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**ORIGINAL ARTICLE**

# Does a history of hypertensive disorders of pregnancy help predict future essential hypertension? Findings from a prospective pregnancy cohort study

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Hypertensive disorder of pregnancy (HDP) is considered an important determinant in the prediction of future hypertension. The aim of this study is to examine whether HDP improves prediction of future hypertension, over prediction based on established risk factors measured during pregnancy. We used a community based cohort study of 2117 women who received antenatal care at a major hospital in Brisbane between 1981 and 1983 and had blood pressure assessed 21 years after the index pregnancy. Of these 2117 women, 193 (9.0%) experienced HDP and 345 (16.3%) had hypertension at 21 years postpartum. For women with HDP, the odds of being hypertensive at 21 years postpartum were 2.46 (95% CI 1.70, 3.56), adjusted for established risk factors including age, education, race, alcohol, cigarettes, exercise and body mass index. Addition of HDP did not improve the prediction model that included these established risk factors, with the area under the curve of receiver operator (AUROC) increasing from 0.710 to 0.716 (*P*-value for difference in AUROC = 0.185). Our findings suggest that HDP is strongly and independently associated with future hypertension, and women who experience this condition should be counselled regarding lifestyle modification and careful ongoing blood pressure monitoring. However, the development of HDP during pregnancy does not improve our capacity to predict future hypertension, over risk factors identifiable at the time of pregnancy. This suggests that counseling regarding lifestyle modification and ongoing blood pressure monitoring might reasonably be provided to all pregnant and postpartum women with identifiable risk factors for future hypertension.

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## INTRODUCTION

From as early as the 1930s clinicians noticed that women who experienced hypertensive disorders of pregnancy (HDP) had a higher rate of future hypertension, diabetes, cardiac disease, renal disease and a higher death rate compared with the rest of the population.[1–5] More recently, it has been recognized that pregnancy provides an important 'metabolic stress test' and potentially allows identification of women at future risk of hypertension, cardiovascular disease (CVD) and metabolic disease.[6,7] Pregnancy potentially provides a unique window of opportunity to identify and target preventative strategies for women at higher risk of future CVD.

In this study, we wish to explore the concept of pregnancy as a 'hypertension stress test'. There is a range of known risk factors for the development of hypertension. These include increased body mass index (BMI), alcohol use, cigarette smoking, family history and socioeconomic status.[8–10] These risk factors are easily identifiable in any young woman, whether they have been pregnant or not, and risk prevention strategies could be targeted for women who are in higher-risk categories. However, it is important to understand how useful the 'hypertension stress test' of pregnancy is in identifying a high-risk group of women.

Understanding the value of pregnancy as a 'hypertension stress test' is important for a number of reasons. First, if the development of hypertension in pregnancy provides better capacity to predict future hypertension over and above easily obtained clinical and demographic information, then this adds to the argument that we should implement careful follow-up and hypertension prevention and treatment strategies for these women. Second, in current clinical practice, very few women undergoing a risk assessment for future hypertension or CVD are asked about their pregnancy history.[7] If HDP were shown to be strongly predictive of future hypertension even after taking other information into account, then there would be a strong argument for asking women about previous episodes of HDP at every clinical encounter. Alternatively, if clinical and demographic information easily obtainable from pregnant women is equally predictive of their risk of future hypertension, it may be more appropriate to counsel all young women with identifiable risk factors regarding lifestyle modification and blood pressure monitoring.

The objective of this paper is to examine whether the 'hypertension stress test' in pregnancy improves prediction of future hypertension, over and above other basic clinical and demographic characteristics easily observed or measured at the time of pregnancy.

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## METHODS

### Participants

The Mater-University of Queensland Study of Pregnancy (MUSP) is a prospective study of 7223 women, and their offspring, who received antenatal care at a major public hospital in South Brisbane between 1981 and 1983 and delivered a live singleton child who was not adopted before leaving hospital.[7] At the first clinic visit (FCV), on average women were in week 18 of gestation. Subsequently women and their children were followed up at 6-months, 5, 14 and 21 years postpartum.[11] The pregnancy studied as part of the MUSP is referred to throughout this paper as the index pregnancy. Women were eligible for this study, if we had information regarding the presence or absence of HDP in their index pregnancy, and further, we had information regarding their measured blood pressure at 21 years post delivery. Therefore, the prediction models presented here were developed in 2117 eligible women.

