Preeclampsia and Gestational Hypertension Are Associated With Childhood Blood Pressure Independently of Family Adiposity Measures

The Avon Longitudinal Study of Parents and Children

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Background—Offspring of women with hypertensive disorders of pregnancy are at increased risk of cardiovascular complications later in life, but the mechanisms underlying these associations are unclear. Our aim was to examine whether adjusting for birth weight and familial adiposity changed the association of hypertensive disorders of pregnancy with offspring blood pressure.

Methods and Results—Using data from 6343 nine-year-old participants in the Avon Longitudinal Study of Parents and Children, we examined the association between hypertensive disorders of pregnancy (preeclampsia and gestational hypertension) and offspring blood pressure. Both preeclampsia and gestational hypertension were associated with systolic and diastolic blood pressures in the 9-year-old offspring; after adjustment for parental and own adiposity and for other potential confounders, the mean difference in systolic blood pressure was 2.05 mm Hg (95% confidence interval, 0.72 to 3.38) and 2.04 mm Hg (95% confidence interval, 1.42 to 2.67) for preeclampsia and gestational hypertension, respectively, compared with those with no hypertensive disorders of pregnancy. Equivalent results for diastolic blood pressure were 1.00 mm Hg (95% confidence interval, 0.01 to 2.10) and 1.07 mm Hg (95% confidence interval, 0.60 to 1.54). The association of preeclampsia with offspring systolic and diastolic blood pressures attenuated toward the null with further adjustment for birth weight and gestational age, whereas these adjustments did not attenuate the association of gestational hypertension with offspring blood pressure.

Conclusions—The associations of hypertensive disorders of pregnancy with higher offspring blood pressure are not explained by familial adiposity. The mechanisms linking preeclampsia and gestational hypertension with offspring blood pressure may differ, with the former mediated at least in part by the effect of preeclampsia on intrauterine growth restriction. (Circulation. 2010;122:1192-1199.)

Key Words: adiposity ■ birth weight ■ blood pressure ■ gestational hypertension ■ offspring ■ preeclampsia

Hypertensive disorders of pregnancy, consisting of preeclampsia and gestational hypertension, are the most common complications of pregnancy and are associated with adverse health outcomes for the mother and her offspring. Preeclampsia, diagnosed by newly elevated blood pressure (BP) and proteinuria after 20 weeks of gestation, occurs in approximately 2% to 7% of otherwise healthy nulliparous women. It is an important cause of perinatal deaths, preterm birth, and intrauterine growth restriction. Gestational hypertension is defined as newly elevated BP after 20 weeks of gestation but without proteinuria. Found to occur in 6% to 17% of pregnancies, it is also associated with preterm delivery and infants who are small for their gestational age.
with preeclampsia had higher diastolic BPs (DBP) than control children born to mothers without preeclampsia. In contrast, Seidman et al.\(^8\) concluded that 17-year-old daughters of mothers with preeclampsia had higher SBP and DBP than control female subjects, whereas only the mean SBP differed between the male subjects. Reports of gender differences such as these are often chance findings, and 2 further cohorts that have reported positive associations of hypertensive disorders of pregnancy with offspring BP in childhood reported no gender differences.\(^9\)\(^-\)\(^11\) In the second of these studies, there were positive associations with both random BP and 24-hour BP in offspring.\(^10\)\(^,\)\(^11\)

The underlying mechanisms explaining the associations of gestational hypertension and preeclampsia with offspring BP remain unclear. To the best of our knowledge, previous studies have not examined whether associations differ between preeclampsia and gestational hypertension. Given the strong association of maternal obesity with preeclampsia and gestational hypertension risk,\(^12\)\(^,\)\(^13\) as well as with BP in general (including in children),\(^14\) together with familial clustering of adiposity,\(^15\)\(^,\)\(^16\) family adiposity as a consequence of either genetic predisposition or shared lifestyle might be important in the mechanism linking maternal hypertensive disorders of pregnancy with offspring BP. Furthermore, the epidemiological and pathophysiological links between preeclampsia and intrauterine growth restriction and the positive associations of birth weight with later fat mass and body mass index (BMI)\(^17\) but inverse association of birth weight with BP could provide a link between preeclampsia and offspring BP. The aim of this study was to examine the associations of gestational hypertension and preeclampsia with offspring BP at 9 years of age and to study whether these associations are changed by adjustment for parental and offspring anthropometry. The Figure shows the pathways that we have examined in this study.

