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### Authors

Bartels, Helena C
Kennelly, Maria A
Killeen, Sarah Louise
et al.



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RESEARCH ARTICLE

Maternal Medicine

# An mHealth-Supported antenatal lifestyle intervention may be associated with improved maternal sleep in pregnancy: Secondary analysis from the PEARS trial

Helena C. Bartels[1] | Maria A. Kennelly[1] | Sarah Louise Killeen[1] | Karen L. Lindsay[1,2] | Rachel K. Crowley[1,3] | Fionnuala M. McAuliffe[1]

[1]UCD Perinatal Research Centre, School of Medicine, University College Dublin, National Maternity Hospital, Dublin, Ireland

[2]UCI Department of Pediatrics, University of California, Irvine, CA, USA

[3]Department of Endocrinology, St Vincent's University Hospital, Dublin, Ireland

**Correspondence**
FM McAuliffe, Obstetrics & Gynaecology, University College Dublin, Head, Women's and Children's Health, University College Dublin, University College Dublin, National Maternity Hospital, Dublin, Ireland.
Email: fionnuala.mcauliffe@ucd.ie

## Abstract

**Objective:** To investigate the effect of an antenatal diet and exercise intervention during pregnancy on sleep duration. As a secondary objective, associations between sleep duration and gestational weight gain (GWG), maternal metabolic parameters and pregnancy outcomes were assessed.

**Design:** Secondary analysis.

**Setting:** Large tertiary Maternity Hospital in Dublin, Ireland.

**Population:** 326 women with overweight or obesity who participated in the Pregnancy Exercise And Nutrition Research Study (PEARS) randomised controlled trial between March 2013 and August 2016.

**Methods:** Secondary analysis of a randomised trial.

**Main outcome measures:** Impact of the PEARS intervention on sleep duration, and association of sleep duration and maternal metabolic parameters, and pregnancy outcomes.

**Results:** Participants had a mean age of 32.5 ± 4.5 years and median (interquartile range [IQR]) body mass index of 28.3 (26.6–31.2) kg/m$^2$. The intervention group had a longer sleep duration in late pregnancy (mean difference 17.1 minutes (95% confidence interval [CI] 0.5–33.7) and a higher proportion achieving optimum sleep duration of 7–9 h (54.3 vs. 42.9%, relative risk [RR] 1.28 (95% CI 1.01–1.62). In late pregnancy, sleep duration of <6 h was associated with lower breastfeeding rates on discharge (RR 0.74, 95% CI 0.57–0.95) and higher triglyceride levels (mean difference 0.24, 95% CI 0.10–0.38). There were no significant associations between sleep and incidence of gestational diabetes mellitus or pre-eclampsia/toxaemia, or other metabolic parameters assessed (insulin, fasting glucose, HOMA-IR).

**Conclusion:** A diet and exercise intervention from early pregnancy may promote longer and optimal sleep duration, with maternal benefits such as lower triglyceride levels and higher breastfeeding rates.

This research was carried out at the UCD Perinatal Research Centre at the National Maternity Hospital, Dublin 2, Ireland.

**KEYWORDS**
breastfeeding, nutrition, obesity, obstetrics, pregnancy, sleep, triglycerides, metabolism, weight

## 1 | INTRODUCTION

Good sleep behaviours are crucial components and contributors to general health and well-being.[1] Sufficient sleep duration for adults aged between 25 and 64 years has been defined by the National Sleep Foundation as between 7 and 9 per night.[2] In pregnancy, up to 80% of women have reported sleep disturbance which is present from early pregnancy and worsens as gestation advances.[3,4] Physiological factors such as backache, fetal movements and urinary frequency play a part,[5] as well as hormonal factors such as increased circulation of sex steroid hormones,[6] which result in alterations in respiratory function and thermogenesis, thus interfering with sleep.[7]

Suboptimal sleep behaviours can exert adverse effects on both maternal and fetal health. There is a direct association between short sleep duration and increased body mass index (BMI)[8,9] hypertensive disorders, impaired glycaemic control[10,11] and gestational diabetes mellitus (GDM).[6] Perinatal complications related to poor maternal sleep quality and duration include preterm birth and fetal growth restriction.[12]