This study was approved by the human research ethics committees of the University of Queensland and Mater Hospital, and was conducted according to the Declaration of Helsinki. Written consent for the use of participant data was obtained from all participants. Full details of the study participants and measurements have been previously reported.[12–14]

### Measurement of outcomes

Blood pressure was assessed at the 21-year follow-up with two readings taken 5 min apart, using the OMRON HEM-703C automatic blood pressure device (Omron Healthcare, Lake Forest, IL, USA), with the woman seated and at rest, and an appropriate cuff size based on arm circumference, used. The average of the two readings was used in all analyses. At 21 years follow-up mothers were asked 'have you taken any medications in the last 24 h?' with response options 'yes', or 'no'. If they reported yes, the names of the drugs were listed. Women were classified as hypertensive if they met the following criteria: systolic blood pressure $>140$ or diastolic blood pressure $>90$, or currently taking blood pressure medication. For the prediction model, we used the binary outcome of hypertensive versus non-hypertensive.

### Definition of hypertensive disorders of pregnancy

The definition used for a HDP at the time of this study (early 1980s) differs from those in common use now. HDP (termed preeclampsia at the time) was defined as a diastolic blood pressure over 90 mm Hg on at least two occasions beyond 20 weeks gestation associated with proteinuria and/or excessive fluid retention (defined as generalized oedema including the face and hands and excessive weight gain). Proteinuria was diagnosed whether there was at least 2+ of protein on dipstick testing (Albustix, Bayer Inc, Pittsburgh, PA, USA). The diagnosis of preeclampsia was made and recorded by consultant obstetricians. According to the currently accepted definitions of the International Society for the Study of Hypertension in Pregnancy (which were introduced in 1986) these women (who were classified as having preeclampsia at the time) would now be classified as having gestational hypertension, preeclampsia or preeclampsia superimposed on chronic hypertension.[15] Therefore, we refer to these disorders together as HDP. Women with hypertension before pregnancy ($n=1$ of the 2127 eligible for our analyses), or who were found to have hypertension before 20 weeks gestation ($n=9$) were excluded from the analyses, leaving 2117 women for analysis.

### Established risk factors and other co-variates

We selected a series of clinical and demographic parameters, which are known to be associated with future hypertension and which could be relatively easily obtained from women. These included maternal age, ethnicity, education, alcohol intake, number of cigarettes smoked, exercise and BMI. We included these variables in the established risk factor prediction model for hypertension.

Maternal educational attainment (did not complete secondary school, completed secondary school and completed further/higher education) and ethnicity (White, Asian or Aboriginal/Islander) were all obtained from questionnaires completed at the FCV of the study. At the FCV, women were asked to select, from a 7-point scale, the (Australian) dollar figure range closest to their total annual family income. This scale was dichotomised as AUS $10 400 or more and $<$AUS $10 400.

At the FCV, women were assessed using the 7-item depression sub-scale from the Delusions Symptoms-States Inventory: State of Anxiety and Depression (DSSI/SAD).[16] The measure was developed to detect signs and symptoms of psychopathology that limit a person's capacity to function and to maintain relationships. This measure has high internal validity,[16,17] correlates well and shares items, with other measures of depression and anxiety such as the Edinburgh Postnatal Depression Scale and the Hospital Anxiety and Depression Scale.[18]

Self-reported tobacco (non-smoker, 1–20 cigarettes per day and 20 or more cigarettes per day) and alcohol (abstainer, light, 1+ serve per day) consumption was recorded during the last trimester of pregnancy. Women were asked whether they did physical exercises often, sometimes or never before the index pregnancy. Maternal pre-pregnancy BMI was calculated based on the maternal measured height in pregnancy and self-reported pre-pregnancy weight. At the first antenatal clinic visit, women were asked to report their prepregnancy weight; women were also weighed at the clinic. Self-reported prepregnancy weight and measured weight at the first antenatal visit were highly correlated (Pearson's correlation coefficient 0.95).

