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Increased planned delivery contributes to declining rates of pregnancy hypertension in Australia: a population-based record linkage study

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ABSTRACT

Objective: Since the 1990s, pregnancy hypertension rates have declined in some countries, but not all. Increasing rates of early planned delivery (before the due date) have been hypothesised as the reason for the decline. The aim of this study was to explore whether early planned delivery can partly explain the declining pregnancy hypertension rates in Australia.

Design: Population-based record linkage study utilising linked birth and hospital records.

Setting and participants: A cohort of 1 076 122 deliveries in New South Wales, Australia, 2001–2012.

Outcome measures: Pregnancy hypertension (including gestational hypertension, pre-eclampsia and eclampsia) was the main outcome; pre-eclampsia was a secondary outcome.

Results: From 2001 to 2012, pregnancy hypertension rates declined by 22%, from 9.9% to 7.7%, and pre-eclampsia by 27%, from 3.3% to 2.4% (trend p<0.0001). At the same time, planned deliveries increased: prelabour caesarean section by 43% (12.9–18.4%) and labour inductions by 10% (24.8–27.2%). Many maternal risk factors for pregnancy hypertension significantly increased (p<0.01) over the study period including nulliparity, age ≥35 years, diabetes, overweight and obesity, and use of assisted reproductive technologies; some risk factors decreased including multifetal pregnancies, age <20 years, autoimmune diseases and previous pregnancy hypertension. Given these changes in risk factors, the pregnancy hypertension rate was predicted to increase to 10.5%. Examination of annual gestational age distributions showed that pregnancy hypertension rates actually declined from 38 weeks gestation and were steepest from 41 weeks; at least 36% of the decrease could be attributed to planned deliveries. The risk factors for pregnancy hypertension were also risk factors for planned delivery.

Conclusions: It appears that an unanticipated consequence of increasing early planned deliveries is a decline in the incidence of pregnancy hypertension. Women with risk factors for hypertension were relatively more likely to be selected for early delivery.

Strengths and limitations of this study

- A strength of the study is the use of longitudinally linked, population-based data over a recent 12-year period.
- Record linkage improves the ascertainment of pregnancy hypertension and covariates, which are known to be reliably reported.
- Population-level data lack detailed clinical information including medications that reduce the risk of hypertensive diseases (eg, aspirin, calcium supplementation, antihypertensives) and, importantly, the detailed reasons for obstetric interventions.
- Pregnancy hypertension can be an indication for planned delivery but early planned delivery may also mean pregnancy hypertension is avoided—and these relationships can be hard to untangle in routinely collected data.

INTRODUCTION

Hypertensive disorders of pregnancy include both pregnancy-induced and pre-existing conditions.[1] For those that are pregnancy-induced, the cause remains elusive. However, a number of factors are known to be associated with increased and decreased risk of new onset of hypertension during pregnancy. Nulliparity, extremes of maternal age, multiple births, diabetes, chronic hypertension, obesity, a hypertensive disorder in a previous pregnancy, renal disease, the presence of antiphospholipid antibodies, prolonged interpregnancy interval, assisted reproductive technologies, family history of pre-eclampsia and a new partner are all associated with increased risk of a woman having a hypertensive disorder.[2–11] Factors associated with a reduced risk include smoking, Asian ethnicity, summer births, aspirin and calcium supplementation in high-risk women, treatment of gestational diabetes and use of antihypertensive medications.[3 6 10–21] At a population level, changes in the frequency of these risk

factors would be expected to influence the overall population rate. Some factors are known to be increasing in many populations (eg, nulliparity, older maternal age, multiple births, diabetes, obesity and assisted reproductive technologies), while smoking among pregnant women is declining in high-income countries.

Since the 1990s, pregnancy hypertension (including gestational hypertension, pre-eclampsia and eclampsia) has declined in Australia, Scotland, Sweden and New York (USA), but not in Alberta (Canada), Denmark or Norway, although pre-eclampsia declined in Norway and Denmark.[5 22 23] In contrast, both pregnancy hypertension and pre-eclampsia increased in the USA.[23–25] As most (~75%) pregnancy hypertension occurs at term,[11] increasing rates of planned delivery before the due date means some women may give birth before they develop pregnancy hypertension. A marked downward shift in gestational age towards births at earlier term gestations since the 1990s has been attributed to increasing rates of planned births with fewer births starting spontaneously.[26–30] This was hypothesised as an explanation for the declining rates of pregnancy hypertension in countries that reported declines.[23] Temporal changes in the management of women with established pregnancy hypertension would not affect the reported incidence rates of de novo hypertension.