**Methods**

**Study Population**

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a prospective population-based birth cohort study that recruited 14,541 pregnant women resident in Avon, UK, with expected delivery dates of April 1, 1991, to December 31, 1992 (http://www.alspac.bris.ac.uk.).\(^18\) The overall aim of ALSPAC was to explore the parental, environmental, lifestyle, and genetic determinants of health and disease from infancy to old age. There were 13,678 mother-offspring pairs from singleton live births who survived to at least 1 year of age; only singleton pregnancies were considered here. Of these women, 94% gave consent for abstraction of data from their obstetric records. A total of 7722 children attended the 9-year follow-up clinic, which is ~65% of those invited (participants who were still alive and still agreed to be involved with the study). In total, 6668 mother-offspring pairs had abstracted maternal antenatal data and an offspring who attended the 9-year follow-up clinic. Of the 6668 eligible mother-offspring pairs, complete data on preeclampsia, hypertensive disorders of pregnancy, offspring anthropometry, and BP were available for 6343 (95%), and they form the main analysis cohort for this study. Ethical approval for all aspects of data collection was obtained from the ALSPAC Law and Ethics Committee (IRB 00003312) and the local research ethics committee.

**Maternal Pregnancy Data**

Six trained research midwives abstracted data from obstetric medical records. There were no between-midwife variations in mean values of abstracted data, and repeated data entry checks demonstrated error rates consistently <1%. Obstetric data abstractions included every measurement of SBP and DBP entered into the medical records and the corresponding gestational age and date at the time of the BP measurement. These measurements were obtained in routine clinical practice by trained midwives and obstetricians. The median number of BP measurements in pregnancy was 12 (interquartile range, 10 to 14) and that of urine measurements was 11 (interquartile range, 10 to 14). We applied the International Society for the Study of Hypertension in Pregnancy criteria to all of the clinic data to determine women with preeclampsia and those with gestational hypertension. With these criteria, preeclampsia was defined as an SBP >139 mm Hg or a DBP >89 mm Hg measured on at least 2 occasions after 20 weeks of gestation with proteinuria, diagnosed if the protein reading on dipstick testing (Albustix; Ames Co, Elkhart, Ind) was at least 1+, occurring at the same time as the elevated BP.\(^19\) Gestational hypertension was defined as the same pattern of elevated BP but without proteinuria occurring with the elevated BP. Thus, all women were categorized into 1 of 3 mutually exclusive categories: no hypertensive disorder of pregnancy, gestational hypertension, and preeclampsia. A total of 12 women with either preeclampsia or gestational hypertension and 9 with no evidence of hypertensive disorder of pregnancy reported a previous diagnosis of hypertension unrelated to pregnancy. None of these women were on antihypertensive treatment, and when they were excluded from the analyses, the results were unchanged from those presented here.

**Offspring Measurements at the 9-Year Follow-Up Clinic**

The current ages of the children in months were recorded as they arrived at the assessment clinic. Offspring BP was measured with a Dinamap 9301 vital signs monitor. Two readings of SBP and DBP were recorded with the child at rest, and the mean of each was used. All BP measurements were completed by trained staff using the appropriate cuff size. A repeat measurement was taken in a random 3% of the sample within 2 weeks of the original measurement, and there was a coefficient of variation of 2%.

Weight and height were measured in light clothing and without shoes. Weight was measured to the nearest 0.1 kg with Tanita scales. Height was measured to the nearest 0.1 cm with a Harpenden stadiometer. Waist circumference was measured to the nearest 1 mm.
Potential Covariates
Maternal age, parity, mode of delivery (caesarean section/vaginal delivery), and the child’s sex and birth weight were obtained from the obstetric records. At the time of recruitment, mothers were asked to report their prepregnancy weight and height, which were used to calculate maternal prepregnancy BMI. Maternal self-report of prepregnancy weight and measured weight at the first antenatal clinic were highly correlated (Pearson correlation coefficient = 0.95; P < 0.0001). At the time of recruitment, mothers were also asked to pass a questionnaire to the father of the child. In this questionnaire, the father was asked to record his height, weight, and date of birth. BMI (at the start of his partner’s pregnancy) was calculated from the father’s reported weight and height. Based on questionnaire responses, the highest parental occupation was used to allocate the children to family social class groups (class I [professional/managerial] to 5 [unskilled manual workers] using the 1991 British Office of Population and Census Statistics classification). Mothers were repeatedly asked about their smoking throughout pregnancy, and these data were used to generate a categorical variable: never smoked, smoked before pregnancy or in the first trimester and stopped, or smoked throughout pregnancy. Any note of diabetes mellitus during the pregnancy in the medical records was recorded, and women were categorized as having existing diabetes (already known to have diabetes before the start of pregnancy), gestational diabetes (a new diagnosis of diabetes during the pregnancy noted in the medical records), or no evidence of diabetes.