### Statistical analyses

We compared the FCV characteristics of the women who were included in the analyses with the excluded women. We used an *F*-test for a continuously distributed data and a $\chi^2$-test for categorical data to statistically test differences. The results of these analyses are presented in Table 1.

The unadjusted associations of established risk factors and HDP with hypertension at 21 years post pregnancy are presented in Table 2. We used $\chi^2$-test for categorical risk factors and *F*-test for continuously distributed risk factors to test for statistical associations.

We used a series of multiple logistic regression models to estimate the adjusted odds ratio (OR) of being hypertensive at 21 years post delivery by HDP (Table 3). In model 1, we adjusted only for maternal age. In the second model, we additionally adjusted for maternal education and ethnicity and in the third, we additionally adjusted for alcohol and cigarette use (pre-pregnancy) and exercise. In the final model (model 4), we further included maternal pre-pregnancy BMI. The area under the curve of receiver operator characteristics (AUROC) was used to test the incremental effect on discrimination of adding HDP to a prediction model including all other established risk factors (Table 3). Discrimination is the capacity of a prediction model to rank each individual in such a way that those who experience the event of interest (hypertension in this study) have a greater predicted risk than those who do not experience this.[19] We used a $\chi^2$-test (with one degree of freedom) to compare the AUROC curve from one model to the other. This analysis was performed on 1892 women with complete data available and additionally on those 1582 women with Caucasian ethnicity who delivered after 34 weeks gestation and whose infants had a birth weight above the tenth percentile. We also repeated the analysis, examining the results of the analysis if we looked only at women who had preterm birth or a baby less than the tenth percentile, in addition to the presence of HDP.

We calibrated the two prediction models (established risk factors and established risk factors with HDP) to measure their accuracy by comparing mean predicted risk with observed risk of the outcome (hypertension) in groups of individuals classified by level of risk. This calibration was performed by first ranking subjects into quantiles based on their predicted risk and then within each quantile comparing the predicted mean risk to the observed risk of hypertension (Figure 1).

All the analyses were undertaken using STATA 11 (STATA Corp., College Station, TX, USA).

## RESULTS

We found that women who were excluded from analyses were more likely to be younger, have had less education, have a lower income, be depressed, smoke and be of Asian or Aboriginal-Islander background (all $P<0.001$). Women who were excluded from analyses because of missing data did not differ markedly in terms of prevalence of HDP (8.8% of excluded versus 9.0% of included, $P=0.711$) (Table 1). Of the 2117 included women, 191 (9%) had HDP and 345 (16%) had hypertension at 21 years postpartum (Table 2).

The frequency of hypertension at 21 years postpartum classified according to the presence/absence of HDP and other established risk factors is presented in Table 2. Women with hypertension, compared with those without, were on average older and had higher BMI during pregnancy. They were also more likely to have lower educational attainment, be of Asian origin and were less likely to have smoked during pregnancy. For other risk factors

