Trimester-specific blood pressure levels in relation to maternal pre-pregnancy body mass index

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Summary

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We evaluated the influence of maternal pre-pregnancy body mass index (BMI), based on reported pre-pregnancy weight and height, on blood pressure (BP) levels during pregnancy by using information from a prospective cohort of 1733 women recruited before 20 weeks' gestation. Maternal antenatal BP values were abstracted from medical records, and we evaluated the mean BP differences according to BMI group in regression models, using generalised estimating equations to account for repeated BP records within each pregnancy.

In each trimester, mean systolic BP (SBP) and diastolic BP (DBP) values were positively associated with maternal pre-gestational BMI. This association persisted after adjustment for maternal age, parity, smoking, education, marital status and physical activity. Overweight women (25–29 kg/m^2) had first-, second- and third-trimester mean SBPs that were 8.1, 7.7 and 8.2 mmHg, respectively, higher than values observed in lean women (<20 kg/m^2). Mean DBP values were 4.5, 5.4 and 5.6 mmHg higher for each successive trimester in overweight vs. lean women. Obese (>30 kg/m^2) women consistently had the highest mean SBP and DBP values. Trimester-specific mean SBP values were 10.7–12.0 mmHg higher among obese women vs. lean women. Corresponding trimester-specific mean DBP values were 6.9–7.4 mmHg higher in obese vs. lean women. Similar patterns were observed when trimester-specific average mean arterial pressures were evaluated. Elevated pregnancy BPs associated with maternal pre-gestational BMI are consistent with a large body of literature that documents increased pre-eclampsia risk among overweight and obese women.

Keywords: *maternal prenatal blood pressure*, *maternal body mass index*, *maternal obesity*, *pregnancy*, *mean arterial pressure*.

Introduction

The increasing prevalence of overweight and obesity in the US adult population has become a major public health and clinical concern. In 2001, 58% of adults (i.e. 122.4 million individuals) were considered overweight [body mass index (BMI) 25.0–29.9 kg/m^2] and 21% (i.e. 44.3 million individuals) were defined as obese (BMI ≥ 30.0 kg/m^2).[1] This trend is associated with an increased chronic disease burden that has not been adequately influenced by national efforts to promote healthier eating habits and increased physical activity. Excessive weight has been linked to several metabolic and haemodynamic abnormalities, including dyslipidaemia, elevated blood pressure, impaired glucose tolerance, insulin resistance and clustering of cardiovascular disease factors. Individuals who are overweight or obese have increased risks of hypertension, type 2 diabetes, hypercholesterolaemia, asthma and osteoarthritis.[1,2]

In pregnant women, increased adiposity, as measured using pre-pregnancy BMI, has been consistently associated with important medical complications of

pregnancy, such as pre-eclampsia, gestational diabetes mellitus, abruptio placentae and operative delivery.[3–5] To date, there are few instances where investigators have assessed the extent to which the current obesity epidemic has had clinically important consequences on maternal blood pressure (BP) levels during pregnancy. Therefore, to fill this identified gap in the current literature, we sought to characterise the structure of longitudinal BP in pregnancy as a means to explore, and possibly generate, mechanistic hypotheses underlying epidemiological associations of maternal pre-gestational BMI and adverse pregnancy outcomes.[3–5] We report the distribution of BP levels and the association of BP with pre-pregnancy BMI in a large population of pregnant women with an average of 12.2 clinical BP readings per person during pregnancy and available for study.[6]

Materials and methods

Study design and population

This analysis uses data initially gathered for the Omega Study, an ongoing prospective study examining the metabolic and dietary predictors of pre-eclampsia, gestational diabetes and other pregnancy outcomes.[7,8] The study population was drawn from women attending prenatal care at clinics affiliated with the Swedish Medical Center and Tacoma General Hospital in Seattle and Tacoma, WA, respectively. Recruiting began in December 1996. Details of the study design, data collection procedures and construction of the database used for this research are presented in the companion paper by Thompson *et al*.[6]

Analytical population

The population eligible to be analysed is derived from participants who enrolled in the Omega Study between 1996 and 2002. During this period, 2556 eligible women were approached, and 2000 (78%) agreed to participate. We excluded from this analysis 19 women whose pregnancy ended (miscarriage $n = 14$; induced abortion $n = 5$) prior to the clinical recording of at least one antepartum BP; 56 women for whom we could not locate a clinic or medical record; and 5 women with a gestational age of first prenatal care visit >20 weeks. We also excluded 100 women with chronic hypertension and an additional 21 women with pre-gestational diabetes. Also excluded were 66 women with missing pre-pregnancy BMI values. Thus, 1733 women remained for analysis.

