Original Research

Maternal body mass index, smoking status and small for gestational age: an Australian retrospective cohort study

C.R. Knight-Agarwal*, D. Mellor, E.N. Georgousopoulos, B. Krause, S. Coghlan

The University of Canberra, School of Clinical Sciences, Faculty of Health, Locked Bag 1, ACT, Australia, 2601

A R T I C L E   I N F O

Article history:
Received 10 March 2020
Accepted 13 May 2020
Available online 2 August 2020

Keywords:
Maternal obesity
Maternal smoking
Small for gestational age

A B S T R A C T

Objectives: Both maternal body mass index (BMI) and smoking during pregnancy have been associated with a range of adverse maternal and infant birth outcomes. This study aimed to identify whether these independent variables had an interacting relationship with small for gestational age in an Australian obstetric cohort.

Study design: A retrospective cohort design used data from the Birthing Outcomes System of a major tertiary hospital in Australia.

Methods: A total of 14,487 singleton births between January 2008 and December 2013 were included in the analysis. Chi-squared tests and one-way analysis of variance were used for the comparison of categorical and continuous variables, respectively. Adjusted odds ratios (AORs) were calculated to determine the association of smoking status with the outcome variable of interest, and these are reported for each maternal BMI category.

Results: Of the 14,487 women, 716 (4.9%) were underweight (BMI < 18 kg/m²), 7268 (50.2%) had healthy weight (BMI = 19–24 kg/m²), 3658 (25.3%) were overweight (BMI = 25–29 kg/m²), 1558 (10.8%) had class I obesity (BMI = 30–34 kg/m²), 711 (4.9%) had class II obesity (BMI = 35–39 kg/m²) and 576 (3.9%) had class III obesity (BMI = 40+ kg/m²). Of all women, 10.8% reported being current smokers, 82.0% reported to have never smoked and 4.0% reported to have stopped smoking during or before pregnancy. Smokers with a BMI > 40 kg/m² were 4.5 (AOR = 4.508; 95% confidence interval: 2.068–9.828) times more likely to give birth to a small-for-gestational-age infant than non-smokers within the same BMI category. This increased risk was not observed in women who ceased smoking before or during pregnancy.

Conclusions: Our study supports the efficacy of antismoking policies within maternal public health. In addition, greater support with respect to smoking cessation is indicated for women during pregnancy with an elevated BMI.

Introduction

Globally, high body mass index (BMI) is a major public health challenge mirrored in Australia, where over the last 30 years, a steady rising prevalence of overweight and obesity has occurred. Perhaps, of greater concern is the increasing incidence of obesity in young women.1 The Australian Health Survey reported 42.4% of women, aged 25–34 years, being overweight or obese.1 Maternal obesity has been shown to increase the risk of caesarean section,2 hypertensive disorders of pregnancy,3 premature birth4,5 and stillbirth.6 Infants born to women with obesity are at increased risk of being small for gestational age and developing cardiovascular disease in the long term.7 Numerous studies have demonstrated an increase in neonatal mortality associated with small-for-gestational-age (SGA) infants, defined as birthweight less than or equal to the 10th percentile for a given gestational age.8 Prematurity, special care nursery admissions, intrapartum foetal compromise and cerebral palsy are more likely to be observed in SGA infants.8

In addition to maternal obesity, prenatal tobacco smoking remains one of the most common preventable causes of infant morbidity and mortality.9,10 A meta-analysis conducted by Rayfield...
and Plagge\textsuperscript{12} included 39 studies with a total of 236,687 children from Asia, Europe, Australia and North and South America. Pooled adjusted odds ratios (AORs) demonstrated an elevated risk of maternal smoking in pregnancy for childhood overweight (OR = 1.37, 95% confidence interval [CI] = 1.28 to 1.46, I\textsuperscript{2} = 45%) and childhood obesity (OR = 1.55, 95% CI = 1.40 to 1.73, I\textsuperscript{2} = 24%). Maternal tobacco exposure affects nutrient and oxygen availability to the foetus through compromised maternal nutrient intake, absorption and placental transport capacity.\textsuperscript{13–16} In contrast to non-smokers, women who smoke during pregnancy have up to a twofold increased risk of preterm birth and a 30% increase in stillbirth rates.\textsuperscript{16} In Australia, a number of public health strategies have been implemented to reduce both tobacco smoke exposure and use in pregnant women.\textsuperscript{17} Notwithstanding such efforts, between the years 2014 and 2015, 12.1% of Australian women still reported smoking tobacco daily.\textsuperscript{18}