Gestation weight gain (GWG) guidelines were defined by the Institute of Medicine and updated in 2009.[13] Both excess and inadequate GWG have been associated with adverse maternal and fetal outcomes, including small-for-gestational age, GDM, pre-eclampsia and offspring obesity.[14–16] Data relating to sleep quality and duration and GWG have been conflicting. Both long and short sleep duration have been associated with excess GWG,[17,18] with conflicting results relating to sleep quality and the risk of not meeting GWG criteria in pregnancy.[19,20]

This is a secondary analysis of participants recruited as part of the Pregnancy Exercise And Nutrition Research Study (PEARS) study, a randomised control trial investigating the impact of a behavioural diet and exercise intervention on the prevention of GDM in pregnant women with overweight or obesity.[21] The primary objective of this study is to explore the impact of a healthy lifestyle intervention on maternal sleep duration and quality, explore to, as a secondary objective, associations with maternal factors including GWG, metabolic parameters and breastfeeding.

## 2 | MATERIAL AND METHODS

This is a secondary analysis of participants recruited as part of the PEARS study.[21] The PEARS study (ISRCTN registry, https://www.isrctn.com/, ISRCTN29316280) was conducted between March 2013 and August 2016 with institutional ethical approval from the National Maternity Hospital and written maternal consent. This was a randomised controlled trial of a mobile health (mHealth) behavioural lifestyle intervention with smartphone app support to prevent GDM in pregnant women with overweight or obesity. Details of the study protocol and cost analysis have been published previously.[21,22] The primary outcome of the trial was the incidence of GDM diagnosed per the International Association of Diabetes in Pregnancy Study Groups criteria at 28–30 weeks' gestation.[21] Although the intervention had no effect on the diagnosis of GDM between the intervention and control group (relative risk [RR] 1.1, 95% confidence interval [CI] 0.71–1.66), $p = 0.71$), it resulted in lower dietary glycaemic index, greater exercise participation and less large-for-gestational-age infants.[21] Further details of the trial results have been published elsewhere.[23] In this analysis, we used data collected as secondary outcomes in the PEARS trial to explore the potential effect of the PEARS intervention on sleep behaviours. There was no patient or public involvement in this secondary analysis.

### 2.1 | Study sample

The primary objective of this study was to assess the impact of the PEARS intervention on sleep duration and quality. Women with sleep data available at baseline (14–16 weeks) and post-intervention (28 weeks) were included. For the secondary objective, where maternal sleep duration was compared with maternal metabolic markers, meeting GWG targets and breastfeeding, women with sleep data plus corresponding laboratory assessments of maternal metabolism (plasma glucose, insulin resistance, lipids from maternal blood) and pregnancy outcomes were included.

### 2.2 | Demographics

Maternal weight was measured at recruitment, 28 and 34 weeks, and the last recorded weight after 36 weeks' gestation was abstracted from medical charts to compute total gestational weight gain. Data pertaining to demographics, antenatal and delivery outcomes were recorded in the patient's medical chart. Breastfeeding refers to mothers who were breastfeeding on discharge.

### 2.3 | Maternal sleep

Aspects of sleep quality were assessed including sleep duration and sleep disordered breathing using combined aspects of the Berlin questionnaire[24] and the Pittsburgh Sleep Quality Index,[25] both of which have been validated in a pregnant population.[26] For the purpose of this study,

moderate sleep restriction was defined as <7 hours' sleep per night, severe sleep restriction as <6 hours' sleep per night, and optimum sleep duration as 7–9 hours' sleep per night.

