 without (38.7 ± 1 vs. 39.4 ± 1.3 weeks; p = 0.06).

In summary, data from larger population-based studies suggest that there is a relationship between maternal OSA and PTB while small studies (<230 women) do not, although the latter studies were unable to be directly compared as different thresholds for the definition of PTB were used. The lack of sample size calculations to demonstrate the number needed for adequate power and the lack of consistent definitions of PTB limit the interpretation of the smaller studies. While large population based linked data studies suggest there is a relationship between OSA and PTB, they are limited by the fact that OSA is rarely diagnosed in pregnancy and there may be significant differences between co-morbidities for PTB and OSA (e.g., morbid obesity) of those who do receive a diagnosis versus those who do not. Therefore large, prospective studies are needed in order to determine whether a relationship with maternal OSA and PTB exists.

Stillbirth

Only three studies, two by Louis et al. [50,55] and one by Bin et al. [56], reported stillbirth between women with and without OSA as an outcome measure. In a prospective cohort of 161 obese pregnant women, Louis et al. reported two stillbirths in the control group but none in the OSA group [50]. The same team, in a 10 year retrospective review of almost 56 million hospital discharge records from a Nationwide US sample, reported no relationship between a clinical diagnosis of OSA and stillbirth [55]. Recently Bin et al. [56] reported an increase in perinatal deaths (comprising both stillbirth and neonatal death) in the OSA group compared to the non-OSA group (RR 2.2, 95% CI 1.2–4.0). However, after adjusting for variables known to be associated with stillbirth, the relationship with OSA and stillbirth was no longer statistically significant (aRR

1.7, 95% CI 0.9–3.3). The distinct lack of data in this area leaves unanswered questions regarding this relationship.

Practice points and research agenda for SDB and fetal outcome

As this review illustrates, the few studies that have investigated the associations between maternal SDB and fetal outcomes do not have consistent findings. Nevertheless, as Figs. 1–4 show, the outcome of random effects meta-analysis indicate overall statistically significant results. The current discrepancies in results from individual studies are likely related to large variations in sample size, variations in definitions of outcome measures (eg SGA, PTB), lack of adequately powered studies and power for rare outcomes such as stillbirth. Other sources of differences are different study designs, use of objective or subjective measures,

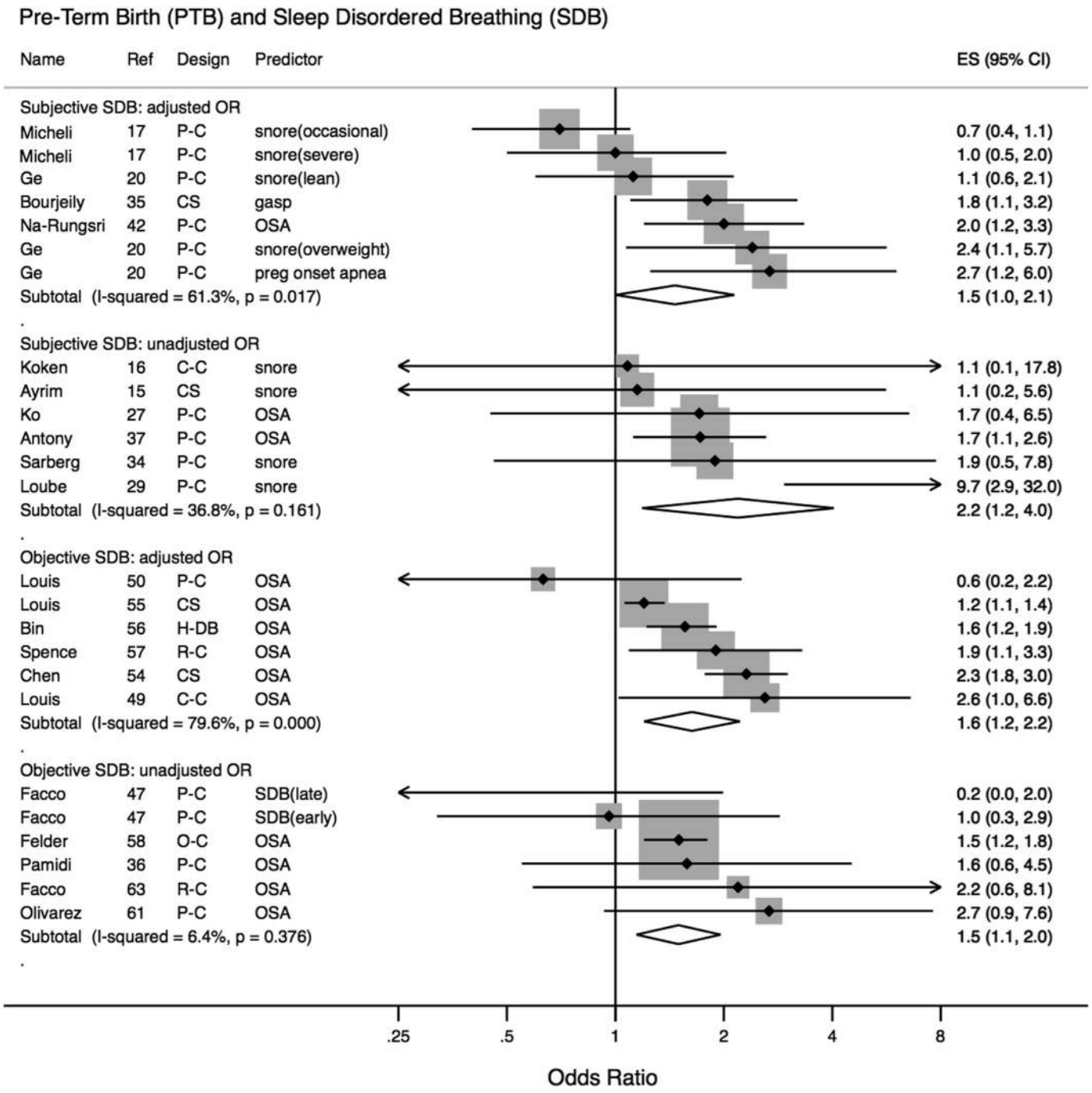

Fig. 2. Pre-Term Birth (PTB): Subjective (upper) and Objective Measures (lower) of SDB (studies from Tables 1 and 2). Black markers represent odds ratios with 95% confidence intervals (whiskers). The size of each grey square represents the relative weight in the random effects meta-analysis. Studies are grouped by those presenting adjusted versus unadjusted estimates. Diamonds represent OR summary values for adjusted and unadjusted studies separately. I2 describes the degree of inconsistency across study results (percentage of total variation across studies that is due to heterogeneity rather than chance). As an interpretation guide - 0–30% would indicate a relatively small effect, 30–50% a moderate effect, and >50% a large effect [105]. Studies are ordered by effect size (smallest to largest). Left columns provide primary author and reference number, primary predictor variable and study design. OSA = obstructive sleep apnea, preg = pregnancy. PeC = prospective cohort; CS = cross-sectional; C–C = case control. Right columns provide OR (95% confidence intervals). Note: Given the variation in variables and study designs, OR summaries should be interpreted with caution. Adjusted OR values not reported in the article were converted from reported adjusted relative risks and sample information. Unadjusted OR values not reported in the article were calculated from counts.