Despite the high prevalence of obesity and tobacco smoking during pregnancy, there is a paucity of evidence with regard to the differential effect of the combined influence of maternal pre-pregnancy BMI and smoking status on important foetal outcomes such as small for gestational age. A Chinese cohort study reported that passive smoking during pregnancy increased gestational diabetes mellitus risk in women independently and synergistically with pre-pregnancy obesity.\textsuperscript{19} A retrospective study of an obstetric population in New Zealand identified maternal obesity and tobacco smoking as independent risk factors for birthing an SGA infant.\textsuperscript{20} To the best of our knowledge, no studies have conducted a simultaneous analysis of association between maternal pre-pregnancy BMI, smoking status and small for gestational age. Therefore, the aim of the present study was to examine the association between maternal pre-pregnancy BMI, smoking status and small for gestational age within an Australian obstetric population.

Methods

Study design, setting, source of data and population

The data for this retrospective cohort study were obtained from the Birthing Outcomes System (BOS) used by the tertiary institution where the study was conducted. This institution is the largest facility of its kind, servicing a catchment population of approximately 540,000. Between January I, 2008, and December 31, 2013, there were 16,131 birth events recorded. Approximately 58% of women in this cohort presented in either their first or second trimester, with a further 3.0% presenting during their third trimester. The remaining cases either had implausible gestational ages recorded (15.7%) or no gestational age recorded (23.3%). Women who appeared more than once in the data set and having experienced more than one birth event during the study period were included in the study. Women with missing BMI data and variables of interest plus multiple pregnancies (twins and so on) were excluded, leaving 14,487 women for analysis.

Study variables and definitions

Maternal pre-pregnancy BMI was calculated by either midwives or obstetricians from height and weight recorded at the woman's first antenatal consultation, which is routinely at 12–14 weeks of gestation. In the BOS, BMI values are rounded up or down to the nearest whole number according to scientific notation. BMI was categorised into six groups: underweight (<18 kg/m\textsuperscript{2}); normal weight (19–24 kg/m\textsuperscript{2}); overweight (25–29 kg/m\textsuperscript{2}); obese class I (30–34 kg/m\textsuperscript{2}); obese class II (35–39 kg/m\textsuperscript{2}) and obese class III (>40 kg/m\textsuperscript{2}). All variables recorded in the BOS are classified using standard operating procedures developed by the tertiary institution where the study was conducted. Small for gestational age was calculated using Australian birthweight percentiles published by Dobbsin\textsuperscript{21} Parity 0 was defined as a woman who has not yet birthed a baby, and parity 1 refers to a woman who has given birth to one baby.\textsuperscript{22} Smoking status information is collected at a woman's first antenatal visit and routinely assessed by either obstetric staff or midwives throughout pregnancy.

Data analysis

Continuous variables are reported as means ± standard deviations, and categorical variables are reported as frequencies (n) and relative frequencies (%). Chi-squared tests were performed to assess the association between categorical variables, and one-way analysis of variance was used for the comparison of continuous variables. AORs were calculated to determine the association of smoking status with outcome variables of interest, and these are reported for each maternal BMI category. The bivariate logistic regression models were adjusted for maternal age, relationship status, country of birth and parity. These were considered by clinicians as the most important and used in recent published articles on this topic.\textsuperscript{15,23} Significance was set at the 5% level for two-tailed tests. Analyses were performed using Statistical Package for Social Sciences (SPSS) version 23 (IBM, Armonk, NY, USA).

Results

Of the 14,487 women, 716 (4.9%) were underweight (BMI <18 kg/m\textsuperscript{2}), 7268 (50.2%) had healthy weight (BMI = 19–24 kg/m\textsuperscript{2}), 3658 (25.3%) were overweight (BMI = 25–29 kg/m\textsuperscript{2}), 1558 (10.8%) had class I obesity (BMI = 30–34 kg/m\textsuperscript{2}), 711 (4.9%) had class II obesity (BMI = 35–39 kg/m\textsuperscript{2}) and 576 (3.9%) had class III obesity (BMI = 40+ kg/m\textsuperscript{2}). Of all women, 10.8% reported being current smokers, 82.0% reported to have never smoked and 4.2% reported to have stopped smoking during or before pregnancy. Demographic data for this cohort can be found in Table 1.

AORs for birth outcomes comparing women who never smoked with those who ceased and were current smokers can be found in Table 2. When we compared women who reported to have never smoked with women who ceased smoking before or during pregnancy, no differences were detected in small for gestational age when adjusted for maternal age, relationship status, country of birth and parity. Current smokers compared with those women who reported to have never smoked were found to have at least 2-fold higher odds of birthing an SGA infant across all BMI categories. Women with a BMI of ≥40 kg/m\textsuperscript{2} had the most significant increased risk of birthing an SGA infant, with an AOR of 4.51 (95% CI: 2.07–9.83).

