BMJ Open

Association of BMI and interpregnancy BMI change with birth outcomes in an Australian obstetric population: a retrospective cohort study

Catherine R Knight-Agarwal,[1] Lauren T Williams,[2] Deborah Davis,[3] Rachel Davey,[4] Tom Cochrane,[4] Huanhua Zhang,[1] Peter Rickwood[5]

To cite: Knight-Agarwal CR, Williams LT, Davis D, *et al*. Association of BMI and interpregnancy BMI change with birth outcomes in an Australian obstetric population: a retrospective cohort study. *BMJ Open* 2016;**6**:e010667. doi:10.1136/bmjopen-2015-010667

► Prepublication history for this paper is available online. To view these files please visit the journal online (http://dx.doi.org/10.1136/bmjopen-2015-010667).

Received 10 December 2015
Revised 22 March 2016
Accepted 5 April 2016

For numbered affiliations see end of article.

Correspondence to
Catherine R Knight-Agarwal; Cathy.Knight-Agarwal@canberra.edu.au

ABSTRACT

Objective: To assess maternal and neonatal outcomes associated with increasing body mass index (BMI) and interpregnancy BMI changes in an Australian obstetric population.

Methods: A retrospective cohort study from 2008 to 2013 was undertaken. BMI for 14 875 women was categorised as follows: underweight (≤18 kg/m^2); normal weight (19–24 kg/m^2); overweight (25–29 kg/m^2); obese class I (30–34 kg/m^2); obese class II (35–39 kg/m^2) and obese class III (40+ kg/m^2). BMI categories and maternal, neonatal and birthing outcomes were examined using logistic regression. Interpregnancy change in BMI and the risk of adverse outcomes in the subsequent pregnancy were also examined.

Results: Within this cohort, 751 (5.1%) women were underweight, 7431 (50.0%) had normal BMI, 3748 (25.1%) were overweight, 1598 (10.8%) were obese class I, 737 (5.0%) were obese class II and 592 (4.0%) were obese class III. In bivariate adjusted models, obese women were at an increased risk of caesarean section, gestational diabetes, hypertensive disorders of pregnancy and neonatal morbidities including macrosomia, large for gestational age (LGA), hypoglycaemia, low 5 min Apgar score and respiratory distress. Multiparous women who experienced an interpregnancy increase of ≥3 BMI units had a higher adjusted OR (AOR) (CI) of the following adverse outcomes in their subsequent pregnancy: low 5-min Apgar score 3.242 (1.557 to 7.118); gestational diabetes mellitus (GDM) 3.258 (1.129 to 10.665) and hypertensive disorders of pregnancy 3.922 (1.243 to 14.760). These women were more likely to give birth vaginally 2.030 (1.417 to 2.913). Conversely, women whose parity changed from 0 to 1 and who experienced an interpregnancy increase of ≥3 BMI units had a higher AOR (CI) of caesarean section in their second pregnancy 1.806 (1.139 to 2.862).

Conclusions: Women who are overweight or obese have a significantly increased risk of various adverse outcomes. Interpregnancy weight gain, regardless of parity and baseline BMI, also increases various adverse outcomes. Effective weight management strategies are needed.

Strengths and limitations of this study

- As far as the authors are aware, this is the first Australian study of its kind.
- Data were collected in the course of routine care without independent verification, so it may be at risk of potential recording bias.
- Body mass index (BMI) data were rounded to the nearest whole number only, giving potential for misclassification of weight status for women with a BMI close to category thresholds.

INTRODUCTION

Australia has seen a dramatic increase in the rate of overweight and obesity. In 2011–2012, the Australian Bureau of Statistics reported that 55.7% of women have a body mass index (BMI) of 25 kg/m^2 or more.[1] Of particular concern is the prevalence of overweight and obesity in women of childbearing age. One urban Australian study reported a prevalence of maternal overweight and obesity to be 20% and 12.7%, respectively.[2] In a rural Australian cohort, almost two-thirds of the obstetric population studied were overweight or obese.[3] The consequences of increased maternal adiposity have been well documented. It is widely accepted that a high BMI during pregnancy increases the risk of maternal and infant morbidity, including gestational diabetes mellitus (GDM), hypertensive disorders, thromboembolic disorders, caesarean section, macrosomia and stillbirth.[4–7] A population-based Danish study of 403 092 women showed a significant increased risk of a wide variety of pregnancy, birth and neonatal complications in overweight, obese and severely obese women.[8] Increasing parity, independent of socioeconomic status, is associated with obesity later in life.[9] This effect may be compounded by excessive weight gain during[10]

or between pregnancies.[11 12] Having a high BMI during pregnancy increases the risk of obesity and premature death in adult offspring.[13 14] However, only a few studies have examined the association of BMI change between pregnancies with birth outcomes,[12 15–17] and these included only women whose parity changed from 0 to 1. To our knowledge, there are no studies that examine the association of BMI change between pregnancies with birth outcomes for women of all parities. The aims of this study were to assess various maternal and neonatal outcomes associated with increasing BMI and to examine interpregnancy BMI changes in an Australian obstetric population.

METHODS

Study population

This retrospective cohort study used data from the Birthing Outcomes System (BOS) at a major tertiary institution in the Australian Capital Territory (ACT) between January 2008 and December 2013. During that time, 16 131 mothers gave birth with the hospital being the major birthing centre for a catchment population of 550 000.[18] Women with missing BMI data and multiple pregnancies (twins, etc) were excluded, leaving 14 857 women for analysis.