**Table 1.** Comparison of the first clinic visit characteristics of women included and not included in the analyses

| *Background characteristics at FCV* | N | *% included* (n = *2117*) | *% excluded* (n = *5106*) | P-*value** |
|---|---|---|---|---|
| *Mothers education* | | | | |
| Did not complete secondary | 1305 | 15.4 | 19.4 | |
| Completed secondary | 4609 | 64.8 | 64.1 | |
| Completed further or high | 1256 | 19.8 | 16.6 | <0.001 |
| *Income at FCV* | | | | |
| AUS $10 400 or more | 4441 | 71.7 | 63.3 | <0.001 |
| $10 400 or less | 2308 | 28.3 | 36.7 | |
| *Maternal depression at FCV* | | | | |
| Not depressed | 6673 | 96.9 | 93.0 | <0.001 |
| Depressed | 412 | 3.1 | 7.0 | |
| *Maternal smoking status at FCV* | | | | |
| None smoker | 4422 | 65.6 | 60.2 | |
| 1–9 cigarettes per day | 2117 | 27.3 | 30.5 | <0.001 |
| 10+ cigarettes per day | 617 | 7.1 | 9.3 | |
| *Racial origin* | | | | |
| White | 6259 | 92.9 | 87.8 | |
| Asian | 307 | 3.6 | 4.7 | <0.001 |
| Aboriginal-Islander | 444 | 3.5 | 7.5 | |
| *HDP* | | | | |
| No | 6546 | 91.0 | 91.2 | 0.711 |
| Yes | 634 | 9.0 | 8.8 | |
| *Physical exercise* | | | | |
| Never | 2302 | 30.3 | 33.3 | |
| Sometimes | 3673 | 54.4 | 50.6 | 0.013 |
| Often | 1125 | 15.3 | 16.1 | |
| *Parity* | | | | |
| None | 2937 | 39.6 | 41.1 | |
| 1 | 2218 | 32.3 | 30.1 | 0.166 |
| 2+ | 2062 | 28.1 | 28.8 | |
| Pre-pregnancy BMI, mean (s.d.) | 6692 | 21.69 (3.61) | 22.00 (4.16) | <0.004 |
| Age at FCV, mean (s.d.) | 7223 | 25.66 (4.96) | 24.78 (5.15) | <0.001 |

Abbreviations: BMI, body mass index; FCV, first clinic visit. **P* indicates the significance level of the difference of the characteristics between included and not included women. We used an *F*-test for a continuous data and a $\chi^2$-test for categorical data.

there was little evidence that distributions differed between women with or without hypertension.

Table 3 shows the multivariable association of HDP and hypertension at 21 years postpartum. The results are presented for the 1892 women with complete data available on all variables included in any of the multivariable models. In the basic age adjusted model, there was a threefold increase in the odds of future hypertension comparing women with HDP to those without. Further adjustment for education, ethnicity, smoking, alcohol and physical activity did not alter this association. Adjustment for BMI resulted in some attenuation but a positive association remained in the fully adjusted model including adjustment for BMI (OR 2.46 (95% CI: 1.70, 3.56)).

Table 4 shows the AUROC for three models (HDP only, established risk factors only and established risk factors plus HDP) and shows that HDP only is a poor discriminator of future risk of hypertension and that the addition of HDP to established risk factors does not markedly improve discrimination. Figure 1 shows predicted versus observed risk of hypertension using a model containing established risk factors only and one with both established risk factors and HDP by strata of predicted risk. Although both models accurately predict hypertension in women at both high and low risk, the addition HDP does not importantly improve the accuracy of a model that only includes the established risk factors.

We repeated the main analyses (Tables 3 and 4) including only Caucasian group ($N = 1761$), excluding women who delivered before 34 weeks ($n = 18$) and also those whose baby's birth weight was below the tenth percentile ($n = 190$). For this selected group of women ($N = 1582$), we found the OR 2.66 (95% CI: 1.77, 4.00) of being hypertensive at 21 years postpartum for model 1 and the OR 2.08 (1.36, 3.18) for the model 4 (fully adjusted model). These estimates are slightly (~10%) lower than those presented in Tables 3 and 4.

Of the 191 women with HDP, 35 women were delivered before 34 weeks or had a baby with a birth weight below the tenth percentile. This identified a group of women who most likely had very severe, early-onset preeclampsia. Fifteen of these women were hypertensive at the 21-year follow-up (42.9%). For this group of women, we found an OR 7.01 (95% CI: 2.78, 17.66) of being hypertensive at 21 years postpartum for model 1 and the OR 6.28 (95% CI: 2.15, 18.31) for model 4 (fully adjusted model).

We compared the AUROC discrimination between established risk factors and established risk factor plus HDP models for each sub-group separately. They were not statistically significant (*P*-value <0.650 for the sub-group $N = 1582$ and *P*-value <0.670 for the sub-group $N = 191$). This demonstrates that even in women with severe, early-onset disease, HDP is still not predictive of future hypertension, over and above the easily identifiable established clinical and demographic risk factors.