Discussion

This study identified that women with morbid obesity, who reported smoking at the time of delivery, were four and a half times more likely to give birth to an SGA infant than those women with morbid obesity who had never smoked. In addition, women with class I and II obesity who continued to smoke at 15 weeks of gestation had double the risk of having an SGA infant compared with women who ceased smoking. These findings suggest there could be an interaction between a high maternal pre-pregnancy BMI and smoking that increases the risk of giving birth to an SGA infant.

Interestingly, women who reported smoking cessation before 15 weeks of gestation had rates of birthing SGA infants similar to those of non-smokers. Small for gestational age has been found to be
ANOVA $\equiv$ $P$-value derived from the chi-squared test for categorical variables and one-way ANOVA for continuous variables.

**Table 1**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>BMI $\leq$ 18 kg/m$^2$ (n = 716)</th>
<th>BMI 19–24 kg/m$^2$ (n = 7268)</th>
<th>BMI 25–29 kg/m$^2$ (n = 3658)</th>
<th>BMI 30–34 kg/m$^2$ (n = 1558)</th>
<th>BMI 35–39 kg/m$^2$ (n = 711)</th>
<th>BMI $\geq$ 40 kg/m$^2$ (n = 576)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking status</td>
<td>Never, n (%) 533 (74.4)</td>
<td>6111 (84.1)</td>
<td>2999 (82.0)</td>
<td>1218 (78.2)</td>
<td>560 (78.8)</td>
<td>437 (75.9)</td>
</tr>
<tr>
<td></td>
<td>Ceased, n (%) 28 (3.9)</td>
<td>272 (3.7)</td>
<td>142 (3.9)</td>
<td>70 (4.5)</td>
<td>31 (4.4)</td>
<td>34 (6.0)</td>
</tr>
<tr>
<td></td>
<td>Missing data, n (%) 24 (3.4)</td>
<td>242 (3.4)</td>
<td>141 (3.8)</td>
<td>44 (2.8)</td>
<td>19 (2.6)</td>
<td>13 (2.2)</td>
</tr>
<tr>
<td>Maternal age</td>
<td>Maternal age (years) 28.46 (5.848)</td>
<td>30.53 (5.405)</td>
<td>30.79 (5.627)</td>
<td>30.63 (5.805)</td>
<td>30.85 (5.383)</td>
<td>30.99 (5.565)</td>
</tr>
<tr>
<td>Relationship status</td>
<td>Single, n (%) 120 (17.5)**</td>
<td>625 (9.0)**</td>
<td>360 (10.4)**</td>
<td>183 (12.2)**</td>
<td>86 (12.5)**</td>
<td>63 (11.3)**</td>
</tr>
<tr>
<td></td>
<td>In a relationship (yes), n (%) 565 (82.5)**</td>
<td>6525 (91.0)**</td>
<td>3116 (89.6)**</td>
<td>1317 (87.8)**</td>
<td>602 (87.5)**</td>
<td>494 (88.7)**</td>
</tr>
<tr>
<td>Country of birth</td>
<td>Born outside of Australia, n (%) 268 (38.8)**</td>
<td>2288 (32.7)**</td>
<td>904 (25.7)**</td>
<td>302 (20.0)**</td>
<td>103 (14.9)**</td>
<td>58 (10.3)</td>
</tr>
<tr>
<td>Indigenous status</td>
<td>Non-Indigenous, n (%) 665 (96.3)**</td>
<td>6758 (97.9)**</td>
<td>3361 (97.7)**</td>
<td>1412 (96.4)**</td>
<td>652 (96.4)**</td>
<td>518 (95.4)**</td>
</tr>
<tr>
<td></td>
<td>Indigenous, n (%) 25 (3.7)**</td>
<td>143 (2.1)**</td>
<td>80 (2.3)**</td>
<td>53 (3.6)**</td>
<td>24 (3.6)**</td>
<td>25 (4.6)**</td>
</tr>
<tr>
<td>Parity</td>
<td>0, n (%) 351 (50.7)**</td>
<td>3413 (48.6)**</td>
<td>1475 (41.9)**</td>
<td>628 (41.5)*</td>
<td>243 (35.1)</td>
<td>203 (36.1)</td>
</tr>
<tr>
<td></td>
<td>1, n (%) 215 (31.1)**</td>
<td>2274 (32.4)**</td>
<td>1200 (34.1)**</td>
<td>484 (32.0)*</td>
<td>228 (32.9)</td>
<td>181 (32.1)</td>
</tr>
<tr>
<td></td>
<td>2, n (%) 77 (11.1)**</td>
<td>927 (13.2)**</td>
<td>536 (15.2)**</td>
<td>233 (15.4)*</td>
<td>116 (16.8)</td>
<td>46 (8.2)</td>
</tr>
<tr>
<td></td>
<td>$\geq$3, n (%) 49 (7.1)**</td>
<td>412 (5.8)**</td>
<td>169 (11.1)*</td>
<td>105 (15.2)</td>
<td>133 (23.6)</td>
<td></td>
</tr>
<tr>
<td>Birth status</td>
<td>SBL, n (%) 5 (0.7)</td>
<td>51 (0.7)</td>
<td>16 (0.5)</td>
<td>14 (0.9)</td>
<td>6 (0.9)</td>
<td>4 (0.7)</td>
</tr>
<tr>
<td></td>
<td>SBL, n (%) 2 (0.3)</td>
<td>30 (0.4)</td>
<td>9 (0.3)</td>
<td>6 (0.4)</td>
<td>2 (0.3)</td>
<td>2 (0.4)</td>
</tr>
<tr>
<td></td>
<td>Live born, n (%) 685 (99.0)</td>
<td>6945 (98.8)</td>
<td>3492 (99.3)</td>
<td>1494 (98.7)</td>
<td>684 (98.8)</td>
<td>557 (98.9)</td>
</tr>
<tr>
<td>Birthweight status</td>
<td>SGA, n (%) 165 (23.9)**</td>
<td>894 (12.8)**</td>
<td>376 (10.7)**</td>
<td>162 (10.7)**</td>
<td>69 (10.0)</td>
<td>53 (9.4)**</td>
</tr>
</tbody>
</table>