Maternal BMI was derived from measured height and weight recorded at the first antenatal visit (usually 12 weeks gestation). In 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^2); normal weight (19–24 kg/m^2); overweight (25–29 kg/m^2); obese class I (30–34 kg/m^2); obese class II (35–39 kg/m^2) and obese class III (40+ kg/m^2). Ethnic-specific cut-offs were not used; however, ethnicity was considered in the adjusted analysis. All variables recorded in BOS are classified using the International Classification of Diseases (ICD)-10 codes and standard operating procedures (SOPs) developed by the tertiary institution where the study was conducted.[19 20] Macrosomia was defined as ≥4000 g.[19] An Apgar score (used to evaluate neonatal well-being immediately after birth)[21] of ≤6 at 5 min is used at The Canberra Hospital (TCH) as an indicator for referral to a neonatal morbidity meeting. Hypertensive disorders were grouped together due to the difficulty of separating essential hypertension, gestational hypertension and pre-eclampsia as they often coexist.[22] Birthweight results were expressed as SD (z) scores corrected for gestation at the time of delivery. Small for gestational age (SGA) and large for gestational age (LGA) were calculated using Australian birthweight percentiles published by Dobbins and associates.[23]

Statistical analyses

Continuous variables are reported as means and SDs and categorical variables as numbers (n) and percentages (%). Adjusted ORs (AORs) including 95% CIs for the association of maternal BMI with outcome variables were calculated for each maternal BMI category. Goodness-of-fit, residual and influence analyses were performed. The bivariate logistic regression model was adjusted for maternal age, parity, country of birth and smoking status being considered by clinicians as the most important and used in most published papers on this topic.[2 5] For skewed data, non-parametric tests such as the Kruskal-Wallis test were employed to assess difference. Significance was accepted at the 5% level on two-tailed tests. Change in BMI between pregnancies, independent of parity and risk of adverse outcomes during the subsequent pregnancy were also investigated. Parity is defined as the delivery of a baby >20 weeks gestation or >400 g in weight. Parity 0 refers to a woman who has not yet delivered a baby, and parity 1 refers to a woman who has given birth to one baby.[24] The bivariate logistic regression model was adjusted for baseline BMI, interpregnancy interval, parity, maternal age, country of birth and smoking. Change in BMI was categorised as a decrease of ≥1 BMI units, 0 BMI unit (reference group), increase of 1 to <3 BMI units and increase of ≥3 BMI units. Interpregnancy interval was calculated as the number of completed months between the birth of the first baby and estimated conception of the second. Analysis was performed using the Statistical Package for Social Sciences (SPSS) V.21.

RESULTS

A total of 14 857 singleton births, with accompanying maternal BMIs, were included: 751 (5.1%) women were underweight, 7431 (50.0%) had normal BMI, 3748 (25.1%) were overweight, 1598 (10.8%) were obese class I, 737 (5.0%) were obese class II and 592 (4.0%) were obese class III. The timing of the first antenatal visit is poorly recorded—completely absent in 23.3% of cases, and having implausible values (<6 or >42 weeks) for a further 15.7% of cases. About 58% of women had their first antenatal visit in either the first or second trimester. Only 3.1% of cases had a first recorded antenatal visit in the third trimester. Demographic characteristics are shown in table 1.

Increasing BMI was associated with reduced rates of spontaneous and assisted vaginal birth and increased rates of caesarean section. Third-degree/fourth-degree perineal tears and episiotomy were less likely to occur with increasing BMI. Increasing maternal BMI was associated with having a macrosomic baby and an increase in gestational diabetes and hypertensive disorders of pregnancy. The adverse neonatal outcomes of low 5 min Apgar score, hypoglycaemia and respiratory distress increased as BMI rose. AORs for maternal, peripartum and neonatal outcomes following bivariate logistic regression are shown in table 2. As BMI increased, so did maternal blood loss of ≥500 mL, GDM and hypertensive disorders of pregnancy.

From the original cohort of 14 857, we extracted data for 1868 women, of ≥1 parity, recorded as having two

Table 1 Demographic characteristics of women in the study sample by BMI category (n=14 857)