## 2.4 | Metabolic health markers

At recruitment (14–16 weeks' gestation) and 28 weeks' gestation, all women had fasting blood samples collected. Fasting serum glucose was analysed following centrifugation by hospital laboratory staff at the shortest possible interval following sample collection using the AU680 Chemistry analyser (Beckman Coulter Inc., High Wycombe, UK) by the hexokinase method. Insulin was quantified by automated immune-assay (Roche Cobas 602; Roche Diagnostics) with typical coefficient of variations <5%. Total cholesterol, HDL cholesterol, and triglycerides were analysed on a Roche Cobas 702 analyser (Roche Diagnostics). LDL cholesterol levels were estimated using the equation of Friedewald et al.[27] Insulin resistance was assessed using the Homeostasis Model Assessment 2 (HOMA2-IR) index, using the programme HOMA CALCULATOR v2.2.2.[28]

## 2.5 | Statistical analysis

Data were graphically assessed for normality using analysis of mean, median, skewness, kurtosis and Kolmogorov–Smirnoff test. Skewed data were log10-transformed prior to analysis. Independent sample *t*-tests were used to compare continuous data between the intervention and control study groups, and between moderately and severely restricted sleep groups. Using epi.2by2 library in R software, RR and 95% CI were calculated between categorical variables. Chi-square tests were used to compare categorical data between the study groups. Associations between sleep duration and pregnancy outcomes (GDM, pre-eclampsia, breastfeeding) and metabolic parameters were compared using Pearson's correlation. Where significant differences were found, these were further analysed using multivariate regression analysis and the standardised Beta coefficients were reported. The linear regressions were controlled for known confounders for cardiometabolic health including maternal age, ethnicity, education, BMI, and control group status. A two-tailed *p*-value of <0.05 was considered significant. All statistical analyses were performed using IBM SPSS software for Windows version 27.0 (SPSS Inc.).

# 3 | RESULTS

A total of 326 women were included in the analysis who had sleep data available at baseline and post intervention, and corresponding metabolic parameters (Figure S1 flow diagram). The baseline characteristics of the cohort and sleep duration, quality and self-reported disturbed breathing at baseline are presented in Table 1. The mean age of the entire group at study entry was 32.5 ± 4.5 years and the median (interquartile range, IQR) BMI of 28.3 (26.6–31.2) kg/m$^2$. There were no differences in maternal characteristics, sleep duration, self-reported sleep quality, sleep disturbed breathing or Metabolic Equivalent of Tasks (METs) between intervention and control groups at baseline (Table 1).

**TABLE 1** Maternal characteristics and sleep data at baseline (14–16 weeks' gestation)

| | Intervention, *N* = 151 | Control, *N* = 175 |
|---|---|---|
| Age mean ± SD | 32.8 ± 4.5 | 32.2 ± 4.1 |
| Primiparous | 78 (51.6) | 98 (56.0) |
| BMI, kg/m$^2$ | 29.4 ± 3.5 | 28.9 ± 3. |
| Education | | |
| Completed 3rd level, (%) | 82 (54.3) | 109 (62.2) |
| METS (minutes), mean (SD) | 537 (407) | 532 (395) |
| Sleep data | | |
| Sleep duration, min | 433.8 ± 76 | 441 ± 77 |
| Sleep quality | | |
| Very good | 25 (16.4) | 25 (14.5) |
| Fairly good | 87 (57.5) | 99 (56.8) |
| Fairly bad | 34 (22.4) | 44.6 (25.5) |
| Very bad | 5 (3.7) | 5.6 (3.2) |
| Snore loudly | | |
| Most nights | 15 (10) | 13 (7.1) |
| 3 or more times per week | 16 (11.3) | 18 (10.3) |
| 1–2 times per week | 18 (12.3) | 25 (14.6) |
| Less than once per week | 22 (15.2) | 27 (15.6) |
| Never or nearly never | 77 (51.2) | 91 (52.4) |

*Note*: Data presented as *n* (%) unless otherwise stated. Baseline data shown for participants who had both baseline and post-intervention sleep data available. METS, metabolic equivalent of tasks (minutes).

## 3.1 | Effects of the PEARS intervention on sleep

Results comparing sleep post intervention are presented in Table 2. At 28 weeks, both groups experienced a decrease in mean sleep time compared with their baseline assessment, but mean sleep duration was significantly higher in the intervention group than in controls (mean difference 17.1 minutes, 95% CI 0.5–33.7; $p = 0.04$). More women in the intervention group achieved the 'optimum sleep duration' of 7–9 h per night (RR 1.28, 95% CI 1.01–1.62; $p = 0.04$). No effects of the intervention were detected on overall sleep quality or frequency of maternal snoring (Table 2). There was no difference in the METs between groups (mean difference 27.1 (95% CI 58.8–113.1; $p = 0.53$).