Description of covariates and trimester-specific blood pressure assessment

At the time of enrolment in the Omega Study (12.7 weeks' gestation, on average), a 45- to 60-min structured questionnaire was administered by a trained interviewer. Information was collected on medical and reproductive histories and sociodemographic and lifestyle characteristics. Pre-pregnancy weight and height were based on self-reports made during the interview. Pre-pregnancy BMI, used as a measure of overall maternal adiposity, was calculated as weight in kilograms divided by height in metres squared. Pre-pregnancy BMI was categorised a priori as follows: <20.0 (lean), 20.0–24.9 (high normal), 25.0–29.9 (overweight) and $\geq$30.0 kg/m^2 (obese). Gestational age of pregnancy was determined using maternal self-reported last normal menstrual period, and this date was confirmed by ultrasound prior to 20 weeks' gestation. Pre-eclampsia[9] and gestational diabetes mellitus[10] were defined according to published diagnostic criteria. We used the definition in the literature[11] to define first, second and third trimesters as follows: first trimester <15 weeks; second trimester 15–28 weeks; and third trimester $\geq$29 weeks of gestation.

All pregnancy-associated BP measurements [i.e. systolic BP (SBP) and diastolic BP (DBP)], along with the date and gestational age when the pressure was taken, were abstracted from participants' clinical and hospital medical records. For the purposes of this study, we primarily used antepartum clinical BPs taken and recorded during routine visits. BPs taken upon admission for inpatient observation or to the emergency room were considered only when the BP from an expected antepartum visit was unavailable. BPs taken during active labour or during the postpartum period were not considered in these analyses.

During the study period, many different healthcare providers made BP readings part of routine clinical practice. Although the measures were not strictly standardised as they would be in a clinical trial, BPs were taken using standard mercury sphygmomanometers (scaled to even numbers) and patients were rested and seated during examination. Mean arterial pressure (MAP), considered an integrated parameter of BP, is known to be more reproducible than individual

SBPs and DBPs.[12] We therefore computed mean arterial pressures for each subject according to the following formula: $\text{MAP} = \frac{2}{3}\text{DBP} + \frac{1}{3}\text{SBP}$.

Statistical analyses

The BP record (SBP, DBP, MAP) was the dependent variable, and categorical BMI the primary covariate. Linear regression models were fitted using generalised estimating equations to adjust for repeated BP measurements on the same woman.[13] Based on exploratory investigation of the correlation between repeated measurements, an exchangeable correlation structure was assumed for all analyses. All models were fitted with trimester as an effect modifier. Robust estimates of the standard errors were used throughout. Test statistics were constructed as the ratio of the relevant point estimate to its robust standard error and associated *P*-values calculated from normal tables. Statistical significance was defined at *P*-value <0.05. Analyses were carried out using STATA Software, version 9.2.[14]

Results

The sociodemographic characteristics of the study cohort, overall and by BMI category, are presented in Table 1. Overall, participants included in this analysis tended to be Caucasian, well-educated and married.

Trimester-specific mean SBP, DBP and MAP values are reported in Table 2. Mean SBP increased across trimesters for the entire cohort. Second- and third-trimester SBP values were both statistically

Table 1. Characteristics of the study cohort according to categories of maternal pre-pregnancy body mass index, Seattle and Tacoma, WA, 1996–2002