Variable	≤18 kg/m^2 (n=751)	19–24 kg/m^2 (n=7431)	25–29 kg/m^2 (n=3748)	30–34 kg/m^2 (n=1598)	35–39 kg/m^2 (n=737)	40+ kg/m^2 (n=592)
Maternal characteristics and outcomes						
Mean age in years (SD)	28.46 (5.848)	30.53 (5.405)	30.79 (5.627)	30.63 (5.805)	30.85 (5.383)	30.99 (5.565)
Parity n (%)						
0	365 (50.3)	3598 (48.4)	1557 (41.5)	664 (41.6)	260 (35.3)	210 (35.5)
1	226 (31.2)	2405 (32.4)	1278 (34.1)	512 (32.0)	244 (33.1)	190 (32.1)
2	821 (1.3)	979 (13.2)	585 (15.6)	244 (15.3)	123 (16.7)	102 (17.2)
3	26 (3.6)	294 (4.0)	196 (5.2)	110 (6.9)	59 (8.0)	49 (8.3)
≥4	26 (3.6)	155 (2.1)	132 (3.5)	68 (4.3)	51 (6.9)	41 (6.9)
Country of birth n (%)						
Australian non-Aboriginal and Torres Strait Islander	423 (58.3)	4888 (65.8)	2710 (72.3)	1223 (76.5)	608 (82.5)	505 (85.3)
Australian Aboriginal and Torres Strait Islander	26 (3.7)	154 (2.1)	85 (2.3)	59 (3.8)	26 (3.6)	28 (4.9)
Asian	190 (26.2)	1241 (16.7)	455 (12.1)	113 (7.1)	33 (4.5)	8 (1.4)
Other	84 (11.6)	1128 (15.2)	495 (13.2)	201 (12.6)	71 (9.6)	51 (8.6)
Smoking status n (%)						
Smoking ceased during pregnancy	28 (3.9)	277 (3.7)	142 (3.8)	71 (4.4)	31 (4.2)	34 (5.7)
Current smoker	136 (18.8)	660 (8.9)	396 (10.6)	323 (14.5)	105 (14.2)	94 (15.9)
Never smoker	561 (77.4)	6494 (87.4)	3210 (85.6)	1295 (81.0)	601 (81.5)	464 (78.4)
Maternal medical complications n (%)						
Pre-existing diabetes (types I and II)	5 (0.7)	96 (1.3)	90 (2.4)	69 (4.3)	47 (6.4)	36 (6.1)
Essential hypertension	2 (0.3)	47 (0.6)	46 (1.2)	50 (3.1)	38 (5.2)	56 (9.5)
Birth status n (%)						
Stillborn	10 (1.38)	86 (1.16)	29 (0.77)	23 (1.44)	8 (1.09)	6 (1.10)
Birth mode n (%)						
Spontaneous vaginal	489 (67.45)	4744 (63.84)	2239 (59.74)	913 (57.13)	385 (52.24)	287 (48.48)
Caesarean	119 (16.41)	1636 (22.02)	982 (26.2)	520 (32.54)	297 (40.3)	238 (40.2)
Instrumental vaginal	114 (15.72)	1016 (13.67)	510 (13.61)	158 (9.89)	54 (7.33)	65 (10.98)
Apgar score n (%)						
5 min Apgar score ≤6	18 (2.49)	268 (3.61)	147 (3.93)	97 (6.08)	42 (5.7)	36 (6.08)
Mean score (SD)	8.85 (1.28)	8.85 (1.31)	8.80 (1.28)	8.69 (1.51)	8.68 (1.47)	8.69 (1.35)
Infant birth weight n (%)						
Macrosomia	36 (4.97)	751 (10.11)	571 (15.23)	271 (16.96)	120 (16.28)	116 (19.59)
Low birth weight <2500 g	111 (15.31)	653 (8.79)	296 (7.9)	172 (10.76)	70 (9.5)	49 (8.28)
Mean standardised z-score (SD)	−0.38 (1.00)	−0.04 (0.95)	0.08 (0.99)	0.05 (1.12)	0.07 (1.08)	0.16 (1.10)
SGA	172 (22.90)	970 (13.05)	410 (10.94)	178 (11.14)	72 (9.78)	54 (9.12)
LGA	20 (2.66)	500 (6.73)	405 (10.81)	218 (13.64)	111 (15.10)	107 (18.10)
Maternal blood loss n (%)						
Haemorrhage ≥500 mL	38 (5.2)	406 (5.5)	219 (5.8)	107 (6.7)	49 (6.6)	32 (5.4)

Continued

Table 1 Continued

Variable	≤18 kg/m² (n=751)	19–24 kg/m² (n=7431)	25–29 kg/m² (n=3748)	30–34 kg/m² (n=1598)	35–39 kg/m² (n=737)	40+ kg/m² (n=592)
Peristatus n (%)						
Third-degree/fourth-degree tear*	44 (7.26)	300 (5.18)	141 (5.10)	37 (3.43)	13 (2.95)	10 (2.82)
Episiotomy*	68 (11.22)	569 (9.82)	235 (8.50)	66 (6.12)	22 (5.00)	18 (5.08)
Obstetric complications n (%)						
GDM	9 (1.24)	118 (1.59)	116 (3.09)	75 (4.69)	45 (6.11)	52 (8.78)
Hypertensive disorders	9 (1.24)	8.2 (1.1)	77 (2.05)	58 (3.63)	43 (5.83)	53 (8.95)
Labour complications n (%)						
Shoulder dystocia	2 (0.28)	76 (1.02)	42 (1.12)	21 (1.31)	13 (1.76)	11 (1.86)
VBAC	2 (0.28)	34 (0.46)	11 (0.29)	8 (0.5)	0 (0.0)	2 (0.34)
Neonatal outcomes n (%)						
Hypoglycaemia	12 (1.66)	107 (1.44)	75 (2.0)	39 (2.44)	24 (3.26)	22 (3.72)
Birth trauma	11 (1.52)	94 (1.26)	54 (1.44)	26 (1.63)	9 (1.22)	12 (2.03)
Respiratory distress	14 (1.93)	187 (2.52)	78 (2.08)	55 (3.44)	37 (5.02)	24 (4.05)

*Women who had a caesarean for the second pregnancy excluded.
BMI, body mass index; GDM, gestational diabetes mellitus; LGA, large for gestational age; SGA, small for gestational age; VBAC, vaginal birth after caesarean.

separate and single birth events within the study period. Data were not included for third or subsequent births in the study period. AORs for outcomes during the subsequent pregnancy in relation to interpregnancy BMI change are shown in table 3. Increase in BMI between two pregnancies occurred in 820 (43.9%) women, 499 (26.6%) women had no change and the remaining 552 (29.5%) women showed a decrease in BMI. Those women who experienced an increase of ≥3 BMI units between pregnancies had a greater risk of having an infant with low 5 min Apgar score, gestational diabetes or a hypertensive disorder in their subsequent pregnancy. They were less likely to have an instrumental birth. Any increase or decrease in BMI units between pregnancies provided higher AORs of having a spontaneous vaginal delivery. Women who experienced a decrease of ≥1 BMI units had a high risk of having an infant with respiratory distress. From the original cohort, we also investigated the 1155 women whose parity changed from 0 to 1 and, who were recorded as having two separate and single birth events within the study period. AORs for outcomes during the second pregnancy in relation to interpregnancy BMI change are shown in table 4. Increase in BMI between the first and second pregnancies occurred in 517 (44.8%) women with no change in 309 (26.7%) women and a decrease in BMI in 329 (28.5%) women. Those women who experienced an increase of ≥3 BMI units between pregnancies had a greater risk of caesarean section, having an infant with low Apgar scores and developing a hypertensive disorder in their second pregnancy. Any decrease in BMI units resulted in a higher AOR of giving birth to a low birthweight infant.