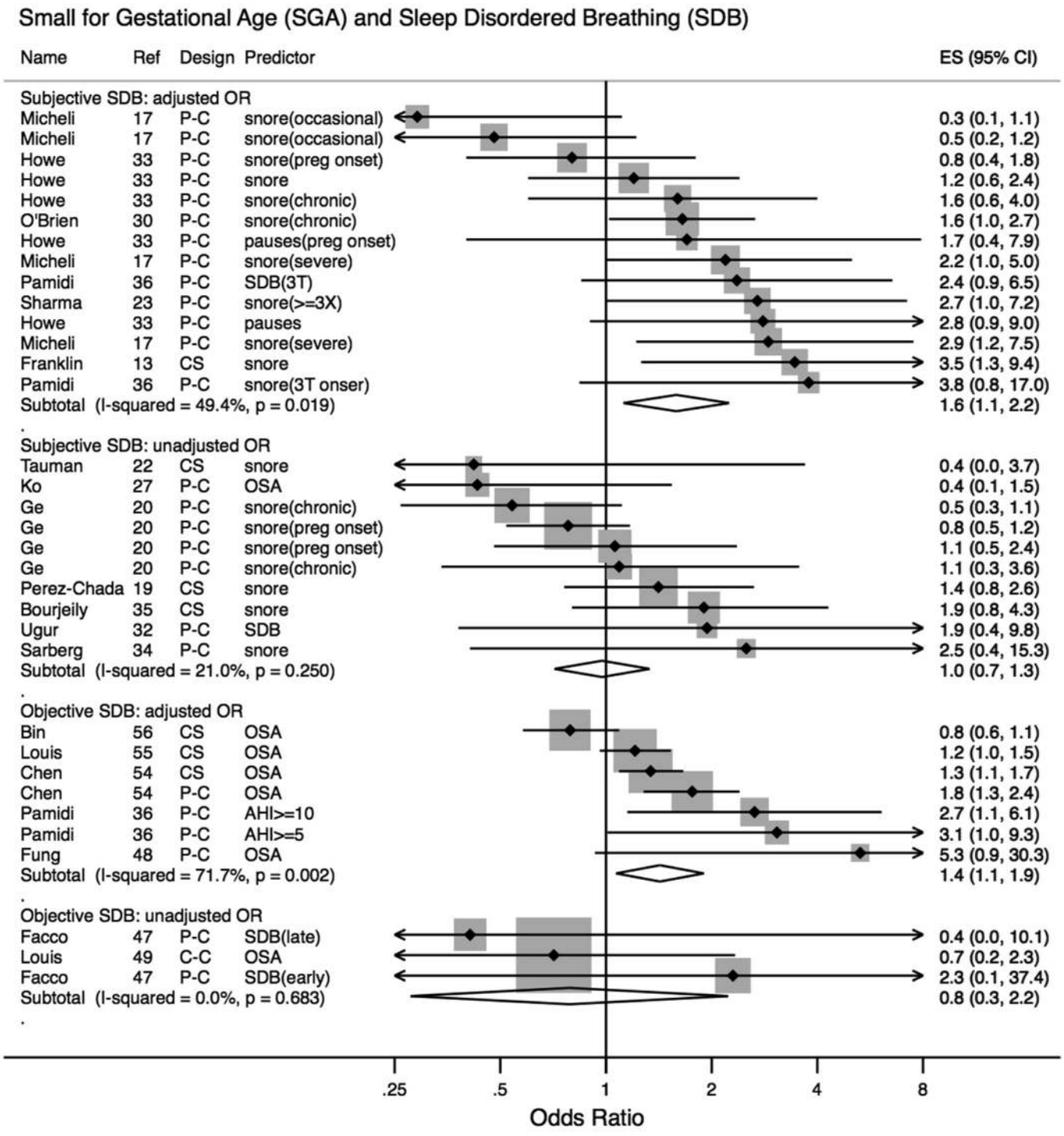

Fig. 3. Small for Gestational Age (SGA): Subjective (upper) and Objective Measures (lower) of SDB (studies from Tables 1 and 2). Black markers represent odds ratios with 95% confidence intervals (whiskers). The size of each grey square represents the relative weight in the random effects meta-analysis. Studies are grouped by those presenting adjusted versus unadjusted estimates. SGA is otherwise referred to in articles as low birth weight, intrauterine growth restriction or fetal growth restriction. I2 describes the degree of inconsistency across study results (percentage of total variation across studies that is due to heterogeneity rather than chance). As an interpretation guide - 0–30% would indicate a relatively small effect, 30–50% a moderate effect, and >50% a large effect [105]. Studies are ordered by effect size (smallest to largest). Left columns provide primary author and reference, primary predictor variable and study design. OSA = obstructive sleep apnea; preg = pregnancy; SDB = sleep disordered breathing; pauses = pauses in breathing; 3T = third trimester; 3X = three times. PeC = prospective cohort; CS = cross-sectional; C–C = case control. Right columns provide OR (95% confidence intervals). Note: Given the variation in variables and study designs, OR summaries should be interpreted with caution. Adjusted OR values not reported in the article were converted from reported adjusted relative risks and sample information. Unadjusted OR values not reported in the article were calculated from counts.

different definitions of SDB/OSA, lack of adjustment for known confounders such as maternal BMI, potential difficulties with appropriate dating of pregnancies, and lack of serial fetal growth assessments during pregnancy, especially when not performed by the same operator. Although the strength of the cited epidemiological studies is that they provide large sample sizes, they should be interpreted with caution since data are based on discharge codes rather than assessment of each individual and no account is