## 3.2 | Association of sleep duration with Institute of Medicine (IOM) gestational weight gain guidelines

Tables 3 and 4 demonstrate the percentage of women meeting IOM GWG guidelines based on optimum sleep duration in early and late pregnancy, respectively.

In early pregnancy, women in the moderately <7 h (52.4 vs. 67.4%, RR 1.38, 95% CI 1.05–1.80; $p = 0.02$) and severely <6 h (46.4% vs. 64%, RR 1.18 [95% CI 1.02, 1.36], $p = 0.01$) sleep-deprived groups were less likely to exceed GWG guidelines than were women with greater sleep attainment. In unadjusted logistic regression, moderate sleep restriction <7 h in early pregnancy was associated with reduced odds of exceeding GWG guidelines (β = 1.88, 95% CI 1.12–3.13; $p = 0.015$). This association remained when controlled for confounders (β = 1.72, 95% CI 1.03–2.91; $p = 0.04$). For severe sleep restriction <6 h in early pregnancy, in unadjusted logistic regression this was associated with reduced odds of exceeding GWG guidelines (β = 0.488, 95% CI 0.268–0.890; $p = 0.02$). This association remained when controlled for confounders (β = 0.508, 95% CI 0.270–0.920; $p = 0.03$). More women who were severely sleep-deprived in early pregnancy (sleep duration <6 h) had inadequate GWG (25% vs. 12.2%, RR 1.89, 95% CI 1.15–3.10; $p = 0.01$), compared with women with >6 h sleep duration. In unadjusted logistic regression, severe sleep restriction (<6 h) in early pregnancy was associated with an increased odds of inadequate GWG (β = 2.40, 95% CI 1.14–5.03; $p = 0.02$). This association remained when controlled for confounders (β = 2.22, 95% CI 1.03–4.82; $p = 0.04$). There was no difference in rates of inadequate GWG when stratified by moderate sleep cut-offs (<7 h; $p = 0.06$).

In late pregnancy, there was no difference in rates of inadequate GWG or exceeding GWG guidelines when stratified by either moderate or severe sleep restriction.

**TABLE 2** Sleep data post PEARS intervention at 28 weeks

| | Mean difference (95% CI) | *p*-value |
|---|---|---|
| Sleep duration at 28 weeks (minutes) | 17.1 (0.51–33.7) | 0.04 |
| Change in sleep duration from baseline | 16.1 (−1.1 to 33.3) | 0.06 |
| | **Relative risk (95% CI)** | ***p*-value*** |
| Optimum sleep (7–9 h/night) | 1.28 (1.01, 1.62) | 0.04 |
| Sleep quality | 1.17 (0.93, 1.48) | 0.16 |
| Snore loudly | 1.00 (0.81, 1.24) | 0.99 |

The table shows the mean differences (95% CI) and relative risks (95% CI) between the intervention ($n = 151$) and control group ($n = 175$) for sleep duration and quality. For sleep quality, two variables were created comparing participants who rated their sleep as 'very to fairly good' with those with 'very to fairly bad' sleep between the intervention and control. For snoring, two variables were created comparing participants who reported snoring 'most nights to 1–2 per week' with those who reported snoring 'less than 1x per week to never' between the intervention and control group.

*Chi-square.

## 3.3 | Association of sleep duration with metabolic parameters

Tables 3 and 4 demonstrate the differences in IOM GWG adherence and metabolic parameters stratified by moderate and severe sleep restriction in early and late pregnancy.