Therefore, the aim of this study was to explore whether early planned delivery can partly explain the declining pregnancy hypertension rates in Australia. Planned early delivery may be non-selective with respect to hypertension risk (eg, elective repeat caesarean section), or selective (women with prior pregnancy hypertension, obesity, advanced maternal age or other risk factors for hypertension may be relatively more likely to be scheduled for early delivery). If planned early delivery is selective, there should be a marked drop in the rates of pregnancy hypertension at ≥40 weeks, as women most at risk are delivered early while lower risk women are allowed to continue. Alternatively, if the decline in the pregnancy hypertension rate is unrelated to the timing of birth, then any decline in gestation-specific rates should be uniform.

METHODS

Study population and data sources

The study population was derived from all women who gave birth in New South Wales (NSW), Australia, 2001–2012. NSW is the most populous state of Australia (~7 million people), and almost one-third of all Australian births occur in NSW. Australia has a national health system, and maternity care in public hospitals is available free of charge to all women. However, about one-third of women choose private maternity care (through health insurance or payment).

Data for the study were obtained from two linked population health data sets: the NSW Perinatal Data Collection (PDC, referred to as birth records) and the NSW Admitted Patient Data Collection (APDC, referred to as hospital records). The PDC is a statutory surveillance system of all births in NSW of at least 20 weeks gestation or at least 400 g birth weight. Information on maternal characteristics, pregnancy, labour, delivery and infant outcomes are recorded by the attending midwife or doctor. The APDC is a census of all NSW inpatient hospital discharges from both public and private hospitals, and day procedure units, and includes demographic and episode-related data; diagnoses and procedures are coded for each admission from the medical records according to the 10th revision of the International Statistical Classification of Diseases and Related Health Problems, Australian Modification (ICD-10-AM) and the affiliated Australian Classification of Health Interventions.[31]

Longitudinal linkage of hospital records (from July 2000 to December 2012) and of birth records starting in 1994 was available. As Australia does not have a unique registration number for citizens, the separate data sets were linked using probabilistic linkage and a best practice approach to preserving privacy.[32 33] This involves a process of blocking and matching combinations of selected variables such as name, date of birth, address and hospital, and assigning a probability weight to the match.[34] Record linkage was undertaken by the NSW Centre for Health Record Linkage (CHeReL).[32] The validity of probabilistic record linkage is high;[35] and the linkage proportion for maternal hospital and birth records is 98.1%.[34] The researchers were provided with anonymised data.

Outcomes

The *primary outcome* was pregnancy hypertension at the time of delivery, which was obtained if recorded in either the birth record (check box) or a hospital record (physician-recorded diagnosis coded according to the ICD-10-AM) at any time during pregnancy.[36–38] Identification of pregnancy hypertension from routinely collected data in this manner (using more than one data source) has been demonstrated to be accurate and reliable when compared with clinician diagnoses in the medical records (eg, sensitivity 82%, positive predictive value (PPV) 92% in NSW).[38] Pre-eclampsia was a *secondary outcome* because reporting in population data is less accurate (eg, sensitivity 71%, PPV 67% in NSW) due to misclassification between gestational hypertension and pre-eclampsia.[38–40]

During the study period, gestational hypertension was defined as de novo onset of hypertension (systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg) from 20 weeks' gestation onwards and pre-eclampsia as the de novo onset of hypertension from 20 weeks' gestation onwards with one or more of proteinuria, renal insufficiency, liver involvement, neurological complications, haematological complications or fetal growth restriction.[1 41]

Explanatory factors

The factor of primary interest was planned birth by either induction of labour or prelabour caesarean section, especially planned births at 37–39 weeks. Hypertension can be identified as the reason for labour induction (1 of 10 tick boxes), but the reasons for caesarean section are limited to failure to progress, fetal distress and 'other' indications.

Information on established risk factors for pregnancy hypertension and pre-eclampsia that was available for analysis included: maternal age at delivery (<20, 20–34, ≥35 years), nulliparity, Asian ethnicity (based on country of birth), multifetal pregnancy, smoking during pregnancy, summer birth and interpregnancy interval ≥5 years (with lookback to 1994). Information on maternal medical conditions and reproductive history was obtained (as reported vs not reported) from birth records starting in 1994, and non-pregnancy and pregnancy-related hospital admissions starting in 2000, including chronic hypertension, diabetes (pregestational or gestational), morbid obesity, previous pregnancy hypertension, renal disease, autoimmune diseases (including rheumatoid arthritis, systemic lupus erythaematosus and other rare autoimmune diseases) and use of assisted reproductive technology (ART). The ascertainment of chronic medical conditions was maximised by using more than one data source (birth and hospital records, and by longitudinal linkage or pre-pregnancy, antenatal and birth records).[36–38 42] We also obtained annual area level overweight/obesity prevalence rates (body mass index ≥25 kg/m^2) for women by age (15–24, 25–34, ≥35 years) from the NSW Adult Population Health Survey.[43] Few records were missing data on these factors (<0.4%) with the exception of interpregnancy interval (4.3%). The explanatory factors are reported with a high level of accuracy.[36 38 44–47]

Statistical analysis

Descriptive statistics were used to summarise the distribution and trends in pregnancy hypertension and pre-eclampsia, and maternal risk factors and pregnancy characteristics. We determined the trends in pregnancy hypertension rates among women with established risk factors. To assess the role of maternal risk factors in selection for early delivery, we calculated the relative risk (RR) and 95% CI for early planned delivery by each maternal risk factor among women without pregnancy hypertension. Trends were assessed using the χ^2 for trend statistic and the p value was set at <0.01 because of the large number of both statistical tests and records.