Statistical Analysis
Maternal, paternal, and offspring characteristics are presented across the 3 categories of no hypertensive disorder of pregnancy, gestational hypertension, and preeclampsia. F tests and χ² tests were used to test for statistical evidence of differences between these 3 categories.

We created age- and sex-specific z scores for all of the adiposity measures using our own cohort as the standard. This enabled us to investigate whether the magnitude of the relationship between hypertensive disorders of pregnancy and adiposity differs between the 3 adiposity measures because all results were on the SD scale. For parental adiposity, it also allowed comparisons between maternal-offspring and paternal-offspring associations. Repeating analyses with paternal BMI on its original scale or offspring anthropometry standardized to the UK 1990 reference charts did not alter any of the results.

We explored each of the individual associations shown in the Figure. In each set of analyses, the characteristic at the point of the arrow is the outcome and that at the blunt end is the exposure. To examine the association of parental adiposity with hypertensive disorders of pregnancy, we used the same approach as that for other parental characteristics (described above; see Table 1). We then extended this by using multinomial regression to obtain the odds ratio (OR) for gestational hypertension and preeclampsia by parental characteristics (Table 2). We then used linear (for continuous outcomes) and logistic (binary outcomes) regression to examine associations of hypertensive disorders of pregnancy with offspring birth weight, gestational age, adiposity, and BP (Tables 3 through 5). For associations of hypertensive disorders of pregnancy with later offspring anthropometric and BP outcomes, we examined potential confounding in a series of multivariable models (Tables 4 and 5).

In all models, we adjusted first for offspring gender and age at examination (model 1). Subsequently, we adjusted for potential confounding factors (maternal age, parity, smoking during pregnancy, paternal BMI, family socioeconomic position [parental education and occupational class], and, in models with fat mass or lean mass for offspring, height and height squared; model 2). Because there were only 26 women (0.4%) with diagnosed gestational diabetes and 39 women (0.6%) with preexistent diabetes, we did not consider gestational diabetes a confounding variable because these numbers would be too small to have any important effect on the associations examined.

Finally, we adjusted for potential mediators (model 3). Mediators were considered characteristics that occur after the main exposure variables (here hypertensive disorder of pregnancy) and are believed to be caused by them and in turn are believed to cause the main outcome of interest (here offspring anthropometry and BP). We considered gestational age at birth, birth weight, and mode of delivery to be potential mediators of the main association because hypertensive disorder of pregnancy can cause preterm birth (by induction of labor) and low birth weight (by shared pathophysiology) and because sibling studies suggest that they may be causally (via intrauterine mechanisms) related to later offspring BP. Mediation, examined using the method suggested by Baron and Kenny, was assumed to occur if the magnitude of the confounder-adjusted association attenuated toward the null by at least 33% with adjustment for potential mediators.

We consider the confounder-adjusted model (model 2) to be the main estimate of a potential causal effect of hypertensive disorders during pregnancy on our outcome. Results are presented jointly for mothers of female and male offspring because there was no strong and consistent evidence of interactions with gender in any of the associations examined (all P > 0.1). The multivariable models were completed only on those participants with complete data on all variables included in any model (n = 3876). Associations were essentially the same for minimally adjusted models (eg, model 1) in this subgroup and in the whole analysis cohort. All statistical analyses were performed with the SPSS version 15.0 for Windows (SPSS Inc, Chicago, Ill).

Results
Table 1 presents the characteristics of mother and offspring by hypertensive disorders of pregnancy categories. Of the 6668 women included in this study, 205 women (3.1%) were diagnosed with preeclampsia, and 1118 women (17.3%) had gestational hypertension. The frequency of hypertensive disorders of pregnancy did not differ among mothers included in this study and mothers not included because of missing outcome or covariable data (202 [3.6%] of the 5609 excluded women had preeclampsia [P for difference = 0.10] and 920 [16.4%] of the 5609 excluded women had gestational hypertension [P for difference = 0.59]).