**TABLE 3** Maternal IOM GWG adherence and metabolic parameters in women comparing optimum sleep duration in early pregnancy (14–16 weeks' gestation)

| | Relative risk (95% CI) between </>7 h slept | *p*-value | Relative risk (95% CI) between </>6 h slept | *p*-value |
|---|---|---|---|---|
| Exceeded IOM GWG guidelines | 1.38 (1.05–1.80) | 0.02* | 1.18 (1.02–1.36) | 0.01* |
| Did not meet IOM GWG guidelines | 1.36 (1.02–1.80) | 0.06 | 1.89 (1.15–3.10) | 0.01* |
| | **Mean difference (95% CI) between < and >7 h slept** | | **Mean difference (95% CI) between < and >6 h slept** | |
| BMI early**, kg/m$^2$ | 0.71 (0.05–1.44) | 0.03 | 0.01 (−0.01 to 0.02) | 0.06 |
| METS (minutes) | 55.2 (11.0–228.7) | 0.03 | 119.5 (2.5–236.4) | 0.05 |
| Glucose fasting, mmol/l | 0.06 (−0.01 to 0.14) | 0.08 | 0.08 (−0.01 to 0.17) | 0.08 |
| Insulin,** mmol/l | 0.02 (−0.01 to 0.06) | 0.17 | 0.04 (−0.06 to 0.09) | 0.08 |
| HOMA2-IR | 0.81 (0.12–1.10) | 0.05 | 1.01 (0.32–1.20) | 0.03* |
| TC, mmol/l | 0.02 (−0.16 to 0.20) | 0.89 | 0.09 (−0.12 to 0.31) | 0.39 |
| Triglycerides,** mmol/l | 0.01 (−0.01 to 0.04) | 0.40 | 0.03 (−0.02 to 0.21) | 0.12 |
| HDL, mmol/l | 0.08 (0.06–0.17) | 0.05 | −0.07 (−0.18 to 0.03) | 0.17 |
| LDL, mmol/l** | 0.01 (−0.1 to 0.03) | 0.38 | 0.12 (−0.86 to 0.34) | 0.42 |

*Note*: *p*-values calculated by Chi-square for relative risk and independent *t*-test for mean difference. *All significant differences are adjusted for covariates. **Log-transformed variables used to determine *p*-values.

BMI, body mass index; HDL, high density lipoprotein; HOMA2-IR, homoeostatic model assessment; LDL, low density lipoprotein; METS, metabolic equivalent of tasks (minutes); TC, total cholesterol.

**TABLE 4** Maternal IOM GWG adherence and metabolic parameters in women comparing optimum sleep duration in late pregnancy (28 weeks)

| | Relative risk (95% CI) between </>7 h slept | *p*-value | Relative risk (95% CI) between </>6 h slept | *p*-value |
|---|---|---|---|---|
| Exceeded GWG guidelines | 0.64 (0.40–1.02) | 0.06 | 0.78 (0.61–1.01) | 0.06 |
| Did not meet GWG guidelines | 0.90 (0.70–1.15) | 0.36 | 1.16 (0.81–1.66) | 0.44 |
| | **Mean difference (95% CI) between </>7 h** | | **Mean difference (95% CI) between </>6 h** | |
| BMI late**, kg/m$^2$ | 0.01 (−0.01, 0.02) | 0.38 | 0.01 (−0.01, 0.02) | 0.15 |
| METS (minutes) | 56.4 (−79.5 to 192.4) | 0.41 | 54.2 (−61.8 to 170.4) | 0.35 |
| Glucose fasting, mmol/l | 0.04 (−0.05 to 0.15) | 0.38 | 0.05 (−0.03 to 0.15) | 0.22 |
| Insulin,** mmol/l | −0.02 (−0.05 to 0.04) | 0.92 | 0.01 (−0.02 to 0.06) | 0.49 |
| HOMA2-IR | 0.02 (−0.12 to 0.34) | 0.15 | 0.11 (−0.01 to 0.34) | 0.57 |
| TC, mmol/l | 0.04 (−0.2 to 0.35) | 0.82 | 0.24 (−0.03 to 0.53) | 0.08 |
| Triglycerides, mmol/l | 0.09 (0.08–0.02) | 0.04* | 0.24 (0.10–0.38) | <0.001* |
| HDL, mmol/l | 0.01 (−0.11 to 0.15) | 0.76 | −0.4 (−0.16 to 0.07) | 0.42 |
| LDL, mmol/l | 0.12 (−0.30 to 0.33) | 0.93 | 0.18 (−0.9 to 0.46) | 0.19 |
| GDM | 0.99 (0.07–1.41) | 0.96 | 1.14 (0.81–1.60) | 0.47 |
| PET or PIH | 1.20 (0.84–1.71) | 0.37 | 1.10 (0.69–1.76) | 0.82 |
| Breastfeeding | 0.95 (0.75–1.20) | 0.64 | 0.74 (0.57–0.95) | 0.02* |