**Table 2.** Unadjusted associations of clinical and demographic risk factors and hypertensive disorders of pregnancy with hypertension at 21 years post delivery ($N = 2117$)

| *Clinical and demographic markers of future hypertension* | N | N *(%) of not hypertensive and hypertensive at 21 year post delivery* | | |
|---|---|---|---|---|
| | | *Not hypertensive* | *Hypertensive* | P-*value** |
| *Hypertensive disorder of pregnancy* | | | | |
| No | 1926 | 1644 (92.8) | 282 (81.7) | |
| Yes | 191 | 128 (7.2) | 63 (18.3) | <0.001 |
| *Maternal education* | | | | |
| Incomplete high school | 324 | 251 (14.3) | 73 (21.3) | |
| Complete high school | 1362 | 1155 (64.7) | 207 (60.4) | |
| Post high school | 415 | 352 (20.0) | 63 (18.4) | 0.005 |
| *Ethnicity* | | | | |
| Caucasian | 1914 | 1611 (93.4) | 303 (90.2) | |
| Asian | 75 | 56 (3.3) | 19 (5.7) | 0.07 |
| Aboriginal-Islanders | 72 | 58 (3.4) | 14 (4.2) | |
| *Maternal cigarettes smoking during pregnancy* | | | | |
| Nil | 1364 | 1133 (64.1) | 231 (67.0) | |
| 1–19 cigarettes per day | 523 | 443 (25.0) | 80 (23.2) | 0.58 |
| 20 or more cigarettes per day | 227 | 193 (10.9) | 34 (9.9) | |
| *Alcohol consumption during pregnancy* | | | | |
| Abstainer | 989 | 825 (46.8) | 164 (48.4) | |
| Light | 1090 | 922 (52.3) | 168 (49.6) | |
| 1 + glass per day | 24 | 17 (1.0) | 7 (2.1) | 0.17 |
| *Exercise during pregnancy* | | | | |
| Never | 632 | 525 (15.6) | 108 (13.5) | |
| Sometime | 1134 | 946 (54.3) | 188 (55.1) | 0.60 |
| Often | 318 | 272 (30.1) | 46 (31.4) | |
| Maternal age at first clinic visit, mean (s.d.) | 2117 | 25.26 (4.9) | 27.69 (5.0) | 0.001 |
| Maternal pre-pregnancy BMI, mean (s.d.) | 2001 | 21.32 (3.2) | 23.70 (4.7) | 0.001 |

Abbreviation: BMI, body mass index. **P*-value indicates the significance level of the difference between hypertension and not hypertension group. We used $\chi^2$-test for categorical risk factors and *F*-test for continuous risk factors to test for statistical associations.

**Table 3.** Multivariable association of hypertensive disorder of pregnancy with maternal hypertension after 21 years of post delivery ($N = 1892$)

| *Model* | *Odds ratio of hypertension at 21 years post delivery (95% CI)* | P-*value* |
|---|---|---|
| Model 1: age adjusted | 3.07 (2.16, 4.38) | <0.001 |
| Model 2: adjusted age, education and race | 3.09 (2.16, 4.40) | <0.001 |
| Model 3: As model 2 plus alcohol, cigarettes and exercise | 2.99 (2.09, 4.28) | <0.001 |
| Model 4: As model 3 plus body mass index | 2.46 (1.70, 3.56) | <0.001 |

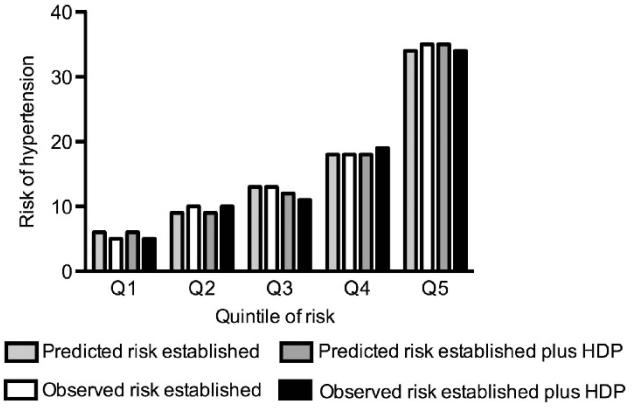


**Figure 1.** Predicted and observed risk of hypertension based on logistic regression models using traditional risk factors alone and with addition of hypertensive disorder of pregnancy ($N = 1908$).