Characteristics	Lean (<20 kg/m^2) (n = 358) %	High normal (20–24.9 kg/m^2) (n = 997) %	Overweight (25–29.9 kg/m^2) (n = 249) %	Obese (≥30 kg/m^2) (n = 129) %	Total cohort (n = 1733) %
Maternal age (years)					
<20	1.1	0.5	0.8	3.1	0.9
20–34	74.3	71.7	69.5	69.8	71.8
≥35	24.6	27.8	29.7	27.1	27.4
Maternal race/ethnicity					
Non-Hispanic white	83.8	87.1	84.6	77.5	85.3
African American	1.4	0.7	4.9	4.7	1.7
Asian	10.6	7.0	6.1	5.4	7.5
Other	4.2	5.2	4.4	12.4	5.4
Multiparous	31.0	30.9	34.5	35.7	31.8
Less than 12 years' education	4.6	4.2	4.9	9.4	4.8
Unmarried	10.6	8.7	11.7	12.4	9.8
Annual household income (US$)					
>70 000	74.4	72.6	63.2	56.7	70.5
30 000–69 999	19.8	24.5	29.8	31.7	24.8
<30 000	5.8	2.9	7.0	11.4	4.7
Smoked during pregnancy	5.0	6.0	9.2	8.5	6.5
Physically inactive during pregnancy	16.5	16.4	16.5	19.4	16.6
Incident pre-eclampsia	3.3	3.2	7.6	10.0	4.3
Incident GDM	1.5	3.7	4.2	13.2	4.0
Gestational age at delivery (weeks)					
<28[a]	7.5	3.7	4.0	7.8	4.9
28–36	7.0	10.4	9.2	10.1	9.5
37–40	72.4	71.5	71.5	69.9	71.5
>40	13.1	14.3	15.3	13.2	14.1

[a]Including pregnancies ending in miscarriage, induced abortion or fetal death.
GDM, gestational diabetes mellitus.

Table 2. Maternal trimester-specific mean (SE) systolic, diastolic and mean arterial blood pressures, for members of the study cohort, Seattle and Tacoma, WA, 1996–2002

	Systolic blood pressure		Diastolic blood pressure		Mean arterial pressure	
	Mean (SE)	[95% CI]	Mean (SE)	[95% CI]	Mean (SE)	[95% CI]
Trimester 1	112.7 (0.2)	[112.3, 113.2]	69.8 (0.2)	[69.5, 70.1]	84.1 (0.2)	[83.8, 84.5]
Trimester 2	113.8 (0.2)	[113.4, 114.2][a]	68.9 (0.1)	[68.6, 69.2][a]	83.9 (0.1)	[83.6, 84.2][c]
Trimester 3	116.4 (0.2)	[116.0, 116.9][b]	72.0 (0.2)	[71.7, 72.3][b]	86.8 (0.2)	[86.5, 87.1][b]

[a]*P*-values < 0.0005 comparing second vs. first trimester values.
[b]*P*-values < 0.0005 comparing third vs. first trimester values.
[c]*P*-value = 0.09 comparing second vs. first trimester values.

Table 3. Unadjusted and adjusted mean (SE) systolic blood pressure, according to trimester and pre-pregnancy body mass index (BMI). Seattle and Tacoma, WA, 1996–2002

	First trimester		Second trimester		Third trimester	
	Mean (SE)[a]	Mean difference [95% CI]	Mean (SE)[a]	Mean difference [95% CI]	Mean (SE)[a]	Mean difference [95% CI]
BMI group	Unadjusted		Unadjusted		Unadjusted	
Lean	108.7 (0.5)	0.0 Reference	110.5 (0.4)	0.0 Reference	112.5 (0.5)	0.0 Reference
High-normal	112.1 (0.3)	3.4 [2.3, 4.5]	113.0 (0.3)	2.6 [1.6, 3.5]	115.8 (0.3)	3.3 [2.3, 4.3]
Overweight	116.8 (0.6)	8.1 [6.6, 9.5]	118.2 (0.5)	7.7 [6.4, 9.0]	120.7 (0.6)	8.2 [6.7, 9.7]
Obese	120.7 (0.9)	12.0 [10.1, 14.0]	121.2 (0.7)	10.7 [9.2, 12.3]	123.6 (0.9)	11.0 [9.1, 13.0]
BMI group	Adjusted		Adjusted		Adjusted	
Lean	109.4 (0.6)	0.0 Reference	111.2 (0.6)	0.0 Reference	113.2 (0.6)	0.0 Reference
High-normal	112.8 (0.5)	3.4 [2.3, 4.5]	113.7 (0.5)	2.5 [1.6, 3.5]	116.5 (0.5)	3.2 [2.2, 4.3]
Overweight	117.5 (0.7)	8.1 [6.6, 9.6]	118.8 (0.7)	7.6 [6.3, 8.9]	121.1 (0.8)	7.9 [6.4, 9.4]
Obese	121.5 (1.0)	12.1 [10.1, 14.0]	121.8 (0.8)	10.6 [9.1, 12.2]	124.0 (0.9)	10.8 [9.0, 12.6]