*Note*: *p*-values calculated by Chi-square for relative risk and independent *t*-test for mean difference. *All significant differences are adjusted for covariates.**Log-transformed variables used to determine *p*-values.

BMI, body mass index; HDL, high density lipoprotein; HOMA2-IR, homoeostatic model assessment; LDL, low density lipoprotein; METS, metabolic equivalent of tasks (minutes); PET, pre-eclampsia/toxaemia; PIH, pregnancy-induced hypertension; TC, total cholesterol.

Maternal IOM GWG adherence and metabolic parameters in women comparing optimum sleep duration in late pregnancy (28 weeks) are shown in Table 4. Women in the severely (<6 h) and moderately (<7 h) restricted sleep groups had significantly higher triglycerides compared with their reference groups (mean difference 0.24 (95% CI 0.10–0.38, $p = 0.001$) and mean difference 0.09 (95% CI 0.08–0.02, $p = 0.04$), respectively.

Correlations performed comparing total sleep duration and maternal metabolic parameters found no significant associations (all *p*-values <0.05) except for total sleep duration at 28 weeks and triglycerides (−0.13, $p = 0.02$). This was further explored with linear regression using the confounders as described above, and whereas results were statistically significant for longer sleep duration associated with lower triglycerides, the differences in triglyceride were minimal ($\beta = -0.001$, 95% CI −0.002 to −0.001; $p < 0.01$).

Breastfeeding rates on discharge from hospital were significantly lower in women who were severely sleep-restricted in late pregnancy (44.3% vs. 58%, RR 0.74, 95% CI 0.57–0.95; $p = 0.02$). There were no associations between women achieving optimum sleep duration and the incidence of GDM or pregnancy-related hypertensive disease (Table 4).

# 4 | DISCUSSION

## 4.1 | Main findings

This secondary analysis examined the effects of a lifestyle intervention on maternal sleep behaviours. Our findings show that an healthy antenatal lifestyle intervention for pregnant women with overweight and obesity improved maternal sleep duration at 28 weeks' gestation. Further exploratory analysis suggests that restricted sleep may influence gestational weight gain, insulin resistance, triglyceride concentrations and breast-feeding initiation rates.

## 4.2 | Strengths and limitations

This was a well characterised cohort of pregnant women with a raised BMI. Data were prospectively collected and had a limited potential for ascertainment bias. Sleep is not normally evaluated in routine antenatal care, nor was it an integral part of the PEARS intervention education session. Furthermore, the interventions utilised in the PEARS study are acceptable to pregnant women in terms of both ease of use and affordability and are also cost effective; hence they represent a feasible intervention to improve sleep outcomes in future studies[29,30]

One of the main limitations is that the study was conducted in a single centre with a predominantly overweight and obese, educated population, somewhat restricting the generalisability of our results. As this was a secondary analysis of data from a randomised control trial, a power calculation was not performed and hence some findings may be significant if repeated in a large cohort.

## 4.3 | Interpretation

Although numerous studies have assessed the impact of antenatal lifestyle interventions on maternal and infant weight,[31,32]

few studies have assessed the impact on sleep behaviours in pregnancy. Previous studies of the PEARS data found that women in the intervention group had a significantly reduced dietary glycaemic index and greater exercise participation. Exercise and sleep tend to have a bidirectional association, i.e. exercise improves sleep but poor sleep may reduce a person's motivation to partake in exercise.[33] Exercise has been linked to improved sleep[34] and some associations have been made with physical activity participation and sleep in pregnancy.[35] Although sleep hygiene advice was not provided as part of the educational component of the PEARS lifestyle intervention, by improving physical activity participation and quality and quantity of dietary carbohydrate intake, the present study indicates that the intervention improved sleep duration.