## DISCUSSION

Given that hypertension is a key contributor to CVD, and that CVD remains a leading cause of death in women, it is important to understand which factors predict hypertension in women. Our study confirmed the previously established independent association of HDP with future hypertension.[20–23] To our knowledge, this is the first study to show that despite this strong independent association, HDP does not importantly improve on the ability to predict future hypertension, over and above simple clinical and demographic risk factors known at the time of pregnancy.

A number of authors have argued that a history of HDP should be used to improve risk prediction of future hypertension and CVD risk in women.[7] However, our results suggest that this is unlikely to be the case if all risk factors around the time of pregnancy are known. Future studies are required to assess whether HDP can improve prediction models for CVD in women. Does the lack of predictive power simply result from HDP being a low prevalence condition? Overall 10% of the sample had HDP, which is not an especially low prevalence. Only one-third of those with HDP have

**Table 4.** AUROC comparing discrimination between clinical and demographic risk factors and clinical and demographic risk factor plus hypertensive disorder of pregnancy models ($N = 1892$)

| *Model description* | *AUROC (95% CI)* | P-*value for difference in AUROC* |
|---|---|---|
| Hypertensive disorder of pregnancy | 0.560 (0.0536, 0.583) | $<0.001$ |
| Clinical and demographic hypertension risk factors | 0.710 (0.676, 0.740) | referent |
| Clinical and demographic hypertension risk factors plus hypertensive disorder of pregnancy | 0.716 (0.684, 0.749) | 0.185 |

Abbreviation: AUROC, area under the curve of receiver operator.

hypertension 21 years later; two-third do not. We showed that simple clinical and demographic risk factors known at the time of pregnancy are predictive of future hypertension, and this predictive capacity is not improved by adding information about HDP. If our findings are replicated, the implication is that a much broader approach needs to be taken to the prevention of hypertension in women. Women at risk can be identified during their pregnancy, based on simple information. Ideally, these women would be identified, and provided with information about regular blood pressure measurement and lifestyle modification. Thus, our results suggest that HDP may not be useful as part of a population screening tool for identifying, at the time of pregnancy, those at risk of hypertension in mid-life. The strongest risk factor and the one that drives overall prediction is pre-pregnancy BMI. This is highlighted by our finding that HDP does not add anything to the strength of pre-pregnancy BMI to predict hypertension. Public health measures to reduce hypertension in women need to strongly consider the issue of overweight and obesity in women of child-bearing age.

However, our findings regarding prediction of hypertension in a population do not apply to the individual. Individual women who suffer HDP should be provided with follow-up, lifestyle advice or treatment as appropriate with respect to their post pregnancy blood pressure. In fact, it has been reported that the proportion of women diagnosed with preeclampsia that were not screened for unresolved preeclampsia at their 6-week postnatal follow-up was significant, which is disconcerting.[24] Further, we have previously published that women with a known history of HDP have unacceptable rates of undiagnosed or inadequately treated hypertension.[25] To assess whether aggressive follow-up and management of women with HDP can prevent CVD will require a randomized controlled trial.

Does our study suggest that clinicians need not bother to take a careful pregnancy history? We have demonstrated that the 'hypertension stress test' of pregnancy does not help to predict who will get hypertension any better than knowing the woman's pre pregnancy BMI, ethnicity, smoking status, alcohol use, educational status and physical activity profile. However, we cannot make any assessment of whether HDP improves the capacity to predict CVD, over and above the assessment of other known risk factors. This would be an important area of future study. Further, a detailed pregnancy history might reveal a range of issues that could be relevant to the general care of the woman, including a pregnancy history of renal dysfunction, thyroid disorders, gestational diabetes, gestational thrombocytopenia or gestational liver function abnormalities. Pregnancy remains a general 'metabolic stress test',[6,7] and wise clinicians will avail themselves of this information.

