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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, preeclampsia and eclampsia) was the main outcome, preeclampsia was a secondary outcome

Results: From 2001 to 2012, pregnancy hypertension rates declined by 22% from 9.9% to 7.7% and preeclampsia 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% to 18.4%) and labour inductions by 10% (24.8% to 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 multi-fetal 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 (e.g. 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 inter-pregnancy interval, assisted reproductive technologies, family history of preeclampsia 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 anti-hypertensive medications.[12-18, 3, 19-21, 6, 11, 10] 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, preeclampsia and eclampsia) has declined in Australia, Scotland, Sweden and New York (USA), but not in Alberta (Canada), Denmark or Norway although preeclampsia declined in Norway and Denmark.[5, 22, 23] In contrast, both pregnancy hypertension and preeclampsia increased in the USA.[24, 23, 25] As most (~75%) pregnancy hypertension occurs at term,[11] increasing rates of planned delivery before the due date means some women may 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 commencing 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 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 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, 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 is available free of charge to all women in public hospitals. 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 datasets: the New South Wales (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 grams 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 was available (from July 2000 to December 2012) and of birth records from 1994. As Australia does not have a unique registration number for citizens, the separate datasets were linked using probabilistic linkage and a best practice approach in 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. Ethics approval for the study was obtained from the NSW Population and Health Services Research Ethics Committee (2002/12/430).

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 ICD10-AM) at any time during pregnancy.[36-38] Identification in this manner (using more than one data source) has been demonstrated to be accurate and reliable when

compared with medical records (e.g. sensitivity 82%, positive predictive value [PPV] 92% in NSW).[38] Preeclampsia was a *secondary outcome* because reporting in population data is less accurate (e.g. sensitivity 71%, PPV 67% in NSW) due to misclassification between gestational hypertension and preeclampsia.[39, 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 preeclampsia 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.[41, 1]

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 (one 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 preeclampsia that was available for analysis included: maternal age at delivery (<20, 20-34, ≥35 years), nulliparity, Asian ethnicity (based on country of birth), multi-fetal pregnancy, smoking during pregnancy, summer birth and inter-pregnancy interval ≥5 years (with lookback to 1994). Information on maternal medical conditions and reproductive history was obtained from for non-pregnancy and pregnancy-related hospital admissions from 2000, including chronic hypertension, diabetes (pregestational or gestational), morbid obesity, previous pregnancy hypertension, renal disease, autoimmune diseases (including rheumatoid arthritis, systemic lupus erythematosis and other rare autoimmune diseases)

and use of assisted reproductive technology (ART). 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.[42] Few records were missing data on these factors (<0.4%). The explanatory factors are reported with a high level of accuracy.[43, 36, 44, 45, 38, 46]

Statistical Analysis

Descriptive statistics were used to summarise the distribution and trends in pregnancy hypertension and preeclampsia, 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% confidence interval (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 [47] and all births in 2001-2002, two multivariable logistic regression predictive models for pregnancy hypertension were developed. The first included all available recognised risk factors for pregnancy hypertension and the second additionally included an indicator of planned birth other than for hypertension. Data from subsequent years were applied to the regression equation to account for the actual changes in maternal factors over time and produce a predicted trend for 2001-2012. Deliveries which had missing data (0.9%) on one or more risk factor were excluded from this analysis. Any difference in

the two predicted trends would suggest that planned births explained some of the predicted increase.

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 which 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).[48]

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 pregnancy hypertension rates declined by 22% from 9.9% to 7.7% Preeclampsia 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% to 10.1% and 5.0% to 3.5% respectively. All trend P-values were <0.0001.

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).

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

Risk factor	Risk factor prevalence		
	2001	2012	Trend over time
	N=84,302	N=96,051	P-value
	n (%)	n (%)	
Established risk factors for pregnancy hypertension			
Nulliparity	35,134 (41.7)	42,189 (43.9)	<0.0001
Maternal age ≥35 yrs	15,222 (18.1)	22,556 (23.5)	<0.0001
Maternal age <20 yrs	3,797 (4.5)	3,158 (3.3)	<0.0001
Diabetes	4000 (4.7)	8,812 (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)	2,971 (3.1)	<0.0001
Smoking	15,629 (18.5)	11,046 (11.5)	<0.0001
Multiple births	1,452 (1.7)	1,316 (1.4)	<0.0001
Autoimmune diseases	387 (0.5)	404 (0.4)	<0.0001
Previous pregnancy hypertension*	3,932 (8.0) †	3,929 (7.3)	<0.0001
Asian ethnicity	7,924 (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
Inter-pregnancy interval ≥5 yrs*	7,156 (15.9) †	7,461 (15.6)	0.62
Summer birth	20,143 (23.9)	23, 193 (24.2)	0.27
Obstetric factors			
Gestational age			