$P$ value $<$ 0.05.

**Table 2**

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>BMI $\leq$ 18 kg/m$^2$ (n = 716)</th>
<th>BMI 19–24 kg/m$^2$ (n = 7268)</th>
<th>BMI 25–29 kg/m$^2$ (n = 3658)</th>
<th>BMI 30–34 kg/m$^2$ (n = 1558)</th>
<th>BMI 35–39 kg/m$^2$ (n = 711)</th>
<th>BMI $\geq$ 40 kg/m$^2$ (n = 576)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking status</td>
<td>Never smoked vs. ceased smoker</td>
<td>OR, 95% CIs</td>
<td>OR, 95% CIs</td>
<td>OR, 95% CIs</td>
<td>OR, 95% CIs</td>
<td>OR, 95% CIs</td>
</tr>
<tr>
<td>Birthweight status</td>
<td>SGA 1.05 (0.259–4.27)</td>
<td>0.847 (0.536–1.34)</td>
<td>0.879 (0.430–1.80)</td>
<td>1.27 (0.548–2.94)</td>
<td>1.49 (0.477–4.65)</td>
<td>1.58 (0.435–5.75)</td>
</tr>
<tr>
<td>Never smoked vs. current smoker</td>
<td>OR, 95% CIs</td>
<td>OR, 95% CIs</td>
<td>OR, 95% CIs</td>
<td>OR, 95% CIs</td>
<td>OR, 95% CIs</td>
<td>OR, 95% CIs</td>
</tr>
<tr>
<td>Birthweight status</td>
<td>SGA 2.66 (1.42–4.99)</td>
<td>3.14 (2.40–4.1)</td>
<td>1.92 (1.27–2.88)</td>
<td>2.03 (1.16–3.58)</td>
<td>2.37 (1.15–4.92)</td>
<td>4.51 (2.07–9.83)</td>
</tr>
</tbody>
</table>

Adjusted odds ratios (AORs) for smoking status and small for gestational age, stratified by maternal BMI (n = 14,487).

**Weight gain, with most of the gain occurring during the first 3 months after cessation.** Women who quit smoking before or during pregnancy may thus be at higher risk of excessive gestational weight gain (GWG). A study by Llambi et al. reported that women who gave up smoking during the antenatal period increased GWG by 2.4 kg (95% CI = 1.3–3.4) after adjusting for pre-pregnancy BMI and other confounders in comparison with women who continued to smoke ($P < 0.001$). GWG was slightly higher in women who quit smoking at any point during pregnancy. Although smoking cessation interventions during pregnancy should continue to be promoted, women who are successful in quitting smoking during the antenatal period should be provided with extra support as well as dietary and lifestyle interventions to facilitate appropriate weight gain.