DISCUSSION

This study demonstrates that being overweight or obese increases the risk of various adverse maternal and neonatal outcomes, including low 5 min Apgar score, postpartum haemorrhage, GDM, hypertensive disorders of pregnancy, macrosomia, LGA and neonatal hypoglycaemia. Numerous studies have documented similar findings.[2 3 5 6] However, to the best of our knowledge, this is the first Australian study to include analyses on interpregnancy BMI change and risk of subsequent adverse maternal and neonatal events. In this cohort, parity increased with higher BMI categories, which may be explained by the tendency of women to accumulate excess weight with each pregnancy.[24] The association between increasing BMI and adverse health behaviours such as smoking was confirmed in our cohort.[25]

Obese women are more insulin resistant than normal-weight women, and the risk of developing GDM has been positively associated with obesity.[2 3 5] Likewise, we demonstrated that as BMI increased, the risk of developing GDM also increased. The Royal Australian and New Zealand College of Obstetricians and Gynaecologists guidelines recommend that overweight and obese

Table 2 Adjusted ORs (AORs) for maternal and neonatal outcomes according to maternal BMI for women in the cohort

	≤18 kg/m^2 (n=751)	19–24 kg/m^2 (n=7431)	25–29 kg/m^2 (n=3748)	30–34 kg/m^2 (n=1598)	35–39 kg/m^2 (n=737)	≥40 kg/m^2 (n=592)
Birth status						
Liveborn	0.844 (0.455 to 1.748)	1.00	1.496 (0.984 to 2.342)	0.837 (0.525 to 1.396)	1.016 (0.517 to 2.300)	1.081 (0.506 to 2.805)
Stillborn	1.185 (0.572 to 2.197)	1.00	0.669 (0.427 to 1.016)	1.195 (0.716 to 1.906)	0.985 (0.435 to 1.935)	0.925 (0.356 to 1.977)
Birth type						
Spontaneous vaginal	1.162 (0.927 to 1.445)	1.00	**1.13 (1.002 to 1.274)**	**0.781 (0.646 to 0.939)**	**0.642 (0.473 to 0.855)**	0.996 (0.742 to 1.315)
Caesarean	0.678 (0.548 to 0.832)	1.00	**1.296 (1.180 to 1.422)**	**1.822 (1.612 to 2.057)**	**2.601 (2.210 to 3.058)**	**2.703 (2.257 to 3.232)**
Instrumental vaginal	**1.215 (1.028 to 1.441)**	1.00	**0.772 (0.710 to 0.840)**	**0.664 (0.592 to 0.745)**	**0.498 (0.424 to 0.584)**	**0.411 (0.343 to 0.490)**
Apgar score						
Low 5 min Apgar score ≤6	0.610 (0.351 to 0.985)	1.00	1.130 (0.915 to 1.389)	**1.743 (1.356 to 2.221)**	**1.552 (1.075 to 2.183)**	**1.799 (1.225 to 2.567)**
Infant birth weight						
Macrosomia	0.534 (0.370 to 0.746)	1.00	**1.529 (1.356 to 1.722)**	**1.720 (1.471 to 2.007)**	**1.527 (1.225 to 1.889)**	**1.935 (1.541 to 2.413)**
LBW <2500 g	**1.706 (1.356 to 2.128)**	1.00	0.852 (0.734 to 0.986)	1.163 (0.966 to 1.394)	0.917 (0.690 to 1.198)	0.799 (0.575 to 1.086)
SGA	**1.804 (1.491 to 2.174)**	1.00	**0.867 (0.765 to 0.982)**	0.882 (0.740 to 1.047)	0.817 (0.627 to 1.049)	**0.752 (0.555 to 0.999)**
LGA	**0.441 (0.271 to 0.676)**	1.00	**1.596 (1.389 to 1.832)**	**2.084 (1.754 to 2.471)**	**2.187 (1.739 to 2.729)**	**2.743 (2.165 to 3.449)**
Maternal blood loss						
Haemorrhage ≥500 mL	0.986 (0.787 to 1.224)	1.00	1.039 (0.928 to 1.164)	**1.340 (1.154 to 1.551)**	**1.528 (1.247 to 1.862)**	**1.349 (1.066 to 1.693)**
Peristatus						
Third-degree/fourth-degree tear*	1.250 (0.876 to 1.744)	1.00	1.208 (0.975 to 1.490)	0.879 (0.604 to 1.243)	0.869 (0.454 to 1.513)	0.777 (0.364 to 1.453)
Episiotomy*	1.111 (0.836 to 1.458)	1.00	0.980 (0.828 to 1.156)	0.763 (0.577 to 0.994)	0.698 (0.429 to 1.077)	0.693 (0.409 to 1.105)
Obstetric complications						
GDM	0.722 (0.338 to 1.356)	1.00	**2.171 (1.668 to 2.828)**	**3.529 (2.587 to 4.785)**	**4.991 (3.424 to 7.161)**	**7.817 (5.415 to 11.154)**
Hypertensive disorders	1.282 (0.595 to 2.437)	1.00	**1.953 (1.419 to 2.686)**	**3.415 (2.392 to 4.842)**	**6.077 (4.103 to 8.890)**	**8.947 (6.110 to 12.990)**
Labour complications						
Shoulder dystocia	0.148 (0.008 to 0.671)	1.00	1.076 (0.729 to 1.566)	1.241 (0.742 to 1.988)	1.624 (0.853 to 2.858)	1.702 (0.845 to 3.112)
VBAC	0.661 (0.107 to 2.191)	1.00	0.544 (0.254 to 1.065)	1.035 (0.443 to 2.144)	NA	0.582 (0.093 to 1.975)
Neonatal outcomes						
Hypoglycaemia	1.101 (0.571 to 1.935)	1.00	**1.414 (1.042 to 1.909)**	**1.740 (1.178 to 2.519)**	**2.415 (1.484 to 3.770)**	**2.892 (1.753 to 4.569)**
Birth trauma	1.192 (0.598 to 2.150)	1.00	1.232 (0.873 to 1.721)	1.428 (0.900 to 2.189)	1.147 (0.534 to 2.171)	1.943 (0.998 to 3.461)
Respiratory distress	0.838 (0.461 to 1.402)	1.00	0.817 (0.620 to 1.068)	1.368 (0.994 to 1.855)	**2.004 (1.364 to 2.867)**	1.566 (0.976 to 2.398)