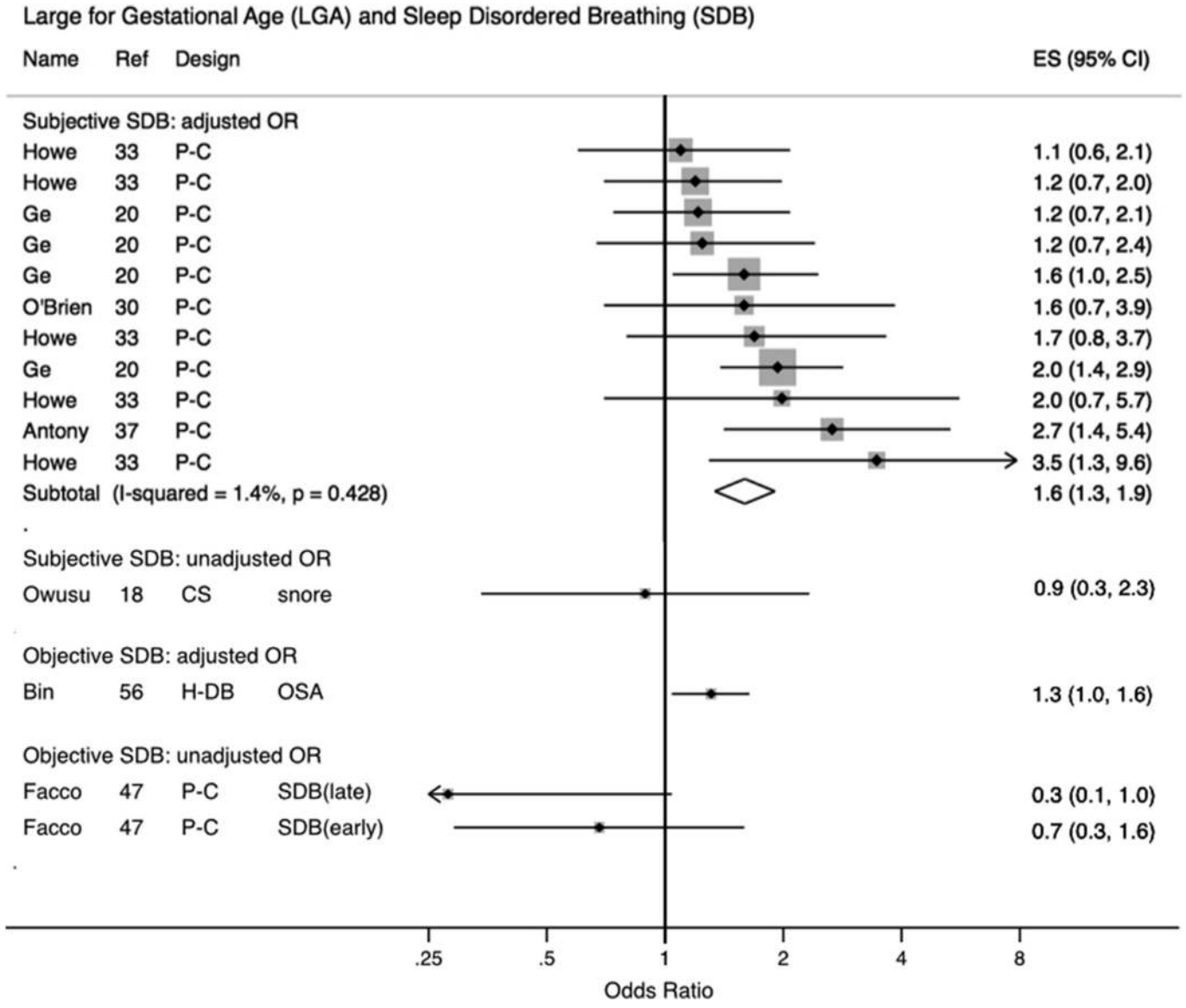

Fig. 4. Large for Gestational Age (LGA): Subjective (upper) and Objective (lower) Measures of SDB (studies from Tables 1 and 2). Black markers represent odds ratios with 95% confidence intervals (whiskers). The size of each grey square represents the relative weight in the random effects meta-analysis. Studies are grouped by those presenting adjusted versus unadjusted estimates. Diamonds represent OR summary values for adjusted and unadjusted studies separately. I2 describes the degree of inconsistency across study results (percentage of total variation across studies that is due to heterogeneity rather than chance). As an interpretation guide - 0–30% would indicate a relatively small effect, 30–50% a moderate effect, and >50% a large effect [105]. Studies are ordered by effect size (smallest to largest). Left columns provide primary author and year, primary predictor variable and study design. OSA = obstructive sleep apnea; preg = pregnancy; SDB = sleep disordered breathing; pauses = pauses in breathing; 3T = third trimester; 3X = three times. PeC = prospective cohort; CS = cross-sectional; C–C = case control. Right columns provide OR (95% confidence intervals). Note: Given the variation in variables and study designs, OR summaries should be interpreted with caution. Adjusted OR values not reported in the article were converted from reported adjusted relative risks and sample information. Unadjusted OR values not reported in the article were calculated from counts.

taken of whether or not treatment was actually received. It is important to note that symptoms and signs of SDB cannot substitute PSG in the definitive diagnosis of an individual patient in clinical practice, but they are well-recognized, highly useful research tools that in comparison to objective measures may be more likely, not less, to reveal associations between SDB and important health outcomes. Early-stage observational research to determine whether such associations are present or absent, in particular, cannot confidently rely on PSG, in part because sleep researchers have yet to understand all the physiological variables that mediate health-related SDB effects and that they should be monitored in the sleep laboratory. Furthermore, from a practical standpoint in prediction of adverse pregnancy outcomes, it would not be possible for an obstetrician to refer all women for PSG. In contrast, he/she could easily ask several simple questions validated to predict higher risk of adverse outcomes.

From examination of this literature it is clear that more large-scale, prospective cohort studies using consistent definitions are needed to further elucidate the temporal relationship between maternal SDB and fetal outcomes. To this end, a large multi-centre study of 10,000 nulliparous women that included a sleep disordered breathing sub-study of 3700 women has recently been completed in the USA [64]. The study aimed to objectively measure the presence of OSA at three time points in pregnancy in order to determine its impact on a range of pregnancy outcomes including growth restriction and PTB. Findings from this study may shed more light and ultimately evidence for practice change for diagnosis of OSA during pregnancy.