Although the impact of maternal smoking on small for gestational age has been previously examined, there is comparatively little evidence as to the effect of smoking cessation on this adverse outcome. As such, this study contributes novel findings to the evidence base. Further investigation of the optimal methods to

associated with 5.8–30% of perinatal mortality in Australia and New Zealand.21

In our study, the reduction of SGA risk demonstrated in women who ceased smoking compared with those who continued smoking throughout pregnancy suggests that smoking cessation could be a highly effective antenatal strategy to reduce preventable infant morbidity and mortality.22,23 However, data on the exact time of smoking cessation were not extensive enough to be further analysed in our study. Nevertheless, inclusion of women who reported ceasing smoking during pregnancy suggests that this behaviour may still be protective of adverse infant outcomes. From a public health perspective, cessation promotion should continue to target women who are currently pregnant and not just those of childbearing age. Interventions promoting smoking cessation have been successful in reducing the proportion of pregnant women who smoke, resulting in some improved pregnancy outcomes among women who quit smoking compared with those who continue. Despite this, in the general population, it is widely recognised that smoking cessation is associated with
promote maternal smoking cessation to improve pregnancy outcomes is warranted.

Strengths and limitations

Our study makes a significant contribution to the body of evidence that the combination of obesity and smoking during pregnancy is associated with an increased risk of having an SGA infant. The major strength of this research is the size of the cohort. The centralised data collection reduces potential bias within medical records as plausible risk factors and outcomes are routinely documented for women accessing antenatal services.

In our study, smoking status was included as a secondary exposure to maternal pre-pregnancy BMI and was only associated with small for gestational age. There are many potential explanations for this. First, in view of previous findings, maternal pre-pregnancy BMI might be an independent risk factor, and as such, it is not modified by smoking.

Data collected during antenatal care and recorded within the BOS may also risk an innate bias. Owing to the highly publicised adverse effects of smoking on health, it is possible that smoking is largely underreported by women in this cohort. Shipton et al.26 found that reliance on self-reported smoking status underestimated true smoking by 25% in pregnant women living in Scotland. Furthermore, Dietz et al.27 found that 22.9% of women who were actively smoking during pregnancy failed to disclose this behaviour.

In addition, the accuracy of data collection may also be impacted by the woman–practitioner relationship, the phrasing and understanding of questions, and potential assumptions made by healthcare professionals. Although other approaches such as testing for exhaled carbon monoxide or urinary nicotine are potentially more reliable in assessing smoking status, these are associated with additional costs and time to both the healthcare system and burden on the pregnant woman herself. Furthermore, inconsistencies in coding of smoking status in terms of timing of smoking cessation and quantity of cigarettes smoked limited the depth and accuracy of analysis possible in this study. We were not able to test whether smoking had a dose–effect response when combined with maternal pre-pregnancy BMI or whether the timing of smoking cessation and quantity previously smoked modified the risk of small for gestational age. As such, standardised data collection procedures and uniformity in coding descriptions would be useful to provide more meaningful evidence.

Anthropometric measurements were taken, on average, at 12 weeks of gestation before any significant gestational weight change is typically observed. Nevertheless, these values are still only an approximation of pre-pregnancy BMI. We were not able to investigate the effects of GWG as such information is not regularly collected at the study hospital. We did not control for gestational hypertension in our analysis, a risk factor for small for gestational age, and this is an acknowledged limitation of our study.

The findings that smoking increases the risk of delivering an SGA infant and cessation reduces this risk affirm current public health messages in Australia.17 Given that 10.8% of women in this cohort disclosed smoking currently, this is a figure that may be underreported and lower than that reported in other Australian surveys;28 health promotion and its associated funding is still necessary to reduce preventable neonatal morbidity and mortality in Australia. Clinicians also have a responsibility to facilitate a safe and non-judgemental environment for expectant mothers to disclose participation in health risk behaviours and be equipped to support, motivate and enable these women to cease such behaviours.28

Conclusion

In this study, a simultaneous analysis of maternal BMI and smoking status was conducted to identify whether these variables displayed an interaction that was associated with an effect on infant birthweight. Women who reported smoking throughout pregnancy were found to have a significantly increased risk of delivering an SGA infant compared with those who ceased smoking before or during pregnancy and those who had never smoked. In addition to affirming smoking cessation as an effective maternal public health action, this study advocates for the value of anthropometric measurements during the antenatal period, the need for screening tools to identify women at increased risk of obstetric complications and the need for standardised documentation within birth outcome—reporting systems.

Author statements

Acknowledgements

Only those who have made substantial contributions to the study and/or preparation of the MS have been named authors.

Ethical approval

Ethical approval was granted by the Research Ethics and Governance Office that is responsible for the coordination and management of ethical and site governance review processes for the area health service (approval code = ETHLR.11.167).

Funding

None declared.

Competing interests

None declared.

References