This study has a number of strengths. It is a large longitudinal cohort of women, who have been followed up for 21 years since the index pregnancy. There is only one study in press[23] where a large number of women with and without HDP have had other risk factors of hypertension including age, smoking and alcohol consumption, physical activity and BMI at pregnancy; others have reported associations on more limited data sets.[26–29] However, our findings regarding physical activity should be interpreted with caution, as we did not have an objective measure of physical activity. Further, we do not have information about a range of well-known risk factors for hypertension, including family history, salt intake, dyslipidemia, vitamin-D deficiency or detailed information regarding lifetime history of psychological stressors. We would expect that if we had this detailed information regarding other risk factors for future hypertension at the time of pregnancy, then the contribution of HDP to the predictive model may be further diminished.

Important other limitations of this study are our inability to examine whether different classes of HDP (in particular more severe forms of preeclampsia) are better predictors of future hypertensive risk than less severe forms. However, when using preterm births before 34 weeks gestation and infants with a birth weight smaller than the tenth percentile as proxies for disease severity, our results were similar, indicating that our general conclusion is valid. A recent follow-up of mothers in the Avon Longitudinal Study of Parents and Children found very similar magnitudes of association between preeclampsia and gestational hypertension with blood pressure assessed $\sim$18 years post-pregnancy,[23] and therefore this inability to separate these two is unlikely to have affected our results. Our inability to examine whether HDP predicts future CVD events such as acute myocardial infarction or stroke over and above established risk factors is another limitation, which is partly due to the relatively low age of the participants. It could be argued that CVD prediction is more important than the prediction of hypertension and it is possible that findings with CVD events would differ from those with hypertension as HDP is associated with other CVD risk factors, including diabetes and dyslipidaemia.

It would be valuable to examine whether more severe forms of HDP are better predictors of future hypertension risk in cohorts, which have more detailed medical records, or in more contemporary cohorts. We replicated our analyses in a subset of women with likely severe, early-onset HDP and did not find that we could improve predictive capacity for future hypertension. We recognize that a weakness of this study is that, as with all longitudinal studies commenced in the early 1980s, the definitions of HDP have changed since this study was commenced. As discussed in detail in the Methods section, it is possible for women with gestational hypertension and severe oedema to have been coded by the original investigators as having 'preeclampsia'. Only one woman in our study was excluded on the basis of pre-existing hypertension, and nine were excluded because they were noted to be hypertensive before 20 weeks gestation. Therefore, 0.5% of women were excluded because of pre-existing hypertension. It is possible that some women had undiagnosed pre-existing hypertension. However, we think this is unlikely since at this age (mean age 26 years at the time of the index pregnancy and relatively low BMI in the early 1980s) this rate of pre-existing hypertension is reasonable.[30]

The loss to follow-up in our cohort was considerable. Those lost to follow-up were more likely to be teenagers at their delivery, to be less educated, single or cohabitating, have three or more children, use tobacco and alcohol during pregnancy, have a

higher BMI and to be anxious and depressed at their first antenatal visit.[12–14] Attrition is associated with disadvantage, and in the Australian context all of these factors are known to be associated with socioeconomic disadvantage. We would expect the hypertension rates to be higher in those who were not followed up. The rates of HDP in women who were and were not followed up were similar. Our results would be importantly biased if the associations assessed were either non-existent or in the opposite direction in non-participants. Although we cannot exclude this possibility, this would be unlikely.

In conclusion, we have confirmed the strong association of HDP with hypertension two decades postpartum. This finding may be useful in designing appropriate postpartum follow-up protocols for women affected by HDP. However, despite this strong and independent association, HDP does not importantly improve on the ability of clinical and demographic risk factors to predict future hypertension. Further research is required to examine whether more severe forms of HDP improve prediction of future CVD events.

*What is known about this topic*

- HDP is associated with increased risk of future hypertension.
- It is not clear whether including a history of HDP to other established risk factors measured during pregnancy improves the prediction of future hypertension.

*What this study adds*

- HDP is strongly and independently associated with future hypertension 21 years after the index pregnancy in a community-based cohort.
- Adding a history of HDP to established risk factors measured during pregnancy for example, age, education, race, alcohol, smoking, exercise and BMI does not improve the predictive model.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

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## DISCLAIMER

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