Sleep duration and fetal outcomes

The United States National Sleep Foundation recommends that adults obtain 7–9 h of sleep per night, with ≤6 h per night considered insufficient [65]. However, insufficient/short sleep duration in pregnancy is challenging to define because the optimal duration of night time sleep needed in pregnancy is unknown and may vary for women of different ages, races, and parity. Reports in the literature also differ in their definition of short sleep (range, 5–8 h). Emerging evidence suggests that short sleep duration can impact maternal glucose levels [66]. Therefore, it is plausible that short sleep may ultimately impact fetal outcomes through a variety of mechanisms.

We reviewed 17 studies that reported sleep duration and fetal outcome (Table 3). None of these used a consistent definition of short sleep duration (range, 5–8 h) and all used subjective, self-report, rather than objective (actigraphy) measures. Most of the prospective studies reported either birth weight or growth as outcome measures [17,18,33,67–71], while three case–control studies reported on stillbirth as the outcome measure [44,72,73].

Birth weight

In a prospective study of 32 depressed and 136 non-depressed women, depressed women who were short sleepers at 30 weeks (<7 h in bed compared to those with >9 h in bed) had smaller babies [69]. A prospective study of n = 739 women found that sleeping 8 h or less was associated with an increase in LBW (aOR2.8, 95% CI 1.5–5.4) [68]. Conversely, Rabkin and colleagues found that women who reported sleeping ≥9 h per night during the second and third trimester, had slightly increased mean birth weights with a linear trend for increased weight evident from the second trimester (p = 0.04; n = 1507) [70]. Of the two cross-sectional studies, one study of n = 220 women showed no association between short sleep and LBW [18] and the other of n = 457 women found that sleeping <8 h was associated with birth weight >3500g [71].

In summary, few studies have reported sleep duration and association with birth weight. Although caution is needed with interpretation, given the small number of studies available, all except one [71], suggest that short sleep duration is associated with lower birth weight.

Fetal growth

One study of over 1000 Greek women reported no association between sleep duration and LBW or FGR [17]. However, a Sri Lankan study of n = 690 women indicated that sleeping ≤8 h per night was not associated with SGA <10th centile in an adjusted model but was associated with SGA <5th centile after adjustment (aOR 2.2; 95% CI 1.1–4.6; p = 0.03) [67]. More recently, in a study of n = 633 women, no relationship was found between short sleep (≤6 h; n = 23) or long sleep (≥9 h; n155) and fetal growth [33]. Another recent study allocated 200 French women into three sub groups to describe the changing 'trajectory' of sleep duration over the course of the pregnancy i.e., 'short-decreasing (<6.5h/night, 10.8% of the sample), medium-decreasing (6.5–8h/night, 57.6%), and long-increasing (>8h/night, 31.6%) and reported that birth weight-z-score was lower in the long-increasing trajectory group [74]. While not finding a statistically significant difference in LBW, one Chinese cohort study reported a difference in birth length in those participants who said they slept for less than 7 h per day (<7 h/day group decreased by 2.42 mm) [75].

In summary, the literature on sleep duration and fetal growth is limited to a handful of studies, none using actigraphy to measure sleep, and most of which do not support a relationship and thus further work in this area is needed.

Preterm birth

Only five prospective studies included PTB as an outcome; in a study of over 1000 women, sleeping ≤5 h was associated with PTB (aRR 1.7, 95% CI 1.1–2.8) with the highest risk observed for medically indicated PTB after adjusting for covariates (aRR 2.4 95% CI 1.0–6.4) [17]. A recent Chinese study of n = 688 women found that women who reported having <7 h of sleep per night were more likely to have PTB (aOR 4.7, 95%CI 1.2–17.5) [76]. The French study mentioned above [74] found those in the 'short-decreasing' sleep duration trajectory group were more at risk for PTB, The other two studies (both from the same authors and both having some of the smallest sample sizes) explored depression in pregnancy, however when reporting PTB as an outcome neither study reported differences between women who spent <7 h, compared to those who spent >9 h in bed [69,77]. In the two case–control studies of approximately 1000 women each, both of which had PTB rather than sleep duration as the exposure, one reported that PTB was associated with short sleep ≤6 h (aOR 1.6 95% CI 1.1–2.2) [78]. In this study there was also a tendency for the OR for PTB to increase in long sleepers (≥10 h/night) compared to the reference group (average sleep 8 h per night (aOR 1.3, 95% CI 0.9–1.9)). The other larger case-controlled study did not find an association using sleep duration ≤7 h, although the authors stated that there were not enough women sleeping <6 h per night to fully analyse the data [79]. Of the cross-sectional studies examining sleep duration and fetal outcomes, one excluded those born preterm [71] and the other relatively small study of 220 women did not find any differences in PTB between women sleeping ≤6 h per night compared to those sleeping ≥ 10 h per night [18].

In summary, although the studies of sleep duration and PTB are relatively few, most do indeed suggest a relationship. Objective measures of duration, using actigraphy, in large prospective cohorts would help to clarify this relationship.

Stillbirth

Four studies have reported investigating sleep duration and risk of stillbirth. One did not find any association [18] but the other three case-control studies each indicated increased odds of stillbirth in cases who reported sleep shorter than 5:49 h (aOR 1.83, 95% CI 1.24–2.68) and 6 h (aOR 1.81, 95% CI 1.14 to 2.88) respectively [72,73] or longer than 8 h per night in late pregnancy (aOR 1.83 95% CI 1.14–2.94) [44].

Practice points and research agenda for sleep duration and fetal outcome

It is clear that evidence regarding any association between sleep duration and fetal outcome is scant and those studies that do exist reported wide variation in results likely due to differing definitions of sleep duration as well as use of subjective measures. While meta-analysis (Fig. 5) indicates overall significance for PTB and growth, this wide variation does significantly limit the ability to draw any firm conclusions from the currently available literature and points to the need for further research.

Sleep quality and fetal outcome

Sleep quality is a commonly-reported measure, albeit rather subjective. It is well known that poor sleep quality is common in pregnancy and intricately linked with daytime functioning and depressive symptoms, yet little research has investigated the impact of poor sleep quality on fetal wellbeing.