<37	5,457 (6.5)	6,649 (6.9)	<0.0001
37	4,215 (5.0)	6,544 (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
Pelabour 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
Socio-demographic factors			
Socio-economic 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	9,237 (11.0)	8,412 (8.8)	<0.0001
Private	19, 317 (22.9)	21,795 (22.7)	<0.0001

Percents may not add to 100% because of missing data

* among multipara

† reported rate is for 2003 (prior pregnancy hypertension) and 2006 (inter-pregnancy interval) allow a sufficient lookback period for previous pregnancies

In contrast changes in factors that would be consistent with a decrease in pregnancy hypertension rates included a significant decline in multi-fetal pregnancies, young maternal age, pregnancies

complicated by maternal autoimmune diseases, previous pregnancy 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 both labour inductions and 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).

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

Risk factor	Pregnancy hypertension rates				
	2001 n (row %)	2012 n (row %)	Trend over time P-value	Rate difference†	Rate ratio (95% CI) †
All women	8,356 (9.9)	7,433 (7.7)	<0.0001	-2.2	0.78 (0.76-0.80)
Maternal risk factors					
Nulliparity	4,827 (13.7)	4,362 (10.3)	<0.0001	-3.4	0.75 (0.72-0.78)
Maternal age ≥35 yrs	1,524 (10.0)	1,907 (8.5)	<0.0001	-1.6	0.84 (0.79-0.90)
Maternal age <20 yrs	394 (10.4)	304 (9.6)	0.18	-0.8	0.93 (0.80-1.07)

Diabetes	623 (15.6)	1,058 (12.0)	<0.0001	-3.6	0.77 (0.70-0.84)
Morbid obesity	56 (45.2)	196 (24.2)	<0.0001	-20.9	0.54 (0.43-0.68)
ART	200 (15.1)	283 (9.5)	<0.0001	-5.6	0.63 (0.53-0.75)
Smoking	1,183 (7.6)	763 (6.9)	<0.0001	-0.7	0.91 (0.83-0.99)
Multiple births	292 (20.1)	226 (17.2)	0.0013	-2.9	0.85 (0.73-1.00)
Autoimmune diseases	67 (16.5)	53 (13.1)	0.007	-3.4	0.79 (0.57-1.11)
Previous pregnancy hypertension*	1144 (36.3)	1249 (31.8)	0.0001	-4.5	0.88 (0.82-0.94)
Asian ethnicity	558 (7.0)	935 (5.3)	<0.0001	-1.6	0.75 (0.68-0.83)
Chronic hypertension	132 (24.1)	160 (25.7)	0.48	1.6	1.07 (0.87-1.30)
Renal disease	49 (26.5)	52 (18.7)	0.009	-7.8	0.71 (0.50-1.00)
Summer birth	1,800 (8.9)	1,722 (7.4)	<0.0001	-1.5	0.83 (0.78-0.89)
Obstetric factors					
Gestational age					
<37	1,005 (18.4)	1,203 (18.1)	0.35	-0.3	0.98 (0.89-1.07)
37	716 (17.0)	929 (14.2)	<0.0001	-2.8	0.84 (0.76-0.91)
38	1,562 (12.5)	1,524 (8.6)	<0.0001	-3.9	0.69 (0.65-0.74)
39	1,805 (9.5)	1,788 (6.3)	<0.0001	-3.2	0.66 (0.62-0.71)
40	2,218 (8.3)	1,404 (5.8)	<0.0001	-2.5	0.70 (0.66-0.75)
≥41	1,047 (6.4)	584 (4.6)	<0.0001	-1.8	0.72 (0.65-0.80)
Planned deliveries (any)	5,719 (18.0)	5,604 (12.8)	<0.0001	-5.2	0.71 (0.69-0.74)
Prelabour CS	1,484 (13.6)	1,819 (10.3)	<0.0001	-3.3	0.76 (0.71-0.81)
Labour inductions	4,235 (20.3)	3,785 (14.5)	<0.0001	-5.8	0.72 (0.69-0.74)
Planned delivery at 37-39 weeks	2,932 (19.5)	3,281 (12.6)	<0.0001	-7.0	0.64 (0.62-0.67)