[a]The adjusted mean values reported in this table are for Non-Hispanic white, nulliparous, college educated women who are 20 ,34 years of age, married, non-smokers and physically inactive during pregnancy.

significantly higher than the mean values for the first trimester. However, a different pattern was noted for trimester-specific DBP values. We noted that second-trimester mean DBP was lower than that recorded in the first trimester, and these differences were statistically significant. Mean third-trimester DBP was statistically significantly higher than in the first trimester. This J-shaped pattern of mean DBP across trimesters is consistent with reports in the literature.[15] The corresponding MAP means by trimester are also shown, and the pattern of changes across trimesters is similar to those seen for DBP.

In Table 3, we summarise results from our analyses designed to assess trimester-specific mean SBPs according to the four maternal pre-pregnancy BMI categories. Within each trimester, maternal pre-pregnancy BMI was positively associated with mean SBP. Furthermore, mean SBP in each BMI category was significantly elevated relative to that of lean women and relative to the neighbouring (leaner) category. Using lean women as the reference group (BMI <20 kg/m^2), we noted that mean SBP values were 12.0, 10.7 and 11.0 mmHg higher for each trimester, respectively, among obese women (BMI ≥30 kg/m^2). Differences in trimester-specific means remained statistically significant after we adjusted for confounding by maternal age, race/ethnicity, parity, educational attainment and marital status, as well as physical activity and smoking status during pregnancy. We noted that overweight women had higher mean SBP values for each trimester compared with lean women (8.1, 7.7 and 8.2 mmHg higher mean SBP values for each successive trimester) after adjusting for confounders. Even women within BMI values of 20.0–24.9 kg/m^2

Table 4. Unadjusted and adjusted mean (SE) diastolic blood pressure, according to trimester and pre-pregnancy body mass Index (BMI). Seattle and Tacoma, WA, 1996–2002

	First trimester		Second trimester		Third trimester	
	Mean (SE)[a]	Mean difference [95% CI]	Mean (SE)[a]	Mean difference [95% CI]	Mean (SE)[a]	Mean difference [95% CI]
BMI group	Unadjusted		Unadjusted		Unadjusted	
Lean	67.6 (0.3)	0.0 Reference	66.5 (0.3)	0.0 Reference	69.4 (0.3)	0.0 Reference
High-normal	69.4 (0.2)	1.7 [1.0, 2.5]	68.4 (0.2)	1.9 [1.3, 2.6]	71.6 (0.2)	2.3 [1.5, 3.0]
Overweight	72.1 (0.4)	4.5 [3.4, 5.5]	71.8 (0.4)	5.4 [4.5, 6.3]	74.9 (0.4)	5.6 [4.5, 6.6]
Obese	75.0 (0.5)	7.3 [6.1, 8.6]	73.9 (0.6)	7.4 [6.1, 8.7]	76.3 (0.6)	6.9 [5.5, 8.3]
BMI group	Adjusted		Adjusted		Adjusted	
Lean	68.1 (0.5)	0.0 Reference	66.8 (0.4)	0.0 Reference	69.9 (0.5)	0.0 Reference
High-normal	69.7 (0.4)	1.6 [0.9, 2.4]	68.7 (0.4)	1.9 [1.2, 2.6]	71.9 (0.4)	2.1 [1.3, 2.8]
Overweight	72.5 (0.5)	4.4 [3.4, 5.5]	72.2 (0.5)	5.4 [4.4, 6.3]	75.2 (0.6)	5.4 [4.3, 6.5]
Obese	75.5 (0.7)	7.4 [6.2, 8.7]	74.4 (0.7)	7.6 [6.3, 8.9]	76.6 (0.7)	6.8 [5.4, 8.1]

[a]The adjusted mean values reported in this table are for Non-Hispanic white, nulliparous, college educated women who are 20, 34 years of age, married, non-smokers and physically inactive during pregnancy.