Reference group is BMI 19–24 kg/m^2. ORs and 95% CIs from logistic models adjusted for parity, maternal age, country of birth and smoking. Significant results are bolded.
*Women who had a caesarean for the second pregnancy excluded.
BMI, body mass index; GDM, gestational diabetes mellitus; LBW, low birth weight; LGA, large for gestational age; NA, not applicable; SGA, small for gestational age; VBAC, vaginal birth after caesarean.

Table 3 Adjusted ORs for outcomes during the subsequent pregnancy in relation to interpregnancy BMI change for women of all parities

	Decrease of ≥1 BMI units n=552 OR (95% CI)	No change in BMI units n=496 OR (95% CI)	Increase of 1 to <3 BMI units n=509 OR (95% CI)	Increase of ≥3 BMI units n=311 OR (95% CI)
Birth status				
Stillborn	0.313 (0.015 to 2.467)	1.00	0.697 (0.091 to 4.250)	2.364 (0.506 to 12.288)
Birth type				
Spontaneous vaginal	**1.501 (1.087 to 2.084)**	1.00	**1.728 (1.253 to 2.395)**	**2.030 (1.417 to 2.913)**
Caesarean	0.603 (0.343 to 1.041)	1.00	0.923 (0.554 to 1.534)	0.518 (0.245 to 1.019)
Instrumental vaginal	0.820 (0.613 to 1.096)	1.00	**0.649 (0.486 to 0.864)**	**0.647 (0.465 to 0.900)**
Apgar score				
5 min Apgar score ≤6	1.369 (0.633 to 3.072)	1.00	0.820 (0.327 to 2.003)	**3.242 (1.557 to 7.118)**
Infant birth weight				
LBW <2500 g	1.706 (0.937 to 3.212)	1.00	1.388 (0.731 to 2.690)	1.836 (0.953 to 3.598)
Macrosomia	1.241 (0.887 to 1.743)	1.00	1.162 (0.822 to 1.647)	1.472 (0.996 to 2.169)
Maternal blood loss				
Haemorrhage ≥500 mL	1.180 (0.812 to 1.720)	1.00	1.115 (0.760 to 1.640)	1.211 (0.783 to 1.860)
Peristatus				
Third-degree /fourth-degree tear*	0.997 (0.399 to 2.490)	1.00	1.245 (0.513 to 3.057)	0.793 (0.212 to 2.452)
Episiotomy*	1.462 (0.641 to 3.455)	1.00	2.143 (0.995 to 4.894)	0.826 (0.223 to 2.531)
Obstetric complications				
GDM†	0.901 (0.248 to 3.271)	1.00	1.548 (0.510 to 5.171)	**3.258 (1.129 to 10.665)**
Hypertensive disorders†	1.508 (0.449 to 5.819)	1.00	1.714 (0.511 to 6.613)	**3.922 (1.243 to 14.760)**
Labour complications				
Shoulder dystocia*	0.706 (0.222 to 2.175)	1.00	1.160 (0.409 to 3.368)	0.885 (0.246 to 2.951)
VBAC‡	1.201 (0.382 to 4.163)	1.00	0.348 (0.048 to 1.717)	0.786 (0.152 to 3.448)
Neonatal outcomes				
Hypoglycaemia	1.728 (0.606 to 5.605)	1.00	1.119 (0.334 to 3.917)	1.843 (0.543 to 6.519)
Birth trauma	1.321 (0.470 to 3.981)	1.00	1.267 (0.436 to 3.882)	0.759 (0.157 to 2.939)
Respiratory distress	**2.359 (1.006 to 6.109)**	1.00	1.452 (0.550 to 4.051)	1.831 (0.644 to 5.320)

Significant results are shown in bold typeface.
The reference group is no change in BMI units.
ORs and 95% CIs from logistic models adjusted for baseline BMI, interpregnancy interval, maternal age, country of birth, parity and smoking.
*Women who had a caesarean for the second pregnancy excluded (n=1482).
†Women who had GDM and hypertension in the first pregnancy excluded (n=1802).
‡Women who had a caesarean for the first pregnancy only (n=367).
BMI, body mass index; GDM, gestational diabetes mellitus; LBW, low birth weight; VBAC, vaginal birth after caesarean.