To determine whether changes in maternal risk factors in the population could account for the observed pregnancy hypertension trend, we used predictive modelling to project the expected trend in pregnancy hypertension. Using methods previously described[48] and all births in 2001–2002, two multivariable logistic regression predictive models for pregnancy hypertension were developed. For the first model, all available risk factors for pregnancy hypertension (listed in table 1) were included to obtain a predictive equation.[2–21] Data from subsequent years were applied to this regression equation to account for the actual changes in risk factors over time and to produce a predicted trend for 2001–2012. If the predicted rate was similar to those observed, this would suggest the results are consistent with a theory that the decline in pregnancy hypertension rates is explained by changes in the demographic and obstetric history risk factors. The second model additionally included an indicator of planned birth other than for hypertension, and a predicted trend was obtained in the same way. Any difference in the two predicted trends would suggest that planned births explained some of the predicted increase. Deliveries with missing data on one or more risk factor were excluded from this analysis.

To specifically assess the impact of changes over time in gestational age at birth, annual gestational age distributions were determined (proportion of all deliveries occurring in a specific gestational week per total deliveries that took place that year) and plotted. These distributions are presented separately for women with and without pregnancy hypertension, and by labour onset (spontaneous, labour inductions and prelabour caesarean section). We also plotted gestation-specific trends in pregnancy hypertension using a pregnancy-at-risk approach (ie, the denominator was women still pregnant at the beginning of each gestational week).[49]

Finally, a priori, all analyses were repeated for nulliparous women with singleton pregnancies, but as the patterns for the most part were the same as for all women, these results are not presented.

RESULTS

From 2001 through 2012, there were 1 076 122 deliveries in NSW. The observed rates of pregnancy hypertension at the time of delivery declined by 22%, from 9.9% to 7.7%. Pre-eclampsia declined by 27%, from 3.3% in 2001, to 2.4% in 2012. Similar declines were observed among nulliparous women with singleton pregnancies, 13.5–10.1% and 5.0–3.5%, respectively. All trend p values were <0.0001. However, there was no significant trend in early onset (<34 weeks) pregnancy hypertension (mean 0.4%, trend p=0.9) or pre-eclampsia (mean 0.3%, trend p=0.5).

During the study period, changes in the maternal risk factors for pregnancy hypertension were mostly in a direction that would likely increase the rate of pregnancy hypertension including increases in nulliparous women, maternal age ≥35 years, maternal diabetes, overweight and obesity, use of ART; and a decline in smoking during pregnancy (table 1).

In contrast, changes in factors that would be consistent with a decrease in pregnancy hypertension rates included a significant decline in multifetal pregnancies, young maternal age, pregnancies complicated by maternal autoimmune diseases, previous pregnancy

Table 1 Trends in maternal hypertension risk factors, obstetric factors and sociodemographic characteristics, NSW 2001–2012

Risk factor	Risk factor prevalence 2001 N=84 302 n (%)	2012 N=96 051 n (%)	Trend over time p Value
Established risk factors for pregnancy hypertension			
Nulliparity	35 134 (41.7)	42 189 (43.9)	<0.0001
Maternal age ≥35 years	15 222 (18.1)	22 556 (23.5)	<0.0001
Maternal age <20 years	3797 (4.5)	3158 (3.3)	<0.0001
Diabetes (any)	4000 (4.7)	8812 (9.2)	<0.0001
Morbid obesity	125 (0.2)	809 (0.8)	<0.0001
NSW overweight/obesity rate	28.4%	33.2%	
Overweight	17.6%	19.7%	Not reported
Obese	10.8%	13.5%	
ART	1323 (1.6)	2971 (3.1)	<0.0001
Smoking	15 629 (18.5)	11 046 (11.5)	<0.0001
Multiple births	1452 (1.7)	1316 (1.4)	<0.0001
Autoimmune diseases	387 (0.5)	404 (0.4)	<0.0001
Previous pregnancy hypertension*	3932 (8.0)†	3929 (7.3)	<0.0001
Asian ethnicity	7924 (9.4)	17 647 (18.4)	<0.0001
Chronic hypertension	548 (0.7)	622 (0.7)	0.12
Renal disease	186 (0.2)	278 (0.3)	0.24
Interpregnancy interval ≥5 years*	7156 (15.9)†	7461 (15.6)	0.62
Summer birth	20 143 (23.9)	23 193 (24.2)	0.27
Obstetric factors			
Gestational age, weeks			
<37	5457 (6.5)	6649 (6.9)	<0.0001
37	4215 (5.0)	6544 (6.8)	<0.0001
38	12 491 (14.8)	17 654 (18.4)	<0.0001
39	18 993 (22.5)	28 395 (29.6)	<0.0001
40	26 780 (31.8)	24 183 (25.2)	<0.0001
≥41	16 354 (19.4)	12 622 (13.1)	<0.0001
Planned deliveries (any)	31 823 (37.8)	43 846 (45.7)	<0.0001
Prelabour CS	10 910 (12.9)	17 705 (18.4)	<0.0001
Labour inductions	20 913 (24.8)	26 141 (27.2)	<0.0001
Any planned delivery at 37–39 weeks	15 007 (17.8)	26 058 (27.1)	<0.0001
Sociodemographic factors			
Socioeconomic status			
Lowest (quintile)	16 903 (20.1)	18 911 (19.7)	0.15
Middle (quintiles 2–4)	51 277 (60.8)	57 231 (59.6)	<0.0001
Highest (quintile)	16 007 (19.0)	19 126 (19.9)	<0.0001
Maternal residence (urban vs rural)	57 537 (68.3)	68 290 (71.5)	<0.0001
Maternity hospital type			
Tertiary obstetric	36 415 (43.2)	47 020 (49.0)	<0.0001
Urban district	19 333 (22.9)	18 824 (19.6)	<0.0001
Rural district	9237 (11.0)	8412 (8.8)	<0.0001
Private	19 317 (22.9)	21 795 (22.7)	<0.0001