Table 2 shows the associations of maternal characteristics and paternal BMI with preeclampsia and gestational hypertension. Both preeclampsia and gestational hypertension were more common in women in their first pregnancy and were less common in women who smoked in pregnancy. Maternal BMI was positively associated with both preeclampsia and gestational hypertension. The associations of nulliparity, smoking, and maternal BMI all appeared stronger for preeclampsia than for gestational hypertension, but there was no strong statistical evidence that any of these associations differed from each other (P for heterogeneity between the 2 estimates all >0.3). Paternal BMI was associated with an increased risk of gestational hypertension, but this association attenuated toward the null after adjustment for maternal BMI (maternal BMI–adjusted association of paternal BMI with gestational hypertension, 1.03; 95% confidence interval [CI], 1.01 to 1.06). Social class was not associated with either hypertensive disorder of pregnancy.

Birth weight was inversely associated with SBP (mean difference per 1 kg birth weight after adjustment for sex, age, and BMI at the time of BP assessment: −1.15 mm Hg; 95%
CI, −1.55 to −0.08; with DBP: −0.04 mm Hg; 95% CI, −0.08 to −0.01). Gestational age was also inversely associated with SBP (−0.28 mm Hg; 95% CI, −0.39 to −0.16 per completed week) but not with DBP (−0.04 mm Hg; 95% CI, −0.12 to 0.05).

Table 3 shows the unadjusted associations of maternal hypertensive disorders of pregnancy with offspring characteristics. Preeclampsia was associated with both lower birth weight and preterm birth. These associations attenuated after adjustment for mode of delivery and gestational age at birth.

<table>
<thead>
<tr>
<th>Table 3. Unadjusted Associations of Maternal Hypertensive Disorders of Pregnancy (n=6668)</th>
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<td>---------------------------------------------------------------</td>
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<tr>
<td>Mothers</td>
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<td>Age at delivery, y</td>
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<td>BMI, kg/m²</td>
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<td>Weight, kg</td>
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<td>Cesaréan section, n (%)</td>
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<td>Preterm birth (&lt;37 wk), n (%)</td>
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<td>No previous pregnancies, n (%)</td>
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<td>Smoked throughout pregnancy, n (%)</td>
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<td>Manual social class, n (%)</td>
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<td>Fathers</td>
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<td>Age, y</td>
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<td>BMI, kg/m²</td>
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<td>Offspring</td>
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<td>Male, %</td>
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<td>Birth weight, g</td>
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<td>Age at visit, mo</td>
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<td>Overweight or obese based on BMI, n (%)</td>
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<td>Waist circumference, cm</td>
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<td>Total body fat mass, g</td>
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<td>Centrally obese, n (%)</td>
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Values are means (SD), medians (95% range), or number (%) as appropriate. *The χ² or F test for the null hypothesis of no differences across the 3 categories (ie, 2 df).
Table 3. Unadjusted Associations of Hypertensive Disorders of Pregnancy With Offspring Characteristics (n=6668)

<table>
<thead>
<tr>
<th>Measure at 9-year follow-up clinic</th>
<th>No Hypertensive Disorder of Pregnancy (n=5345)</th>
<th>Gestational Hypertension (n=1116)</th>
<th>Preeclampsia (n=265)</th>
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<td>Reference</td>
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<td>Reference</td>
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<tr>
<td>Birth weight, mean difference, g</td>
<td>–10.3 (–43.9–23.3)</td>
<td>–341.9 (–416.0–267.9)</td>
<td></td>
</tr>
<tr>
<td>Preterm birth, OR</td>
<td>1.07 (0.79–1.45)</td>
<td>6.39 (4.50–9.08)</td>
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</table>

Values are means of differences or ORs (95% CIs) and reflect the difference in offspring characteristic in women with either preeclampsia or gestational hypertension compared with women without any hypertensive disorder of pregnancy (reference group) (ie, in this table, hypertensive disorder of pregnancy is the exposure, and offspring characteristics are the outcomes).

(for birth weight) or birth weight (for preterm delivery), but positive associations remained. Gestational hypertension was not associated with birth weight or preterm delivery. Gestational hypertension was associated with greater offspring adiposity at follow-up, whereas preeclampsia did not show strong evidence of association with offspring adiposity. Preeclampsia was inversely associated with lean mass. In these unadjusted associations, preeclampsia and gestational hypertension were both positively associated with offspring SBP and DBP.