* among multipara

† Among women with the specified risk factor in 2012 compared with 2001

The relative decrease (rate ratio) in pregnancy hypertension rates was greatest among morbidly obese women and least (but still statistically significant) among smokers. Compared to 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 1.56-1.59), multiple pregnancy (RR=1.76, 95%CI 1.71-1.81), diabetes (RR=2.07, 95%CI 2.04-2.10), chronic hypertension (RR=1.95, 95%CI 1.87-2.03), morbid obesity (RR=1.94, 95%CI 1.83-2.06), renal disease (RR=1.21, 95%CI 1.12-1.32), autoimmune diseases (RR=1.81, 95%CI 1.72-1.91), use of assisted reproductive technology (RR=1.63, 95%CI 1.60-1.67), and pregnancy hypertension in a previous pregnancy (RR=1.62, 95%CI 1.59-1.65). 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 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, 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 (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, both inductions and prelabour caesarean sections have increased from 36 – 39 weeks with fewer women reaching 40 weeks and going into spontaneous labour. These patterns were also observed for preeclampsia 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 preeclampsia 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 preeclampsia 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 rates. 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. [43, 36, 44, 45, 38, 46] International studies consistently demonstrate that pregnancy hypertension at the time of delivery are reliably and accurately reported in population health data.[39, 37, 38, 40, 49] Furthermore, ascertainment is improved by accessing data from more than one data source and by longitudinal record linkage.[36, 37, 7] 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.[43, 37, 38, 50] 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 preeclampsia, 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. 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.[51] Pregnancy hypertension, or the potential for it, can be the cause of an early planned delivery, as well as being an outcome which 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 the most marked decline in rates of pregnancy hypertension occur at from 40 weeks (Figure 2, 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).[52-58] Only one trial (among pregnancies with suspected growth restriction) reports pregnancy hypertension as an outcome, finding a significant reduction in progression to preeclampsia in the induction group.[52] Of note, implementation of findings of the HYPITAT trial (2009),[59] 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 multi-organ disease in the clinical diagnosis of preeclampsia.[60] 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.

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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COMPETING INTERESTS STATEMENT

Competing interests: All authors have completed the ICMJE uniform disclosure form at http://www.icmje.org/coi_disclosure.pdf and declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.

CONTRIBUTION TO AUTHORSHIP

CR and JF conceived the study. CA undertook data preparation and CR provided statistical analysis, with CA providing statistical and JM clinical oversight. CR, CA, JM and JF 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, drafting the manuscript, approve and take responsibility for the final manuscript.

TRANSPARENCY DECLARATION

The lead author (CR) affirms that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned have been explained.

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DATA SHARING

Data are not available for sharing.

ETHICAL APPROVAL

Ethical approval was obtained from the NSW Population and Health Services Research Ethics Committee (Reference No. 2012-12-430).