Table 5. Unadjusted and adjusted mean (SE) of mean arterial pressure, according to trimester and pre-pregnancy body mass index (BMI). Seattle and Tacoma, WA, 1996–2002

	First trimester		Second trimester		Third trimester	
	Mean (SE)[a]	Mean difference [95% CI]	Mean (SE)[a]	Mean difference [95% CI]	Mean (SE)[a]	Mean difference [95% CI]
BMI group	Unadjusted		Unadjusted		Unadjusted	
Lean	81.3 (0.3)	0.0 Reference	81.1 (0.3)	0.0 Reference	83.8 (0.3)	0.0 Reference
High-normal	83.6 (0.2)	2.3 [1.5, 3.1]	83.3 (0.2)	2.1 [1.5, 2.8]	86.4 (0.2)	2.6 [1.8, 3.4]
Overweight	87.0 (0.4)	5.7 [4.6, 6.7]	87.3 (0.4)	6.2 [5.2, 7.1]	90.2 (0.5)	6.4 [5.3, 7.6]
Obese	90.2 (0.6)	8.9 [7.6, 10.2]	89.7 (0.6)	8.5 [7.3, 9.8]	92.1 (0.7)	8.3 [6.8, 9.8]
BMI group	Adjusted		Adjusted		Adjusted	
Lean	81.9 (0.5)	0.0 Reference	81.6 (0.5)	0.0 Reference	84.3 (0.5)	0.0 Reference
High-normal	84.1 (0.4)	2.2 [1.4, 3.0]	83.7 (0.4)	2.1 [1.4, 2.8]	86.8 (0.4)	2.4 [1.7, 3.2]
Overweight	87.5 (0.6)	5.7 [4.6, 6.7]	87.7 (0.5)	6.1 [5.1, 7.1]	90.5 (0.6)	6.2 [5.1, 7.4]
Obese	90.9 (0.7)	9.0 [7.7, 10.3]	90.2 (0.6)	8.6 [7.4, 9.8]	92.4 (0.7)	8.1 [6.8, 9.5]

[a]The adjusted mean values reported in this table are for Non-Hispanic white, nulliparous, college educated women who are 20, 34 years of age, married, non-smokers and physically inactive during pregnancy.

(although generally classified as having a 'high normal' BMI) had trimester-specific mean SBPs that were 2.6–3.4 mmHg higher than values observed among lean women (BMI <20 kg/m^2).

A positive relationship between maternal pre-pregnancy BMI with trimester-specific mean DBP and MAP was also observed (Tables 4 and 5 respectively). Mean first-trimester DBP, before adjusting for confounders, was lowest for lean women (67.6, standard error [SE] 0.3, mmHg) and highest for obese women (75.0, SE 0.5 mmHg). Mean DBP in each BMI category was significantly elevated relative to that of lean women and relative to the neighbouring (leaner) category. As can been seen in the second part of Table 4, significant differences in first-trimester mean DBP values across BMI categories remained after we controlled for potential confounders (i.e. maternal age, race/ethnicity, parity, educational attainment, marital status, smoking and physical activity during pregnancy). This pattern of increasing mean DBP with increasing pre-pregnancy BMI held for all three trimesters. We noted similar patterns for the integrated measure of maternal BP, MAP, to those observed for SBP and DBP. Obese women, as compared with lean

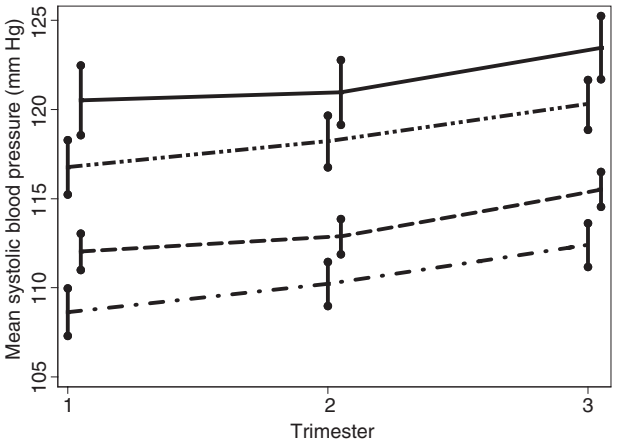

Figure 1. Association between maternal trimester-specific mean systolic blood pressure. The vertical lines represent 95% confidence intervals. The positions of the high-normal and obese trimester means have been perturbed for visual clarity. Lean, <20kg/m^2 — · — · —; High normal, 20–24.9kg/m^2 ------; Overweight, 25–29.9kg/m^2 — ·· —; Obese, ≥30.0kg/m^2 ——.