Per cents may not add to 100% because of missing data: 1579 (0.01%) parity, 298 (0.03%), 520 (0.05%) smoking, 4232 (0.39%) country of birth, 45 765 (4.3%) interpregnancy interval, 159 (0.01%) gestational age, 178 (0.02%) planned births, 4163 (0.39%) socioeconomic status, 2740 (0.25%) maternal residence and 20 (0.001%) hospital type.
*Among multipara.
†Reported rate is for 2003 (prior pregnancy hypertension) and 2006 (interpregnancy interval) to allow a sufficient lookback period for previous pregnancies.
ART, assisted reproductive technology; CS, caesarean section; NSW, New South Wales.

hypertension among multiparous women and an increase in Asian-born women. There was no significant change in the prevalence of chronic hypertension, renal disease, prolonged pregnancy interval or summer births.

Planned deliveries increased overall by 21%, from 37.8% of all deliveries in 2001, to 45.7% in 2012, and at 37–39 weeks by 52%, from 17.8% to 27.1% (table 1). Increases occurred in labour inductions as well as in prelabour caesarean sections. Among the labour inductions, the proportion where hypertension was recorded as the reason for induction declined from 13.6% to 9.4%. Among women undergoing a prelabour caesarean

section, the incidence of pregnancy hypertension also declined from 13.7% to 10.3%.

Among women with risk factors for pregnancy hypertension, the rate of pregnancy hypertension declined significantly over time (p for trend <0.001), with the exception of chronic hypertension, maternal age <20 years and preterm births (table 2). The relative decrease (rate ratio) in pregnancy hypertension rates was greatest among morbidly obese women and least (but still statistically significant) among smokers. Compared with the overall decline (by 22%, rate ratio 0.78), the decline in pregnancy hypertension was greater among planned births at 37–39 weeks (by 36%, rate ratio 0.64) and for all gestations from 38 weeks (by ≥28.0%, rate ratios ≤0.72).

Among women who never developed pregnancy hypertension, many of the established risk factors for hypertension were also positively associated with planned births at 37–39 weeks, including maternal ≥35 years (RR=1.58, 95% CI 1.56 to 1.59), multiple pregnancy (RR=1.76, 95% CI 1.71 to 1.81), diabetes (RR=2.07, 95% CI 2.04 to 2.10), chronic hypertension (RR=1.95, 95% CI 1.87 to 2.03), morbid obesity (RR=1.94, 95% CI 1.83 to 2.06), renal disease (RR=1.21, 95% CI 1.12 to 1.32), autoimmune diseases (RR=1.81, 95% CI 1.72 to 1.91), use of ART (RR=1.63, 95% CI 1.60 to 1.67) and pregnancy hypertension in a previous pregnancy (RR=1.62, 95% CI 1.59 to 1.65). Nulliparity, young maternal age, smoking and Asian ethnicity were negatively associated with planned birth among women without pregnancy hypertension.

Based on changes in the risk profile of the maternity population, the pregnancy hypertension rate was predicted to increase to 10.5% (figure 1). When planned birth was included in the predictive model, the pregnancy hypertension trend was forecast to decrease to 9.5%. The difference in the two predicted rates (1.0%) suggests planned births explained at least 36% of difference between the observed and predicted rates (based on maternal risk factors alone).