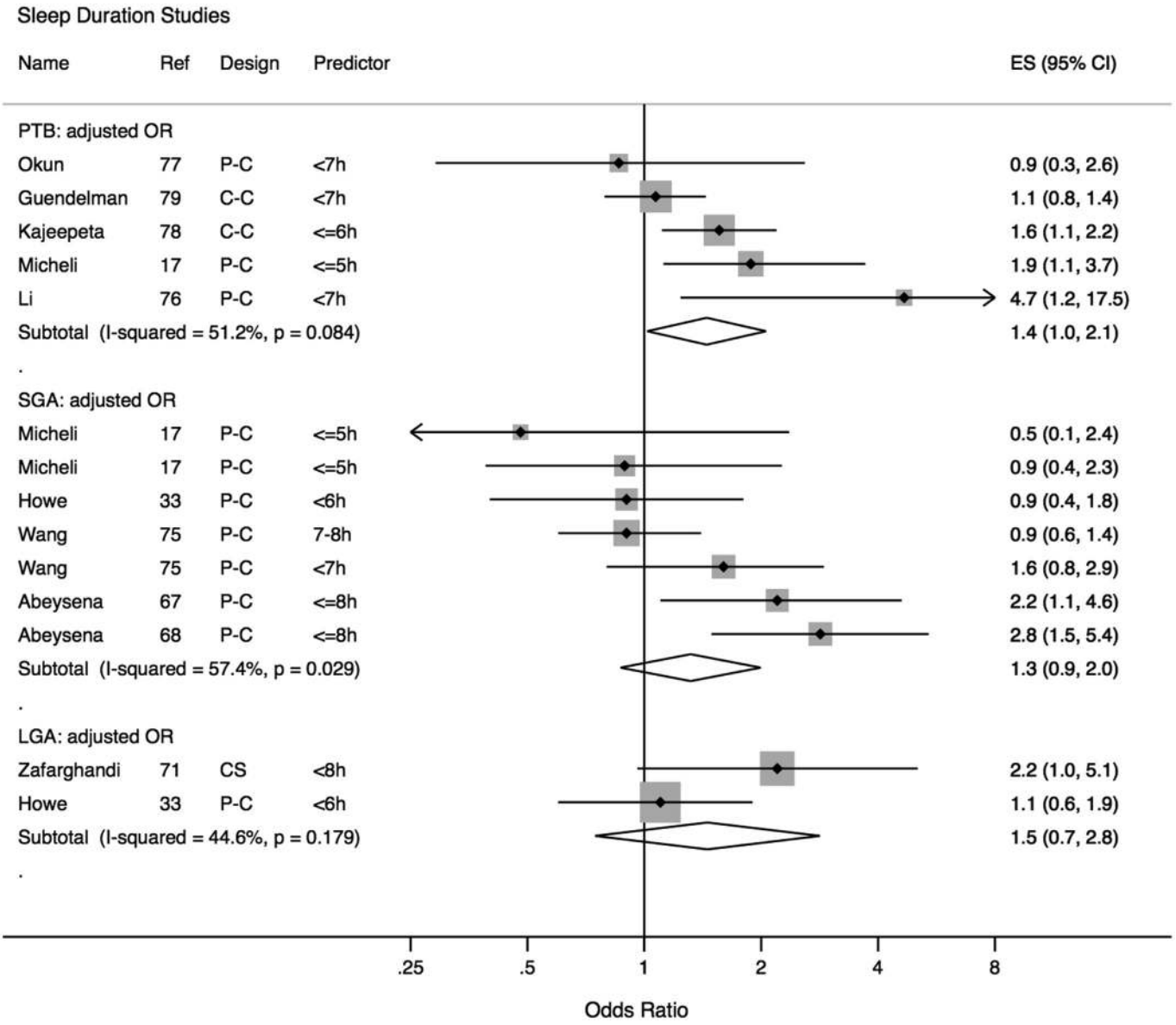

Fig. 5. Sleep Duration, PTB and Growth (studies from Table 3). Black markers represent odds ratios with 95% confidence intervals (whiskers). The size of each grey square represents the relative weight in the random effects meta-analysis. Studies are grouped by those presenting adjusted versus unadjusted estimates. Studies are also grouped by dependent variable: PTB = studies examining pre-term birth; SGA = studies examining small for gestational age, otherwise referred to in articles as low birth weight, intrauterine growth restriction or fetal growth restriction; LGA = studies examining large for gestational age. Diamonds represent OR summary values for PTB and LGA adjusted studies separately. I2 describes the degree of inconsistency across study results (percentage of total variation across studies that is due to heterogeneity rather than chance). As an interpretation guide - 0–30% would indicate a relatively small effect, 30–50% a moderate effect, and >50% a large effect [105]. Studies are ordered by effect size (smallest to largest). Left columns provide primary author and year, primary predictor variable and study design. PeC = prospective cohort; CS = cross-sectional; C–C = case control. Right columns provide OR (95% confidence intervals). Note: Given the variation in variables and study designs, OR summaries should be interpreted with caution. Adjusted OR values not reported in the article were converted from reported adjusted relative risks and sample information.

Sleep disruption during pregnancy is a well-recognised phenomenon, although few longitudinal studies of objective measures exist. Factors such as parity and gestational length may also impact on sleep quality. For example, Signal et al. [80] demonstrated that nulliparous women generally had less efficient sleep, spent more time in bed and had greater wake after sleep onset during the second trimester than their multiparous counterparts and Wilson and colleagues reported that the third trimester of pregnancy is characterised by decreased sleep efficiency, more awakenings and less deep sleep [81]. As all of these can negatively impact on maternal pregnancy outcomes [4], there is also potential for them to impact fetal outcomes.

We identified 14 studies that reported aspects of sleep quality and fetal outcomes (Table 4). The most common scale used to identify poor sleep quality was the Pittsburgh sleep quality index (PSQI) [82], which was utilised in eight studies [23,76,83–88]. The general sleep disturbance scale [89] was used in three studies [18,33,90] and the insomnia severity index [91] in one study [92], with the remaining two studies using either “disturbed sleep” [93] or “non-refreshing sleep” [71] as their indictor for sleep quality.

Birth weight/Fetal growth

Few studies investigated birth weight and/or fetal growth in women with poor sleep quality; of the eight studies that did, none demonstrated clear evidence for an association [18,23,33,71,84,85,87,92]. In the study by Rajendiran et al., no

differences in birth weight between those with and without poor sleep was noted; however, in a sub analysis worse sleep (PSQI score >18) had lower birth weight (2.6 ± 0.4 kg vs. 2.9 ± 0.4 kg, p = 0.02) however, the justification for this sub-analysis of a small number of subjects (n = 14 vs. n = 16) was not clear [87]. Dolation et al. [92] reported a difference in what? between birth weight categories (categorised as <2500g, 2600–3000g, 3001–4000g, and >4000g). They stated that birth weight was lower in the 'sleep disorder' group (p = 0.07) but no mean birth weight data were provided. One study reported a higher frequency of babies born >3500g in women with 'unrefreshed' sleep compared to those refreshed/somewhat refreshed sleep (26% vs. 14%; p = 0.03) [71].