Multivariable associations of hypertensive disorders of pregnancy and offspring adiposity are presented in Table 4. The inverse association of preeclampsia with offspring lean mass at 9 years of age was essentially unaltered by adjustment for potential confounding factors and mediation by birth size and gestational age. With adjustment for potential confounding factors, the previously null associations of preeclampsia with all measures of offspring adiposity became inverse. The main covariable that resulted in this change was maternal prepregnancy BMI. Thus, offspring of women with preeclampsia are less adipose and less likely to be obese once the positive association of maternal BMI with preeclampsia risk and with offspring adiposity has been controlled for.

With adjustment for potential confounders, the previous positive association of gestational hypertension and offspring adiposity was attenuated to the null. Based on the greatest change in the coefficient, adjustment for parental BMI was the key covariable responsible for this attenuation; the adjusted association was essentially the same with adjustment only for maternal and paternal BMI as for adjustment for these and all other covariables.

Table 4. Multivariable Associations of Hypertensive Disorders of Pregnancy With Offspring Adiposity Measures at the 9-Year Follow-Up Clinic (n=3876 With Complete Data on Any Variable Included in Any Model)

<table>
<thead>
<tr>
<th>Measure at 9-year follow-up clinic</th>
<th>No Hypertensive Disorder of Pregnancy (n=5345)</th>
<th>Gestational Hypertension (n=1065)</th>
<th>Preeclampsia (n=196)</th>
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</tbody>
</table>

Values are regression coefficients or ORs (95% CIs) and reflect the difference in offspring characteristic for maternal hypertensive disorder of pregnancy (ie, in this table, hypertensive disorder of pregnancy is the exposure, and offspring characteristics are the outcomes). Model 1, adjusted for offspring sex and age at the 9-year visit. Model 2, additionally adjusted for maternal age at delivery, parental prepregnancy BMI, parity, social class, and maternal smoking during pregnancy, plus offspring weight, height, and height squared at the 9-year visit (the confounder-adjusted model). Model 3, additionally adjusted for mode of delivery, gestational age at birth, and birth weight (to examine mediation by these determinants).
Associations of hypertensive disorders of pregnancy with offspring BP are presented in Table 5. Both preeclampsia and gestational hypertension were associated with greater SBP and DBP in the 9-year-old offspring, with the magnitudes of these associations being similar for each hypertensive disorder of pregnancy in the confounder-adjusted model (model 2); on average, both preeclampsia and gestational hypertension were associated with a 2-mm Hg greater SBP and a 1-mm Hg greater DBP in 9-year-old offspring. With further adjustment for mode of delivery, birth weight, and gestational age (potential mediators), the association of preeclampsia with offspring BP attenuated toward the null, whereas that of gestational hypertension remained.

### Discussion

In our prospective birth cohort, offspring of women with a history of preeclampsia or gestational hypertension had increased SBP and DBP compared with women without hypertensive disorders of pregnancy. These associations did not appear to be explained by shared familial characteristics (genetic or behavioral) related to family adiposity because they remained after adjustment for maternal and paternal prepregnancy BMI and offspring adiposity. The association of preeclampsia with offspring BP appeared to be mediated at least in part by the association of preeclampsia with lower birth weight and preterm delivery. In contrast, gestational hypertension was not associated with offspring gestational age or birth weight; hence, they did not mediate its association with offspring BP. An additional interesting finding of our study was that of an inverse association of preeclampsia (but not gestational hypertension) with offspring lean mass in all multivariable models and an inverse association with measurements of adiposity once maternal BMI had been taken into account.

The major strengths of our study are its prospective design and large sample size. To the best of our knowledge, this is the first study to examine the associations of hypertensive disorders of pregnancy with detailed measures of offspring adiposity and measures of parental BMI and to compare differences in associations between preeclampsia and gestational hypertension. We were able to define preeclampsia and gestational hypertension by applying standard definitions to detailed clinical data (repeated assessments throughout pregnancy of BP and proteinuria) abstracted from the antenatal medical records rather than having to rely on retrospective maternal report or clinical diagnoses (made with unclear criteria) as in some previous studies. Although the majority (95%) of participants attending the follow-up clinic had adequate data on all variables included in any analyses, there was less to follow-up, with 65% of those eligible actually attending. Participants who attended were more likely to be from higher socioeconomic position families and less likely to have had teenage mothers and mothers who smoked during pregnancy. However, we found those attending the clinic had mothers with similar proportions of preeclampsia and gestational hypertension compared with those lost to follow-up, and we can think of no reasons why the associations we have examined here should be markedly different in those lost to follow-up. Parental BMI was based on self-report of weight and height. For mothers, there was strong correlation with measured weight at their first antenatal clinic visit; however, it is possible that measurement error in parental BMI may have reduced the ability to fully adjust for its confounding. As demonstrated in the Figure, the confounding pathway from parental adiposity is likely to act via the child’s adiposity. We had very good measurements for the child’s adiposity and therefore think this pathway is adequately controlled for in our study.