From 2001 through 2012, there was a marked difference in the distribution of gestational age for women with and without pregnancy hypertension at delivery (figure 2). For women *without* pregnancy hypertension,

Table 2 Trend in pregnancy hypertension rates among women with maternal and obstetric risk factors, 2001 and 2012

	Pregnancy hypertension rates				
Risk factor	2001 N=84 302 n (row %)	2012 N=96 051 n (row %)	Trend over time p Value	Rate difference*	Rate ratio (95% CI)*
All women	8356 (9.9)	7433 (7.7)	<0.0001	−2.2	0.78 (0.76 to 0.80)
Maternal risk factors					
Nulliparity	4827 (13.7)	4362 (10.3)	<0.0001	−3.4	0.75 (0.72 to 0.78)
Maternal age ≥35 years	1524 (10.0)	1907 (8.5)	<0.0001	−1.6	0.84 (0.79 to 0.90)
Maternal age <20 years	394 (10.4)	304 (9.6)	0.18	−0.8	0.93 (0.80 to 1.07)
Diabetes (any)	623 (15.6)	1058 (12.0)	<0.0001	−3.6	0.77 (0.70 to 0.84)
Morbid obesity	56 (45.2)	196 (24.2)	<0.0001	−20.9	0.54 (0.43 to 0.68)
ART	200 (15.1)	283 (9.5)	<0.0001	−5.6	0.63 (0.53 to 0.75)
Smoking	1183 (7.6)	763 (6.9)	<0.0001	−0.7	0.91 (0.83 to 0.99)
Multiple births	292 (20.1)	226 (17.2)	0.0013	−2.9	0.85 (0.73 to 1.00)
Autoimmune diseases	67 (16.5)	53 (13.1)	0.007	−3.4	0.79 (0.57 to 1.11)
Previous pregnancy hypertension†	1144 (36.3)	1249 (31.8)	0.0001	−4.5	0.88 (0.82 to 0.94)
Asian ethnicity	558 (7.0)	935 (5.3)	<0.0001	−1.6	0.75 (0.68 to 0.83)
Chronic hypertension	132 (24.1)	160 (25.7)	0.48	1.6	1.07 (0.87 to 1.30)
Renal disease	49 (26.5)	52 (18.7)	0.009	−7.8	0.71 (0.50 to 1.00)
Summer birth	1800 (8.9)	1722 (7.4)	<0.0001	−1.5	0.83 (0.78 to 0.89)
Obstetric factors					
Gestational age, weeks					
<37	1005 (18.4)	1203 (18.1)	0.35	−0.3	0.98 (0.89 to 1.07)
37	716 (17.0)	929 (14.2)	<0.0001	−2.8	0.84 (0.76 to 0.91)
38	1562 (12.5)	1524 (8.6)	<0.0001	−3.9	0.69 (0.65 to 0.74)
39	1805 (9.5)	1788 (6.3)	<0.0001	−3.2	0.66 (0.62 to 0.71)
40	2218 (8.3)	1404 (5.8)	<0.0001	−2.5	0.70 (0.66 to 0.75)
≥41	1047 (6.4)	584 (4.6)	<0.0001	−1.8	0.72 (0.65 to 0.80)
Planned deliveries (any)	5719 (18.0)	5604 (12.8)	<0.0001	−5.2	0.71 (0.69 to 0.74)
Prelabour CS	1484 (13.6)	1819 (10.3)	<0.0001	−3.3	0.76 (0.71 to 0.81)
Labour inductions	4235 (20.3)	3785 (14.5)	<0.0001	−5.8	0.72 (0.69 to 0.74)
Planned delivery at 37–39 weeks	2932 (19.5)	3281 (12.6)	<0.0001	−7.0	0.64 (0.62 to 0.67)

*Among women with the specified risk factor in 2012 compared with 2001.
†Among multipara.
ART, assisted reproductive technology; CS, caesarean section.

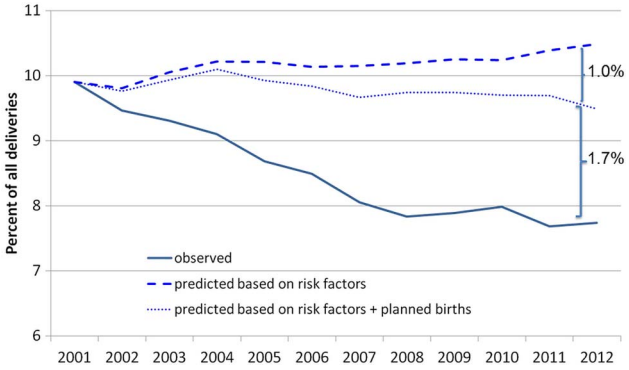

Figure 1 Observed and predicted trends in pregnancy hypertension, New South Wales 2001–2012.