Overall, the data suggest that there is no difference in birth weight or fetal growth with poor maternal sleep quality. However, the largest sample size was n = 633, with most other studies having considerably fewer women. Since sleep quality is inherently subjective and highly prevalent, large longitudinal studies are needed to thoroughly investigate these relationships.

Preterm birth

Of ten studies reporting PTB/gestational age [18,76,83–86,88,90,92,93], four prospective studies reported that poor sleep quality was associated with PTB [76,83,86,92]. This relationship was particularly pronounced in African American women (n = 79) where the odds of PTB were 10-fold higher in those with poor sleep quality compared to those without (OR 10.2 95% CI 1.1, 91.9, p = 0.04), a finding that was not present in a sample of n = 53 European American women [83]. While the confidence intervals were wide in this relatively small sample, the relationship between poor sleep quality and length of gestation was mediated via inflammation. The authors speculated that African Americans could exhibit heightened sensitivity to the adverse physiological sequelae of sleep disturbance. In one study of n = 220 women where the exposure was PTB, data showed that women with PTB had increased sleep latency (26.09 ± 19.91 min vs. 18.53 ± 14.94 min, Cohen's d = 0.43) but no relationship with PSQI scores [88]; the lack of association between sleep quality and PTB was also found in a similarly sized prospective study (n = 220) of which n = 153 women reported poor sleep quality [84]. However, another study of n = 359 women showed '*good*' sleep (subjective definition and unspecified number of women) was associated with both preterm labour and PTB in an unadjusted model, but only with preterm labour in an adjusted model (aOR 2.4, 95% CI 1.1–5.1) [90]. One cross-sectional study of n = 220 women reported gestational age at delivery and demonstrated no differences in women with and without poor sleep quality [18]. A case–control study of n = 258 women, where cases were PTB and controls were full term, reported that PTB was associated with sleep disruptions [93], which were not defined.

In summary, there is a lack of association between poor sleep quality and PTB. However, only one study stratified findings by race and found that an association was present for African American women but not Caucasian women. These preliminary findings are worthy of further study particularly since African American race is known to be associated with increased adverse pregnancy outcomes. Whether there is a differential impact of sleep in fetal well-being in this population remains to be studied.

Stillbirth

Only one study investigated the association between poor sleep quality and stillbirth but did not find an association [18].

Practice points and research agenda for sleep quality and fetal outcome

The current evidence for sleep quality and fetal outcome does not support any clear associations with birth weight or growth in individual studies, and meta-analysis (Fig. 6) of the four studies reporting on this variable was also inconclusive. Some studies reported results suggestive of an effect on PTB, which was seen in the adjusted meta-analysis of 7 studies (Fig. 6). However, the paucity of studies, wide variability in sample sizes, study designs, definitions of poor sleep quality, as well as differences in exposures (e.g., sleep disruption as the exposure vs. PTB as the exposure), make it difficult to understand whether indeed poor sleep quality *per se* impacts fetal outcome. However, it is plausible that the impact of disturbed sleep on fetal outcome may begin in early pregnancy, suggesting that longitudinal studies are necessary for fully delineating any associations. Okun [94] has put forward a hypothesis postulating that disturbed sleep during early pregnancy contributes to an increased inflammatory response or decreased uterine blood blow that may disrupt the normal remodelling of maternal blood vessels that perfuse the placenta and could subsequently result in poor pregnancy outcomes, this may be useful to explore more fully in a prospective study.

Sleep position and fetal outcome

An emerging area of research worldwide is the association between maternal sleep position and poor fetal outcome, specifically fetal growth and stillbirth. It is well known that maternal supine position in late pregnancy is associated with compression of the inferior vena cava [95], and thus such a position is avoided in order to prevent any concomitant fall in cardiac output and maternal blood flow to the placenta [96]. Indeed, it is standard practice to shift a labouring woman onto her left side during acute fetal distress, as this position aids fetal recovery [97]. Furthermore, maternity care providers routinely position women in a left lateral tilt position when they are undergoing obstetric procedures such as ultrasound, caesarean section, and abdominal palpation in order to avoid both maternal and fetal effects. Recommendations have also been made that women with certain conditions such as supine hypotensive syndrome or position dependant SDB should avoid the supine position [98]. Thus, it is well known that if the mother lies supine for a short period when she is awake, there may be an acute negative effect on the fetus.

Despite this, there has been little consideration given to the position that women may be regularly sleeping in every night. Contrary to popular assumption, third trimester women do spend time in the supine sleep position. We have demonstrated that the majority (about 80%) of pregnant women spend some time in the supine position, with approximately 25% of any given night spent sleeping supine [99].This provides evidence that the fetus may be regularly and repetitively exposed to reduced delivery of oxygen and nutrients from the placenta on a nightly basis, and it is therefore plausible that this may result in fetal vulnerability, growth restriction and ultimately fetal demise.

Stillbirth

The Auckland Stillbirth Study, a case–control study in New Zealand [44] sparked worldwide interest and commentary [100,101] when it was reported that women who settled to sleep on their back on the last night of their pregnancy had a two and a half fold increased odds of stillbirth (aOR 2.54; 95% CI 1.04–6.18).

In the ensuing years, five more epidemiological studies have been conducted [18,45,72,73,102]. The largest and most recent of