Table 4 Adjusted ORs for outcomes during the subsequent pregnancy in relation to interpregnancy BMI change for women from parity 0 to 1

	Decrease of ≥1 BMI units (n=329) OR (95% CI)	No change in BMI units (n=309) OR (95% CI)	Increase of 1 to <3 BMI units (n=328) OR (95% CI)	Increase of ≥3 BMI units (n=189) OR (95% CI)
Birth status				
Stillborn	0.973 (0.038 to 24.759)	1.00	2.066 (0.196 to 44.776)	3.955 (0.372 to 86.065)
Birth type				
Spontaneous vaginal	0.944 (0.654 to 1.362)	1.00	**0.671 (0.470 to 0.954)**	0.727 (0.480 to 1.104)
Caesarean	1.344 (0.890 to 2.040)	1.00	**1.534 (1.026 to 2.310)**	**1.806 (1.139 to 2.862)**
Instrumental vaginal	0.580 (0.297 to 1.104)	1.00	1.107 (0.631 to 1.954)	0.582 (0.250 to 1.240)
Apgar score				
5 min Apgar score ≤6	2.005 (0.700 to 6.526)	1.00	1.575 (0.518 to 5.282)	**5.197 (1.932 to 16.434)**
Infant birth weight				
Macrosomia	1.202 (0.779 to 1.864)	1.00	1.138 (0.734 to 1.770)	1.537 (0.939 to 2.505)
LBW <2500 g	**2.553 (1.088 to 6.695)**	1.00	0.863 (0.288 to 2.581)	1.734 (0.621 to 5.027)
Maternal blood loss				
Haemorrhage ≥500 mL	1.078 (0.670 to 1.738)	1.00	1.043 (0.648 to 1.680)	1.183 (0.682 to 2.026)
Peristatus				
Third-degree/ fourth-degree tear*	0.922 (0.290 to 2.854)	1.00	1.560 (0.582 to 4.411)	0.848 (0.178 to 3.171)
Episiotomy*	1.074 (0.293 to 3.934)	1.00	**3.571 (1.342 to 11.255)**	0.379 (0.020 to 2.415)
Obstetric complications				
GDM†	1.224 (0.263 to 6.357)	1.00	2.267 (0.48 to 16.006)	4.211 (0.881 to 29.957)
Hypertensive disorders†	4.626 (0.737 to 89.051)	1.00	3.696 (0.54 to 72.696)	**9.642 (1.517 to 186.621)**
Labour complications				
Shoulder dystocia*	0.768 (0.207 to 2.843)	1.00	1.160 (0.408 to 3.369)	0.849 (0.235 to 2.839)
VBAC‡	1.605 (0.476 to 6.210)	1.00	0.256 (0.013 to 1.841)	0.906 (0.117 to 5.127)
Neonatal outcomes				
Hypoglycaemia	1.197 (0.312 to 4.901)	1.00	0.976 (0.228 to 4.184)	1.790 (0.414 to 7.740)
Birth trauma	1.472 (0.482 to 4.944)	1.00	1.108 (0.329 to 3.892)	0.297 (0.015 to 1.886)
Respiratory distress	2.618 (0.877 to 9.584)	1.00	1.215 (0.317 to 4.971)	1.196 (0.231 to 5.545)

Significant results are shown in bold typeface.
The reference group is no change in BMI units.
ORs and 95% CIs from logistic models adjusted for baseline BMI, interpregnancy interval, maternal age, country of birth and smoking.
*Women who had a caesarean excluded (n=856).
†Women who had gestational diabetes and hypertension in the first pregnancy excluded (n=924).
‡Women who had a caesarean for the first pregnancy only (n=231).
BMI, body mass index; GDM, gestational diabetes mellitus; LBW, low birth weight; VBAC, vaginal birth after caesarean.

women should be offered early glucose tolerance test (GTT) screening, repeated at 26–28 weeks.[22] However, qualitative evidence from study hospital indicates that early GTT screening of obese women may not always be taking place.[26] Our analysis of multiparous women found that an interpregnancy increase of ≥3 BMI units is associated with elevated risk of GDM in a second, subsequent pregnancy with an AOR of 3.258 (1.129 to 10.665). We excluded those who had experienced GDM in their previous pregnancy. The 2006 Swedish study by Villamor and Cnattingius[12] reported an AOR of 2.09 (1.68 to 2.61) for developing GDM in women with an interpregnancy weight gain of ≥3 BMI units. Other studies have reported similar findings.[15 16]

As BMI increased in our cohort, the risk of hypertensive disorders of pregnancy also increased, a finding consistent with similar studies.[2 3 5 7] Gestational hypertension has an increasing incidence with elevated BMI, whereas pre-eclampsia is less common in a subsequent pregnancy than in the first pregnancy. The results of our analysis indicate the risk of becoming hypertensive increases significantly with interpregnancy weight gain of ≥3 BMI units, making the current finding even more important because the incidence of pre-eclampsia has been shown to be less in subsequent pregnancies.[27]

The women in our cohort with a BMI >30 kg/m^2 were at an increased risk of postpartum haemorrhage, a finding that has been reported elsewhere.[7 28] Documentation of maternal blood loss, at the time of birth, is usually an estimate only and is therefore open to bias. However, the accuracy of blood loss estimation at the study hospital is likely increased by their practice of weighing drapes and measuring clots (personal communication Dr DC Knight 26 July 2015). Our findings

support previous research that maternal obesity is associated with a significantly elevated risk for low 5 min Apgar scores.[6 29] A recent Swedish study of 1 764 403 live singleton infants delivered at term found rates of low 5 min Apgar scores increased from 0.4 per 1000 among infants of underweight women (BMI <18.5) to 2.4 per 1000 among infants of women with obesity class III (BMI ≥40).[30] Conversely, a Welsh population-based study of 60 167 deliveries reported that a 5 min Apgar score <7 was not more common in obese compared with normal-weight women.[31]