the gestational age distribution curves shift to the left from 2001 through 2012, with a substantial increase in the percentage of births occurring at 39 weeks (figure 2A). In contrast, for women *with* pregnancy hypertension (figure 2B), there was a decline in the percentage of births from 38 weeks onwards but most notably ≥40 weeks. Births at these latter gestations disappear from the distribution in the later years of the study period, thereby changing the shape of the distribution. This is also demonstrated by the statistically significant declines in gestation-specific trends in pregnancy hypertension rates at delivery among pregnancies at risk from 38 weeks, with the steepest declines at the later gestations (see online supplementary figure). Planned deliveries drove these changes in the gestational age distribution, as can be seen by the gestational age distribution in the labour onset groups (figure 3). Since 2001, inductions and prelabour caesarean sections have both increased from 36 to 39 weeks, with fewer women reaching 40 weeks and going into spontaneous labour. These patterns were also observed for pre-eclampsia and among nulliparae with a singleton pregnancy.

DISCUSSION

Principal findings

Continuing from the trend observed in the 1990s,[23] the rates of pregnancy hypertension and pre-eclampsia have continued to decline in this Australian population. On balance, the trends in maternal risk factors for pregnancy hypertension and predictive modelling suggest that pregnancy hypertension and pre-eclampsia

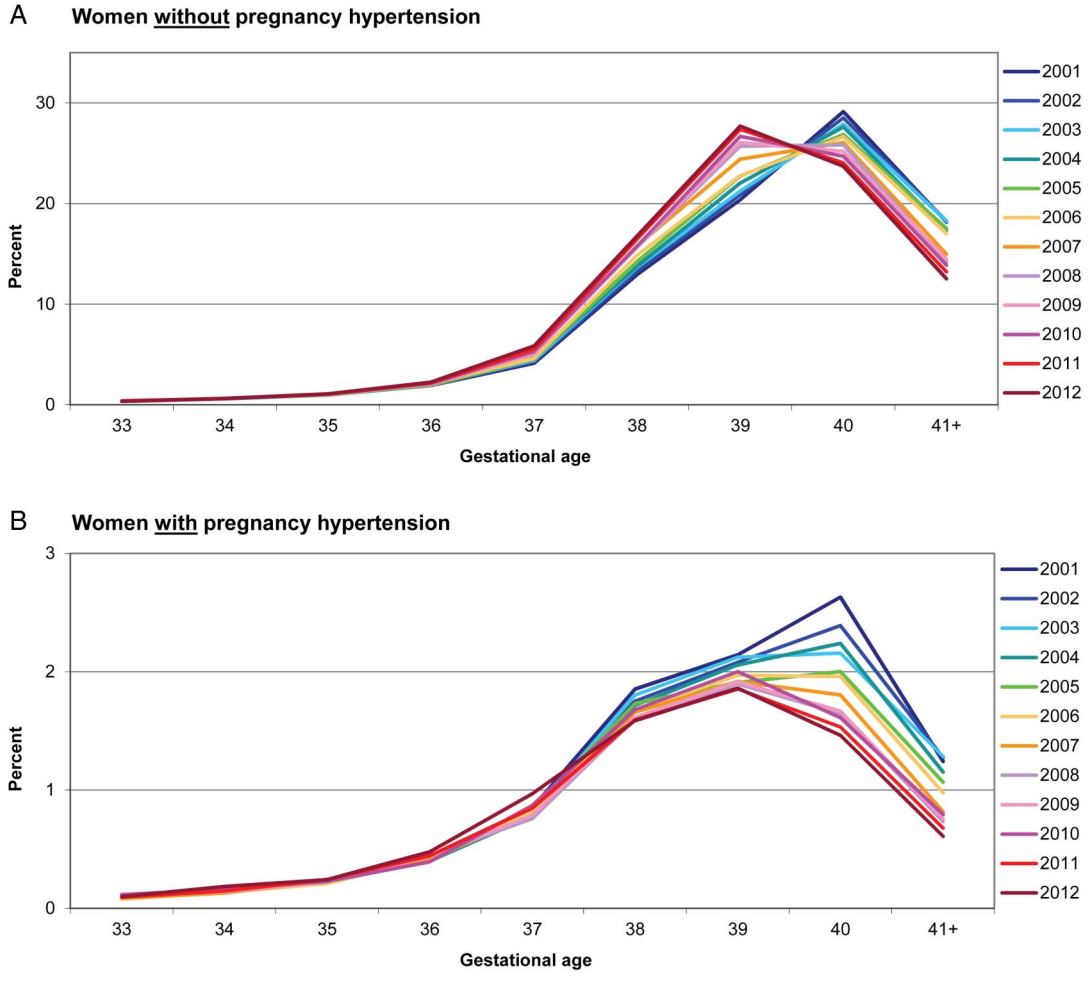

Figure 2 Annual distribution of gestational age 2001–2012 for women (A) without pregnancy hypertension at delivery and (B) with pregnancy hypertension at delivery.