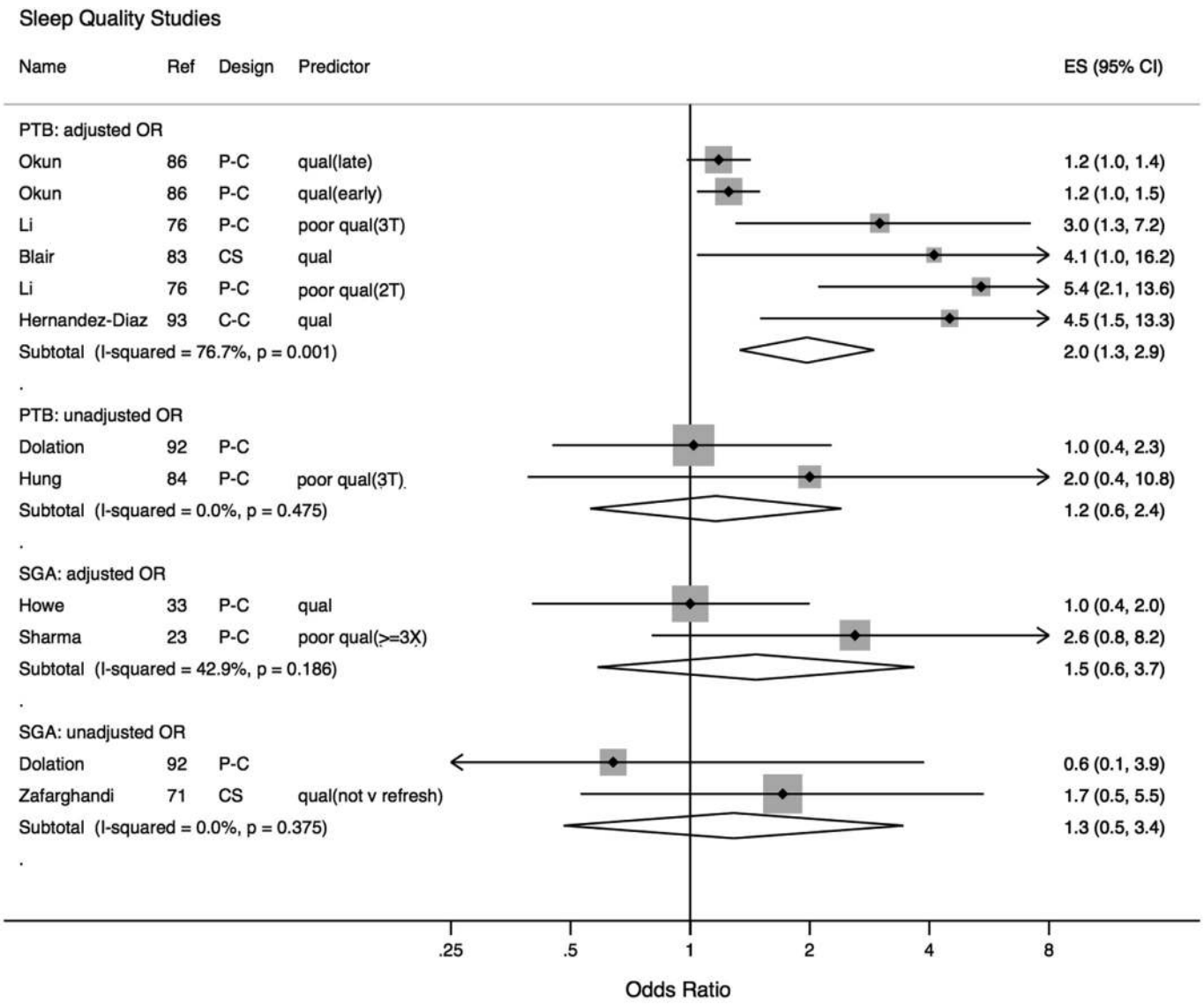

Fig. 6. Sleep Quality, PTB and Growth (studies from Table 4). Black markers represent odds ratios with 95% confidence intervals (whiskers). The size of each grey square represents the relative weight in the random effects meta-analysis. Studies are grouped by those presenting adjusted versus unadjusted estimates. Studies are also grouped by dependent variable: PTB = studies examining pre-term birth; SGA = studies examining small for gestational age, otherwise referred to in articles as low birth weight, intrauterine growth restriction or fetal growth restriction. Diamonds represent OR summary values. I2 describes the degree of inconsistency across study results (percentage of total variation across studies that is due to heterogeneity rather than chance). As an interpretation guide - 0–30% would indicate a relatively small effect, 30–50% a moderate effect, and >50% a large effect [105]. Studies are ordered by effect size (smallest to largest). Left columns provide primary author and reference number, primary predictor variable and study design. Qual = sleep quality; 3T = third trimester; 3X = three times; PeC = prospective cohort; CS = cross-sectional; C–C = case control. Right columns provide OR (95% confidence intervals). Note: Given the variation in variables and study designs, OR summaries should be interpreted with caution. Adjusted OR values not reported in the article were converted from reported adjusted relative risks and sample information. Unadjusted OR values not reported in the article were calculated from counts.

these was a prospective case–control study conducted in 41 centres in the UK [72]. Women who had a stillbirth after ≥28 weeks gestation (n = 291) and women with an ongoing pregnancy (n = 733) were interviewed. Multivariable analysis indicated that the supine going-to-sleep position the night before stillbirth had a 2.3-fold increased risk of late stillbirth (aOR 2.31, 95% CI 1.04–5.11) compared with the left side. Another seven centre study conducted in New Zealand included 164 singleton stillborn and 569 women with on-going singleton pregnancies [73]. This group also reported that supine going-to-sleep position on the last night was associated with increased late stillbirth risk (aOR 3.67; 95% CI 1.74–7.78).

Another cross-sectional study of maternal sleep and pregnancy outcomes in a Ghanaian population [18] is the only study to investigate maternal supine sleep as an exposure and stillbirth as an outcome; and reported an eight fold odds (aOR of 8.0; 95% CI 1.5–43.2; p = 0.016) for stillbirth in women who spent time sleeping supine. Two smaller case–control studies [45,102] also reported increased risk of stillbirth in supine sleepers (aOR 6.26, 95% CI 1.2–34.0) and (aOR 2.95 95% CI 1.5–5.8) respectively.

Fetal growth

Any interaction between supine sleep and fetal growth is currently unclear. Gordon [45] reported that SGA (<10th centile) was over-represented in the supine sleep group but also conceded that her study was not powered to test this interaction. However, Heazell's larger case control study [72]