In the developed world, there had been a rapid rise in the rate of caesarean section, mirroring the increase in obesity trends.[32] In one systematic review and meta-analysis, the authors reported that compared with women with normal BMI, the risk of caesarean section in nulliparous, singleton pregnancies is increased 1.5 times in overweight, 2.25 times in obese and even more in morbidly obese women.[33] Women in our cohort (parity 0–1) who gained ≥3 BMI units were more likely to experience a caesarean section for their second birth. The women of higher parity, who experienced an increase in BMI between pregnancies, were more likely to experience a spontaneous vaginal birth than other modes of delivery. This could be explained by a tendency for the rate of instrumental births to decrease following the first delivery. From 2008 to 2013 at the study hospital, the percentage of instrumental births in nulliparous women was 23% compared with an incidence of only 5% in women experiencing subsequent births. In relation to intrauterine growth, we found a strong association in women (parity 0–1) with decreased BMI units between their first and second pregnancies with delivery of a low birthweight baby. A 2011 systematic review and meta-analyses of maternal BMI and risk of preterm birth and low birth weight found that singletons born to underweight women have higher risks of having a low birth weight than those born to women with a normal BMI.[34] In addition, women (all parities) who experienced an interpregnancy decrease of ≤1 BMI units had a higher risk of having an infant with respiratory distress following their subsequent birth. This appears to be a new finding and requires additional research to elucidate the contributing mechanisms.

Since the prevalence of lifestyle-associated risk factors for maternal and perinatal complications, such as smoking and alcohol consumption, is decreasing in several developed countries, high BMI before and increasing BMI between pregnancies is likely to contribute substantially to the causes of adverse pregnancy outcomes.[35 36] Our study makes a significant contribution to the growing body of evidence that entering pregnancy within a healthy BMI range, regardless of parity, is essential for the health and well-being of mother and child.

There are some limitations to our study that should be acknowledged. Data were collected in the course of routine care by obstetric and midwifery staff and are therefore at risk of potential recording bias. Weight and height measurements are taken, on average, at 12 weeks gestation before any real impact of gestational weight gain is observed. Nevertheless, these values are still only an approximation of prepregnancy BMI. A major limitation of the BOS software is that it does not allow the user to record actual BMI, but rounds up to the nearest whole figure. Ideally, we would have liked to also control for gestational age at the time of initial BMI recording, but these data are not routinely collected in our dataset, so we were unable to do it, and this is an acknowledged limitation of our analysis. We were not able to investigate the effects of weight gain during pregnancy as such information is not regularly collected at the study hospital. In addition, we cannot exclude the possibility of residual confounding by unknown risk factors or illnesses that could be associated with interpregnancy weight gain and various adverse gestational outcomes. The BMI distribution reported here is positively skewed compared with similar Australian cohorts.[2 5] Therefore, we are unable to suggest that our study is representative of the Australian obstetric population at large. Our study was not adequately powered to detect significant differences in outcomes such as stillbirth, shoulder dystocia and birth trauma. We had insufficient numbers to stratify the interpregnancy weight change analyses according to whether a woman's BMI was above or below a particular cut-off as was done by Villamor and Cnattinguis in 2006.[12]

It is interesting to note the differences observed in interpregnancy BMI change in our cohort for parity 0–1 as opposed to women of higher parity. From a clinical perspective, it could be important to distinguish between the influence of weight gain during pregnancy and the influence of interpregnancy BMI change. This is because weight retention may be independently affected by either life stage. Routine weighing of women during their pregnancy is an important measure that is not always undertaken in practice. Healthy weight for women planning on becoming pregnant is the ideal, and excessive weight gain during and between pregnancies should be prevented. Maternity care providers should be encouraged to counsel women regarding the health complications associated with high BMI and provide those women with postpartum support particularly if weight loss is indicated.

Author affiliations
[1]School of Public Health and Nutrition, The University of Canberra, Canberra, Australian Capital Territory, Australia
[2]School of Allied Health Sciences, Griffith University, Gold Coast Campus, Southport, Queensland, Australia
[3]Department of Midwifery, The University of Canberra, Canberra, Australian Capital Territory, Australia
[4]Centre for Research and Action in Public Health, The University of Canberra, Canberra, Australian Capital Territory, Australia
[5]Institute for Sustainable Futures, The University of Technology Sydney, Broadway, New South Wales, Australia

Acknowledgements The authors would like to thank Dr David Knight FRCOG, FRANZCOG, for his assistance with this research and Mr Julio Romero for his advice on statistics.

Contributors CRK-A and DD conceived the research idea. CRK-A, LTW, DD and RD designed the research. CRK-A, PR and HZ performed the statistical analysis with help from TC. CRK-A drafted the manuscript and LTW, DD and RD commented on it. All authors read and approved the final draft of the manuscript.

Funding A small amount of Australian Government Research Training Scheme money was used.

Competing interests None declared.

Ethics approval The ACT Health Human Research Ethics Committee and the University of Canberra Human Research Ethics Committee (No: 11.167).

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement No additional data are available.