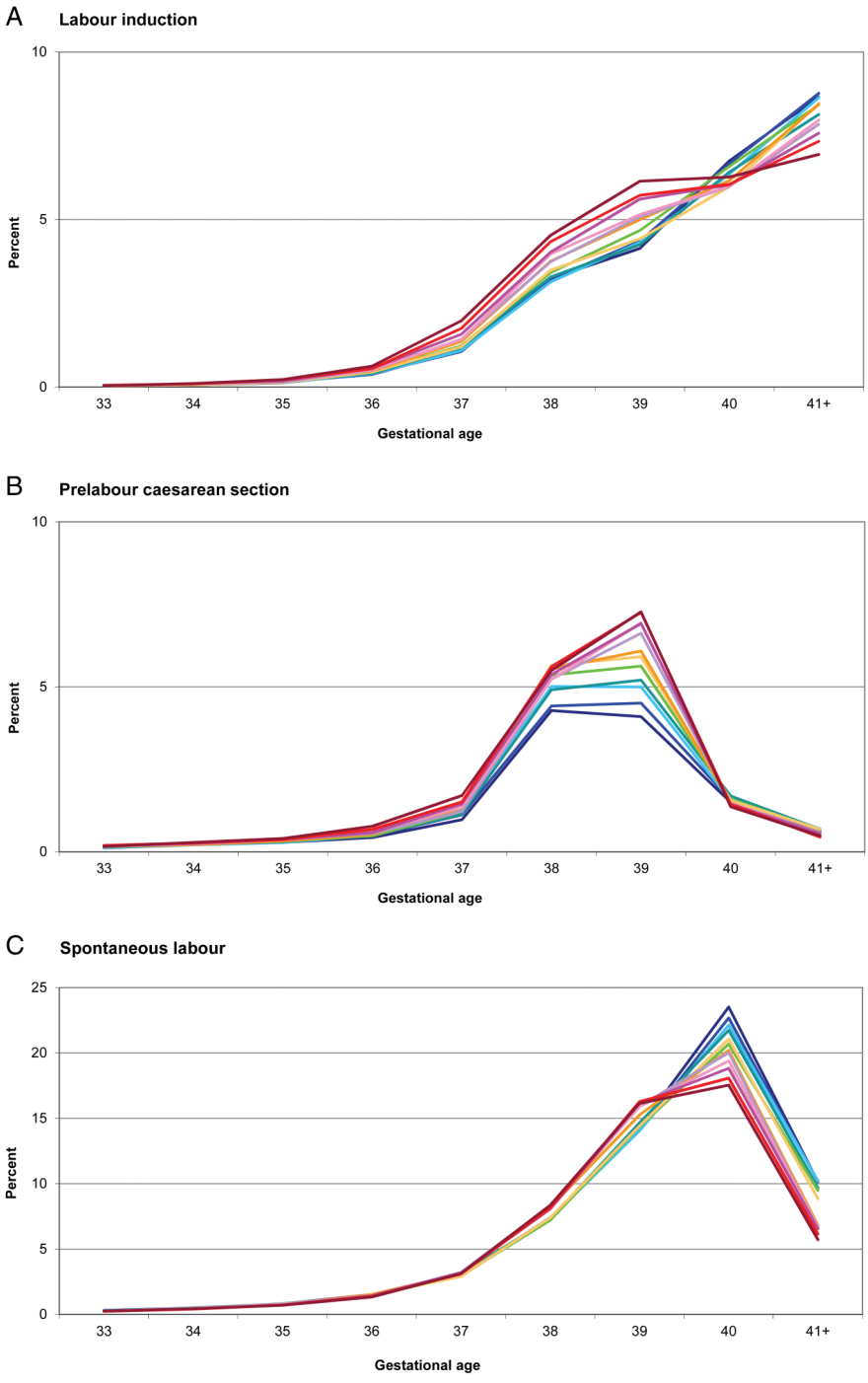

Figure 3 Distribution of gestational age among all deliveries 2001–2012, by year and labour onset.

should have increased over the 12 years, and that early planned deliveries explain some of the decline. Of note, there was no change in preterm pregnancy hypertension or pre-eclampsia rates over time. Rates start to decline among pregnancies that reached 38 weeks gestation, with the steepest declines at the latest gestations. While it is hard to conclusively demonstrate that hypertension has been prevented, our findings support the hypothesis that a consequence of increasing rates of planned early delivery is a reduction in the number of women who develop a hypertensive disorder of pregnancy. Furthermore, the gestation-specific trends are consistent with our hypothesis that women with risk factors for hypertension may be relatively more likely to be selected for early delivery. These findings are likely to be generalisable to other high-income countries with contemporaneous increases in earlier elective deliveries, although other contemporaneous changes could offset the trend.