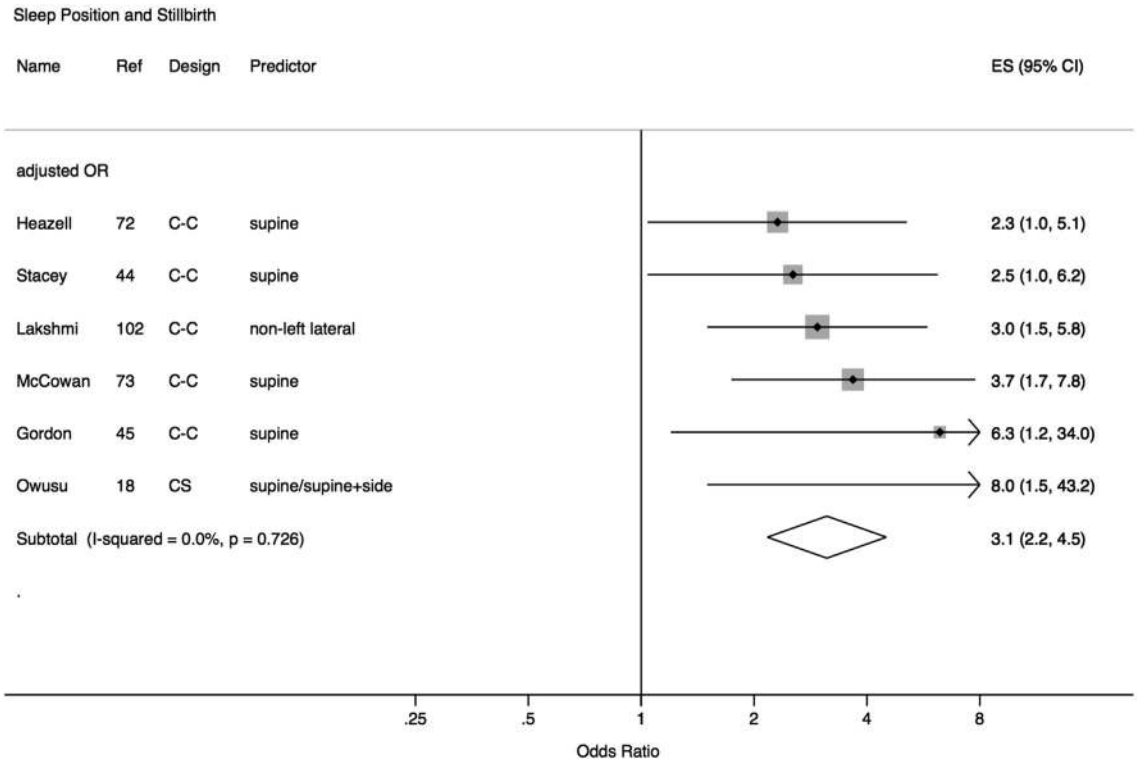

Fig. 7. Sleep Position and Stillbirth (studies from Table 5). Black markers represent odds ratios with 95% confidence intervals (whiskers). The size of each grey square represents the relative weight in the random effects meta-analysis. The diamonds represents the OR summary value. I2 describes the degree of inconsistency across study results (percentage of total variation across studies that is due to heterogeneity rather than chance). As an interpretation guide - 0–30% would indicate a relatively small effect, 30–50% a moderate effect, and >50% a large effect [105]. Studies are ordered by effect size (smallest to largest). Left columns provide primary author and year, primary predictor variable and study design. CS = cross-sectional; C–C = case control. Right columns provide OR (95% confidence intervals). Note: Given the variation in variables and study designs, OR summaries should be interpreted with caution. Adjusted OR values not reported in the article were converted from reported adjusted relative risks and sample information. Unadjusted OR values not reported in the article were calculated from counts.

reported no detectable interaction between the effect of supine going-to-sleep position and a SGA baby (p for interaction = 0.44). Nevertheless, the cross sectional study [18] indicated that the risk of supine sleep was mediated by the association of supine sleep with LBW since its addition to the model eliminated the statistically significant relationship between supine sleep and stillbirth (aOR 4.9; 95% CI 0.80–31.4; p = 0.09). Notably, pregnancy outcomes were not known to the investigators at the time of enrolment, thus minimising the likelihood of bias.

Preterm birth

While none of these studies has reported increased risk of preterm birth associated with the supine going to sleep position, McCowan et al. [73] reported that the risk associated with supine-going-to-sleep position was greater for term (aOR 10.26; 3.00 to 35.04) than preterm stillbirths (aOR 3.12; 0.97 to 10.05). This is biologically plausible, especially if the mechanism for fetal compromise is venal caval compression, as this compression would naturally increase as the pregnancy advances.

Practice points and research agenda for maternal sleep position and fetal outcome

Summary analysis of the reported studies to date (Fig. 7) indicates an association between supine sleep and stillbirth, which together suggests that avoiding the supine going to sleep position may be a simple means to modify risk for stillbirth. In fact the population attributable risk from all these studies ranges between 4 and 37% [18,44,72,73] meaning that if indeed maternal supine sleep plays a causal role in late stillbirth, between 4 and 37% of stillbirths could be prevented by the avoidance of supine sleep. An IPD analysis of all the case–control studies is currently underway [103] and will provide further support for the role of supine sleep on pregnancy outcome.

In summary, findings from studies published to date suggest that there may be an association between events occurring during maternal sleep and fetal well-being, particularly if the fetus is already vulnerable [104]. Such association may include maternal oxygen desaturation from SDB occurring during supine sleep, in turn leading to regular nightly insults on placental perfusion. This may result in bouts of fetal hypoxemia and thus fetal compromise leading to FGR and ultimately fetal death. Clinicians should be aware of this emerging area of research as advice to pregnant women to settle to sleep on their side in late pregnancy seems warranted.

Limitations

In addition to the study limitations detailed in the sections above, a general limitation of this scoping review is the exclusion of non-English studies. Thus, the potential for cultural differences in how sleep and fetal outcomes may be related might not have been addressed.

Overall practice points

- This scoping review of available literature suggests that several aspects of maternal sleep may impact key fetal outcome measures.
- Clinicians should be aware that although findings were not consistent, in general there was evidence that OSA, short sleep duration, and poor sleep quality may be associated with PTB and perhaps also stillbirth.
- Findings were conflicting with regards to birth weight/growth but emerging data suggests that one measure of weight at delivery may not be an appropriate outcome; rather growth trajectory measures will likely be more informative.
- There is minimal literature regarding maternal sleep and stillbirth, yet the few available studies all point to a role for supine sleep position in this devastating outcome.
- In many cases, conclusions were difficult to draw because each study used slightly different definitions of FGR and PTB, thus careful attention must be paid to future study design.

Research agenda

- Since many aspects of maternal sleep are modifiable and a number of studies do suggest associations with poor fetal outcomes, priority should be given for research with large, adequately powered studies utilising standardised definitions of maternal sleep disruption and fetal/newborn health.
- This is an area ripe for investigation if there is to be reduction in the physical, emotional, and financial burden of poor fetal outcomes.

Conflicts of interest

The authors do not have any conflicts of interest to disclose.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.smrv.2018.03.004.

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