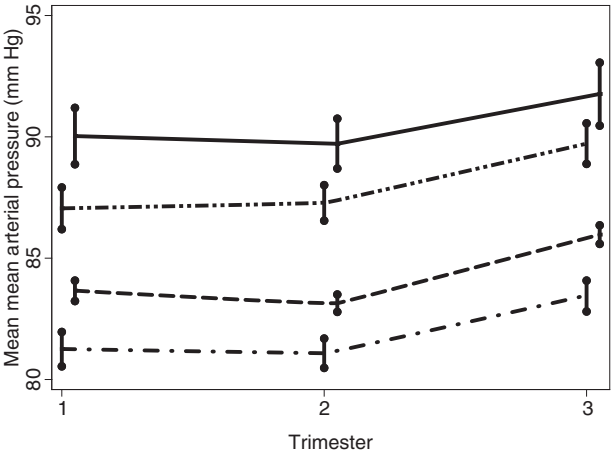

Figure 3. Association between maternal trimester-specific mean arterial pressure. The vertical lines represent 95% confidence intervals. The positions of the high-normal and obese trimester means have been perturbed for visual clarity. Lean, <20kg/m^2 — · — · —; High normal, 20–24.9kg/m^2 ------; Overweight, 25–29.9kg/m^2 — ·· —; Obese, ≥30.0kg/m^2 ——.

women, had higher mean MAP values for each trimester (8.9, 8.5 and 8.3 mmHg). A graphical summary of the associations between maternal trimester-specific mean SBP, DBP and MAP is presented in Figures 1–3. Exclusion of women with pre-eclampsia from these analyses did not materially alter observed relationships between pregnancy BPs and pre-pregnancy BMI (data not shown).

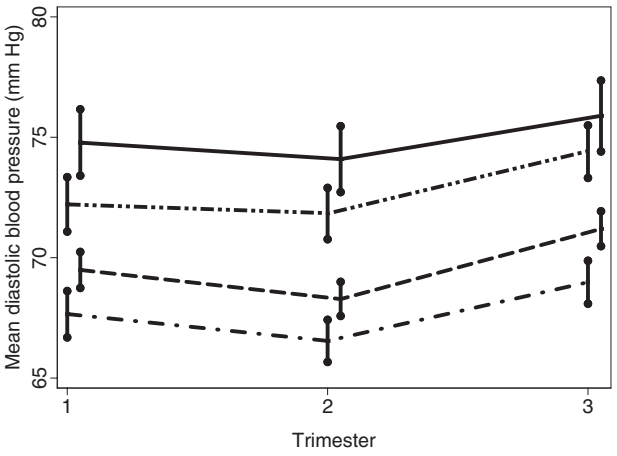

Figure 2. Association between maternal trimester-specific mean diastolic blood pressure. The vertical lines represent 95% confidence intervals. The positions of the high-normal and obese trimester means have been perturbed for visual clarity. Lean, <20kg/m^2 — · — · —; High normal, 20–24.9kg/m^2 ------; Overweight, 25–29.9kg/m^2 — ·· —; Obese, ≥30.0kg/m^2 ——.

Discussion

Highly statistically significant trends in trimester-specific mean SBP, DBP and MAP were observed across categories of maternal pre-pregnancy BMI. For instance, the differences in mean MAP for obese compared with lean women for each trimester were 9.0, 8.6 and 8.1 mmHg, respectively, after adjustment for maternal age, race/ethnicity, parity, educational and income status, as well as smoking and physical activity habits during pregnancy. Overall, our findings are compatible with a larger body of evidence documenting a strong association between increasing adiposity and blood pressure values in all populations studied during the current obesity epidemic.[1,16–18]