Strengths and weaknesses of the study

Our study utilises large, reliably collected linked population health data.[36 38 44–47] International studies consistently demonstrate that pregnancy hypertension at the time of delivery is reliably and accurately reported in population health data.[37–40 50] Furthermore,

ascertainment is improved by accessing data from more than one data source and by longitudinal record linkage.[7 36 37] Use of broad diagnosis categories in administrative data has been demonstrated to overcome the under-ascertainment and misclassification that can occur with more specific diagnoses.[37 38 44 51] However, population-level data lack detailed clinical information on the reasons for obstetric interventions and on some factors that are known to increase (family history of pre-eclampsia, a new partner), or decrease (low-dose aspirin and calcium supplementation in high-risk women, treatment of gestational diabetes and use of anti-hypertensive medications) the risk of the hypertensive disorders of pregnancy. All these factors likely contribute to the lack of ability to fully explain the decline in pregnancy hypertension rates. Furthermore, investigation of the association between early planned delivery and a diagnosis of pregnancy hypertension is not well suited to the non-experimental methods, due to confounding by indication.[52] Pregnancy hypertension, or the potential for it, can be the cause of an early planned delivery, as well as being an outcome that could be averted by early planned delivery. The limited degree of information on indications for planned delivery (which may be multifactorial) necessitated examining the association in multiple ways, to confirm that the results were mutually consistent. In the absence of randomised controlled trials to specifically address this question, the contemporaneous changes provide circumstantial evidence of the impact of planned deliveries.

Interpretation

Several factors support a hypothesis of selective planned delivery for women at increased risk of pregnancy hypertension. First, the gestation-specific trends show that the most marked decline in rates of pregnancy hypertension occurs from 40 weeks (figure 2 and online supplementary figure). Second, we show strong associations between the established risk factors and early planned birth in the absence of hypertension. Third, the pregnancy hypertension rates declined significantly among nearly all the hypertension risk factors but also among planned births, and especially among planned birth at 37–39 weeks. Finally, including an indicator of planned birth in the predictive modelling suggests that planned births explain at least 36% of the decline in pregnancy hypertension. However, this is likely an underestimate because of the difficulty of accounting for indication.

A definitive answer to whether early planned birth prevents pregnancy hypertension would be obtained from a randomised trial. While no such trial exists, we reviewed 39 intervention trials of immediate delivery versus expectant management for specific conditions (eg, suspected growth restriction, macrosomia, prelabour rupture of the membranes, prolonged pregnancy, high-risk pregnancies, diabetes).[53–59] Only one trial (among pregnancies with suspected growth restriction) reports pregnancy hypertension as an outcome, finding a significant reduction in progression to pre-eclampsia in the induction group.[53] Furthermore, a programme aimed at decreasing early term elective delivery noted that, coincident with the decline in elective delivery at 37–38 weeks (from 28% to 3%), there was a 30% increase in pre-eclampsia (from 0.57% to 0.81%).[60] Of note, implementation of the findings of the HYPITAT trial (2009),[61] which showed that induction of labour for women with mild hypertensive disease at term improved maternal outcomes, should result in a decrease in the severity of the hypertensive disorders but not the overall incidence.

Changes to reporting of, or diagnostic criteria for, pregnancy hypertension are potential alternate explanations for declining pregnancy hypertension rates. In 2008, Australian guidelines changed to include non-proteinuric hypertension with multiorgan disease in the clinical diagnosis of pre-eclampsia.[62] However, this would not change the rate of the broad category of pregnancy hypertension and supports the use of this outcome over the study period. Data collection and coding has remained unchanged, and the ascertainment of pregnancy hypertension from the administrative data sets was consistent over the study period.

The clinical decision for a planned delivery requires balancing the potential benefits and harms of early birth for the mother and baby against those for continuing the pregnancy. The rising trend towards early planned delivery appears to be predicated on beliefs that the benefits outweigh the risks and that there is no significant short-term or long-term harm for the baby. However, recent research shows that babies born even 1–2 weeks early are more likely to have adverse sequelae in the newborn period, and are at increased risk of childhood hospitalisations and poorer school performance.[63–67] This has led to interventions aimed at reducing early term births unless there are compelling medical indications.[60 68–71]

In conclusion, planned deliveries increased dramatically over the study period, so that, by 2012, almost half (46%) of all women birthing in NSW had a planned delivery. Women with risk factors for de novo hypertension during pregnancy are increasingly likely to have a planned delivery. It appears that an unanticipated consequence of increasing rates of early planned delivery is a decline in the incidence of pregnancy hypertension. Reducing the length of gestation by even a few days means that a substantial number of women deliver before they become hypertensive.

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Contributors CLR and JBF conceived the study. CSA undertook data preparation and CLR provided statistical analysis, with CSA providing statistical and JMM clinical oversight. CLR, CSA, JMM and JBF had full access to all of the data (including statistical reports and tables) in the study, take responsibility for the integrity of the data and the accuracy of the data analysis, took part in interpretation of results and drafting of the manuscript, and approve and take responsibility for the final manuscript.

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