Our findings are in line with other studies that have reported positive associations between hypertensive disorders of pregnancy and offspring BP. Our study extends this previous work by suggesting that family adiposity does not confound these associations. It also suggests that intrauterine growth restriction might mediate in part the association of preeclampsia, but not gestational hypertension, with offspring BP. This requires further replication in other studies. However, the difference in the associations of preeclampsia (inverse association) and gestational hypertension (no association) with birth weight adds to evidence that these 2 conditions are distinct. Our findings suggest that the relationship between gestational hypertension and offspring BP may represent shared genetic or other shared familial mechanisms and as such point toward gestational hypertension being related to nonpregnancy hypertension. In contrast, preeclampsia appears to be linked to later offspring outcomes via intrauterine mechanisms.

We are not aware of previous studies showing a reduced lean and fat mass (after adjustment for maternal BMI) in offspring of mothers who experienced preeclampsia in their pregnancy. Several mechanisms could explain this association. Preeclampsia is associated with fetal growth restriction and preterm delivery resulting from placental hypoperfusion, and we confirmed a specific association of preeclampsia with lower birth weight and increased risk of preterm...
delivery, which was not seen for gestational hypertension. Because lower birth weight is associated with reduced lean and fat mass in later life, the association of preeclampsia with later offspring reduced fat and lean mass may be explained by its effect on intrauterine growth restriction. Another explanation could be maternal smoking during pregnancy. Smoking is associated with lower BMI and is protective against preeclampsia. However, several studies have shown a positive association between maternal smoking in pregnancy and greater offspring BMI in later life. Thus, when we control for maternal BMI, we may unmask a positive association of maternal smoking with offspring BMI and, because of the protective effect of smoking on preeclampsia, an inverse association between it and offspring BMI. Although these explanations are plausible, the inverse association was present even with adjustment for maternal smoking and was unaffected by adjustment for birth weight and gestational age. Further research is required to examine whether this finding is replicated in other independent cohorts and, if so, to explore likely underlying mechanisms.

Conclusions

Our findings suggest that women who experience hypertensive disorders of pregnancy have children with higher BP in childhood. These associations do not appear to be confounded by familial adiposity. Although interventions to reduce obesity might reduce the occurrence of hypertensive disorders of pregnancy, they are unlikely to reduce its link to higher offspring BP. The mechanisms underlying the associations of preeclampsia and gestational hypertension with offspring BP may differ from each other, with evidence that the relationship between preeclampsia and intrauterine growth restriction plays a role in its association but does not have any role in the association between gestational hypertension and offspring BP.

Acknowledgments

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Disclosures

None.

References


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**CLINICAL PERSPECTIVE**

Offspring of women with hypertensive disorders of pregnancy are at increased risk of cardiovascular complications later in life, but the mechanisms underlying these associations are unclear. Using data from 6343 mother-offspring pairs enrolled in a UK birth cohort, we examined differences in associations between preeclampsia and gestational hypertension with offspring blood pressure measured at 9 to 10 years of age and the role that birth weight and parental and offspring adiposity might have in explaining any associations. Both preeclampsia and gestational hypertension were associated with systolic and diastolic blood pressures in the offspring. These associations were similar in magnitude for the 2 exposures and were not explained by parental or own adiposity. After adjustment for parental and own adiposity and for other potential confounders, there was a mean difference in systolic blood pressure of 2.05 mm Hg (95% confidence interval, 0.72 to 3.38) and 2.04 mm Hg (95% confidence interval, 1.42 to 2.67) for preeclampsia and gestational hypertension, respectively, compared with those with no hypertensive disorders of pregnancy. Equivalent results for diastolic blood pressure were 1.00 mm Hg (95% confidence interval, −0.01 to 2.10) and 1.07 mm Hg (95% confidence interval, 0.60 to 1.54). The association of preeclampsia with offspring systolic and diastolic blood pressures attenuated to the null with further adjustment for birth weight and gestational age, whereas these adjustments did not attenuate the association of gestational hypertension with offspring blood pressure. Our findings suggest that the relationship between gestational hypertension and offspring blood pressure might reflect shared genetic or familial environmental characteristics (unrelated to familial adiposity) but that the relationship between preeclampsia and offspring blood pressure is driven largely by intrauterine characteristics.
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