An interesting finding of this study was that women who slept >7 h in early pregnancy were more likely to exceed GWG, whereas those with severe sleep restriction <6 h had higher rates of inadequate GWG during pregnancy. Outside of pregnancy, poor sleep quality in adults is associated with a significantly increased risk of obesity and metabolic syndrome[36,37] and in an adolescent population, for every hour of sleep lost, the odds of obesity increased 80%.[38] However, in pregnancy the association of sleep duration and quality with GWG is less clearly defined. A previous study of pregnant women found that those who reported improved sleep quality during pregnancy gained more weight during the 2nd and 3rd trimesters, but found no interaction between sleep duration and pregnancy BMI.[17] Another study found that those women with perceived poor sleep quality in pregnancy were more likely to report excess GWG, but no association was found between sleep duration and excess GWG.[18] Interestingly, that study did find that pregnant women with overweight prior to pregnancy who had a shorter sleep duration were more likely to have excess GWG.[18] Furthermore, an observational study using self-reported bodyweight at 6 weeks postpartum, reported that women with longer sleep duration had lower odds of excess GWG.[17] This is in contrast to a large study including over 5000 pregnant nulliparous women which found longer sleep duration was associated with excess GWG, with those sleeping more than 10 h twice as likely to exceed GWG targets.[18] This reflects the findings of our study, where women who were not sleep-deprived had higher rates of excess GWG. Just as excess GWG is associated with adverse pregnancy outcomes, inadequate GWG is associated with poor fetal outcomes, such as growth restriction, preterm birth and infant mortality.[39–41] We found that women who were severely sleep-restricted in early pregnancy were more likely to have inadequate GWG, which reflects the findings of a previous study in pregnancy, where shorter sleep duration was identified as a risk factor for inadequate GWG.[42] Hence lifestyle interventions implemented in pregnancy which improve sleep duration may potentially modify the risk factor of inadequate GWG.

To our knowledge, the association of sleep during pregnancy and maternal lipid profiles has not been previously explored. In the non-pregnant population, poor sleep has been linked with atherogenic lipid profiles both in adolescent and older females.[43,44] There are at least two likely processes whereby poor sleep quality can be related to higher lipid levels. First, through disruption to the circadian rhythm and subsequent melatonin production,[45–47] which influences a number of key metabolic processes that play an important role in control of dietary lipid absorption.[48] Secondly, poor sleep quality is thought to contribute to higher plasma lipids due to the consumption of a poorer quality diet containing more calories and total fat intake compared with individuals with optimal sleep duration and quality.[49] Although these are plausible explanations for an association between sleep deprivation and adverse lipid profiles outside of pregnancy, in our cohort of patients we did not find strong associations between sleep restriction and lipid profiles. This may be explained by the study population, which consisted of women with overweight and obesity and an overall adverse lipid profile from baseline. Furthermore, some studies have found U-shaped associations between sleep duration and lipids, with restricted and long sleep associated with adverse lipid profiles.[50]

## 5 | CONCLUSIONS

This analysis suggests that a lifestyle intervention adopted in pregnancy in a cohort of women with overweight or obesity may result in improved sleep duration, with maternal benefits such as lower triglyceride levels and higher breastfeeding rates. The association between sleep and excess and inadequate GWG requires further work.

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### CONFLICT OF INTERESTS

None declared. Completed disclosure of interest forms are available to view online as supporting information.

### ETHICS STATEMENT

The PEARS study (ISRCTN registry, https://www.isrctn.com/, ISRCTN29316280) was conducted between March 2013 and August 2016 with institutional ethical approval from the National Maternity Hospital and written maternal consent.

### DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

## ORCID

*Helena C. Bartels* https://orcid.org/0000-0002-6470-9364
*Fionnuala M. McAuliffe* https://orcid.org/0000-0002-3477-6494

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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