Several important limitations should be considered when interpreting the results of our study. First, although we adjusted for several potential confounders, we cannot exclude the possibility of residual confounding due to misclassification of adjusted variables or confounding by other unmeasured variables. For instance, we were unable to assess the impact of maternal weight gain during pregnancy, a factor that may influence blood pressure changes throughout pregnancy. Data sets are needed which allow for careful characterisation of maternal net weight gain for each trimester and which account for weight-gain changes secondary to medical complications of pregnancy (e.g. oedema, polyhydramnios, oligohydramnios). Second,

we were not able to assess trimester-specific BP changes in relation to distribution of maternal fat. It is important to note, however, that BMI is highly correlated with intra-abdominal (visceral) fat assessed using computerised tomography in reproductive-aged women participating in an ancillary study of the CARDIA project.[19]

Third, maternal weight and height were self-reported, so we cannot exclude the possibility of some misclassification. We know that self-reported bodyweight tends to be underestimated. In our study, this would mean that some women were falsely categorised in lower BMI categories than they should have been. Such under-reporting would lead to underestimation of the true association between mean trimester-specific BP and pre-pregnancy BMI values. The generalisability of our study may also be limited, as our cohort was primarily composed of non-Hispanic white and well-educated women. The concordance of our results with those from other studies that have included racially, ethnically and geographically diverse populations has served to attenuate somewhat these concerns.[20–22] Our results are comparable to those of Stervens *et al*., who recently reported that SBP and DBP levels are positively associated with BMI among pregnant women receiving antepartum care in Lund, Sweden.[21] Our findings are also consistent with reports from Ma and Lao, who have, in their study of Chinese women in Hong Kong, noted statistically significant positive correlations between maternal pre-pregnancy BMI and MAP values recorded throughout pregnancy.[20]

BP measurement plays a central role in the screening and management of hypertension during pregnancy.[23,24] As noted by Bergel *et al*., the validity of conventional (clinic) BP measurement has been questioned and efforts have been made to improve technique with ambulatory automated devices that provide a large number of measurements over a period of time, usually a 24-h period.[24] To date, however, no randomised trials that have evaluated the two methods have been published, and clinical BP measurements taken during antepartum visits continue to form the basis upon which clinical diagnoses of pre-eclampsia and other hypertensive diagnoses are made in clinical settings throughout the world. Because we used clinical BP measurements abstracted from medical records, errors and unmeasured sources of variation in BP ascertainment may have influenced our findings. For instance, if some healthcare providers used standard-size arm cuffs on obese patients, we would expect that BPs in this context may be overestimated. Use of incorrect cuff sizes, however, cannot completely explain our findings, as the BMI–BP effects are seen even for overweight women and those with high-normal BMI. In these two comparisons, the likelihood of women with arm circumference values exceeding 34 cm is exceedingly low, and thus observed differences cannot be attributable to cuff-size errors.

We are not aware of other studies that have assessed trimester-specific BP changes in relation to maternal pre-pregnancy BMI. The pattern of increasing BP with increasing BMI has, however, been noted in non-pregnant women.[16,17] Furthermore, the continuous longitudinal association between BP and maternal pre-gestational BMI during pregnancy is currently unknown. Work in progress considering BP records at individual gestational ages (rather than grouped by trimester), however, indicates that the nature of the association between BP and maternal pre-gestational BMI changes with gestational age (Thompson, personal communication).

The mechanisms by which excess weight may lead to elevated BP are poorly understood. Disturbances in autonomic function and, in particular, sympathetic nervous system hyperactivity have been postulated as being possibly important mechanisms of the consistently observed statistical associations.[25,26] Emerging evidence, however, suggests that sympathetic overactivation leads to hypertension and adult weight gain which further contributes to worsening of hypertension.[27–29] Alternatively, adiposity-related insulin resistance may indirectly influence BP as hyperinsulinaemia is known to be positively associated with increases in BPs, particularly SBP.[16] Whatever the mechanisms, the positive relationship between maternal pre-pregnancy adiposity and trimester-specific BP values is substantial. This association explains, in part, the increased predisposition of overweight and obese women to pre-eclampsia and other hypertensive disorders of pregnancy. The observed increases in BP even in non-obese women suggest that public health efforts in the US directed towards encouraging all children, adolescents, young adults and pregnant women to exercise, consume healthy diets and avoid adult weight gain, may result in improved BP profiles during pregnancy. Such improved profiles may lead to reductions in obesity-associated medical complications of pregnancy, including pre-eclampsia, gestational diabetes mellitus and operative deliveries.

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