REFERENCES

1. Australian Bureau of Statistics. 4338.0—Profiles of Health, Australia, 2011–13. 2013 (latest issue 07/06/2013).
2. McIntyre HD, Gibbons KS, Flenady VJ, *et al*. Overweight and obesity in Australia mothers: epidemic or endemic? *Med J Aust* 2012;196:184–8.
3. Cunningham C, Teale GR. A profile of body mass index in a large rural Victorian obstetric cohort. *Med J Aust* 2013;198:39–42.
4. Bryant M, Santorelli G, Lawlor DA, *et al*. Comparison of South Asian specific and established BMI thresholds for determining obesity prevalence in pregnancy and predicting pregnancy complications: findings from the Born in Bradford cohort. *Int J Obes* 2014;38:444–50.
5. Callaway LK, Prins JB, Chang AM, *et al*. The prevalence and impact of overweight and obesity in an Australian obstetric population. *Med J Aust* 2006;184:56–9.
6. Magann EF, Doherty DA, Sandlin AT, *et al*. The effects of an increasing gradient of maternal obesity on pregnancy outcomes. *Aust N Z J Obstet Gynaecol* 2013;53:250–7.
7. Scott-Pillai R, Spence D, Cardwell CR, *et al*. The impact of body mass index on maternal and neonatal outcomes: a retrospective study in a UK obstetric population, 2004–2001. *BJOG* 2013;120:932–9.
8. Ovesen P, Rasmussen S, Kesmodel U. Effect of prepregnancy maternal overweight and obesity on pregnancy outcome. *Obstet Gynecol* 2011;118(Pt 1):305–12.
9. Bastian LA, West NA, Corcoran C, *et al*. Number of children and the risk of obesity in older women. *Prev Med* 2005;40:99–104.
10. Amorim AR, Rössner S, Neovius M, *et al*. Does excess pregnancy weight gain constitute a major risk for increasing long-term BMI? *Obesity* 2007;15:1278–86.
11. Rooney BL, Schauberger CW. Excess pregnancy weight gain and long-term obesity: one decade later. *Obstet Gynecol* 2002;100:245–52.
12. Villamor E, Cnattingius S. Interpregnancy weight change and risk of adverse pregnancy outcomes: a population-based study. *Lancet* 2006;368:1164–70.
13. Reynolds RM, Allan KM, Raja EA, *et al*. Maternal obesity during pregnancy and premature mortality from cardiovascular event in adult offspring: follow-up of 1 323 275 person years. *BMJ* 2013;347:f4539.
14. Sridhar SB, Darbinian J, Ehrlich SF, *et al*. Maternal gestational weight gain and offspring risk for childhood overweight or obesity. *Am J Obstet Gynecol* 2014;211:259.e1–8.
15. Ehrlich SF, Hedderson MM, Feng J, *et al*. Change in body mass index between pregnancies and risk of gestational diabetes in a second pregnancy. *Obstet Gynecol* 2011;117:1323–30.
16. Bogaerts A, Van der Bergh BR, Ameye L, *et al*. Interpregnancy weight change and risk of adverse perinatal outcome. *Obstet Gynecol* 2013;122:999–1009.
17. Cnattingius S, Villamor E. Weight change between successive pregnancies and risks of stillbirth and infant mortality: a nationwide cohort study. *Lancet* 2016;387:558–65.
18. The Canberra Hospital statistics. http://www.act.gov.au downloaded information 20 July 2015.
19. World Health Organisation. ICD-10 Version: 2015. International Statistical Classification of Diseases and Related Health Problems 10th Revision. http://apps.who.int/classifications/icd10/browse/2015/en
20. Australian Capital Territory (ACT) Health. Standard Operating Procedure: Safe manual handling of larger bariatric adult patients. No:DGD12-014. www.health.act.gov.au
21. Apgar V. Proposal for new method of evaluation of newborn infant. *Curr Res Anesth Analg* 1953;32:260–7.
22. The Royal Australian and New Zealand College of Obstetricians and Gynaecologists. Management of Obesity in Pregnancy: C-Obs49 College statement. Endorsed March 2013. www.ranzcog.edu.au.
23. Dobbins T, Sullivan EA, Roberts CL, *et al*. Australian national birthweight percentiles by sex and gestational age, 1998–2007. *Med J Aust* 2012;197:291–4.
24. Gunderson EP. Childbearing and obesity in women: weight before, during and after pregnancy. *Obstet Gynec Clin North Am* 2009;36:317–32, ix.
25. Chatkin R, Mottin CC, Chatkin JM. Smoking among morbidly obese patients. *BMC Pulm Med* 2010;10:61.
26. Knight-Agarwal CR, Williams LT, Davis DL, *et al*. The perspectives of obese women receiving antenatal care: a qualitative study of women's' experience. *Women Birth* 2016;29:189–95.
27. Duckitt K, Harrington D. Risk factors of pre-eclampsia at antenatal booking: systematic review of controlled studies. *BMJ* 2005;330:565.
28. Bhattacharya S, Campbell DM, Liston WA, *et al*. Effect of body mass index on pregnancy outcomes in nulliparous women delivering singleton babies. *BMC Pub Health* 2007;7:168.
29. Athukorala C, Rumbold AR, Willson KJ, *et al*. The risk of adverse pregnancy outcomes in women who are overweight or obese. *BMC Pregnancy Childbirth* 2010;10:56.
30. Persson M, Johansson S, Villamor E, *et al*. Maternal overweight and obesity and risks of severe birth-asphyxia-related complications in term infants: a population-based cohort study in Sweden. *PLoS Med* 2014;11:e1001648.
31. Usha Kiran TS, Hemmadi S, Bethel J, *et al*. Outcome of pregnancy in a woman with an increased body mass index. *BJOG* 2005;112:768–72.
32. Gibbons L, Belizán JM, Lauer JA, *et al*. *The global numbers and costs of additionally needed and unnecessary caesarean sections performed per year: overuse as a barrier to universal coverage*. WHO World Health Report, 2010; Background Paper, 30.
33. Poobalan AS, Aucott LS, Gurung T, *et al*. Obesity as an independent risk factor for elective and emergency caesarean delivery in nulliparous women—systematic review and meta-analysis of cohort studies. *Obes Rev* 2009;10:28–35.
34. Han Z, Mulla S, Beyene J, *et al*. Maternal underweight and the risk of preterm birth and low birth weight: a systematic review and meta-analyses. *Int J Epidem* 2011;40:65–101.
35. Cnattingius S, Lambe M. Trends in smoking and overweight during pregnancy: prevalence, risks of pregnancy complications and adverse pregnancy outcomes. *Semin Perinatol* 2002;26:286–95.
36. Cameron CM, Davey TM, Kendall E, *et al*. Changes in alcohol consumption in pregnant Australian women between 2007 and 2011. *Med J Aust* 2013;199:355–7.