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List of Figures

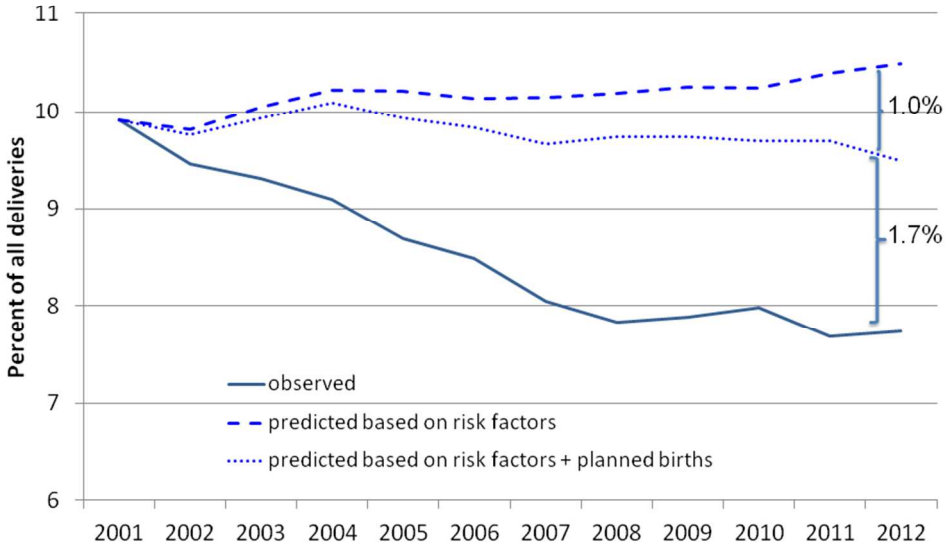

Figure 1: Observed and predicted trends in pregnancy hypertension, NSW 2001-2012
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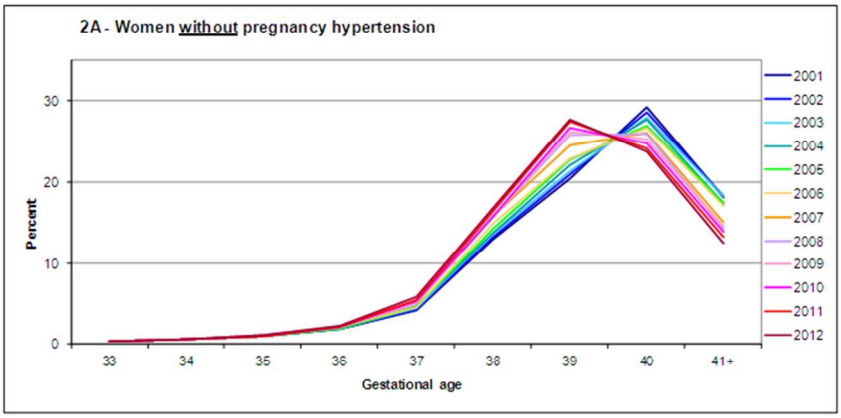


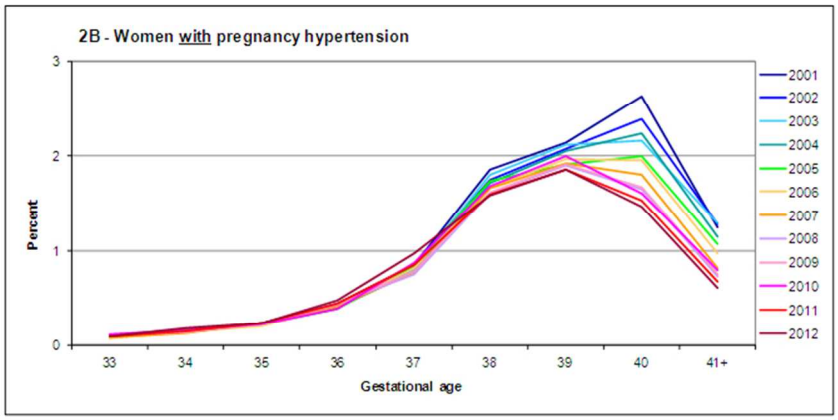


Figure 2: Annual distribution of gestational age 2001-2012 for women A) without pregnancy hypertension at delivery and B) with pregnancy hypertension at delivery
190x254mm (96 x 96 DPI)

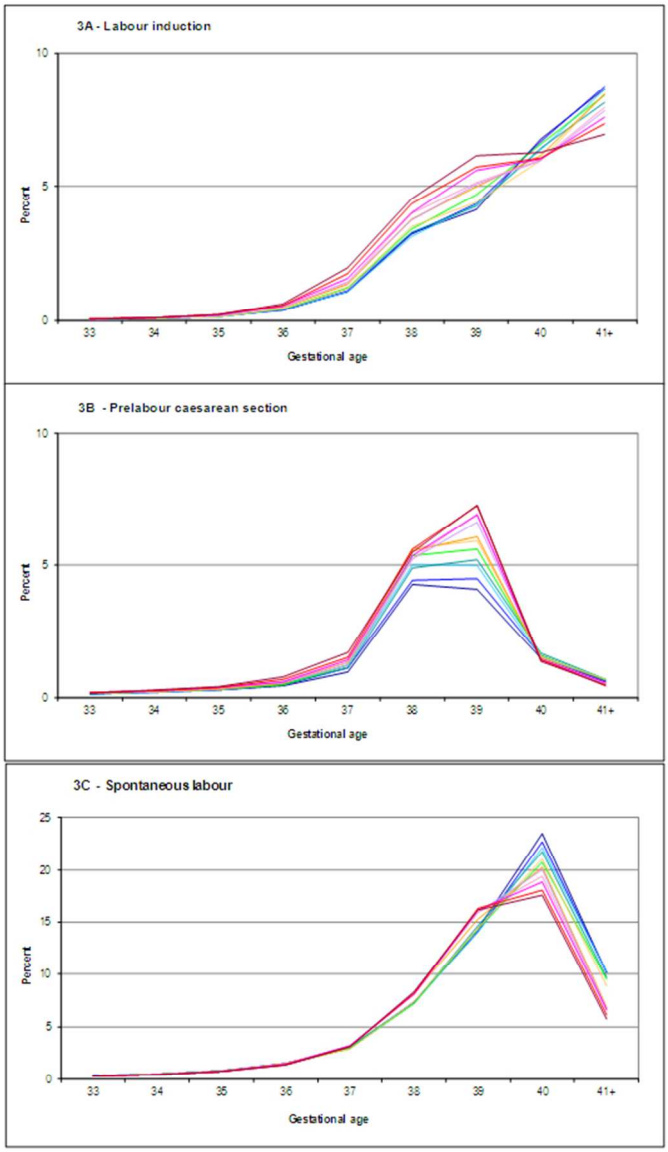
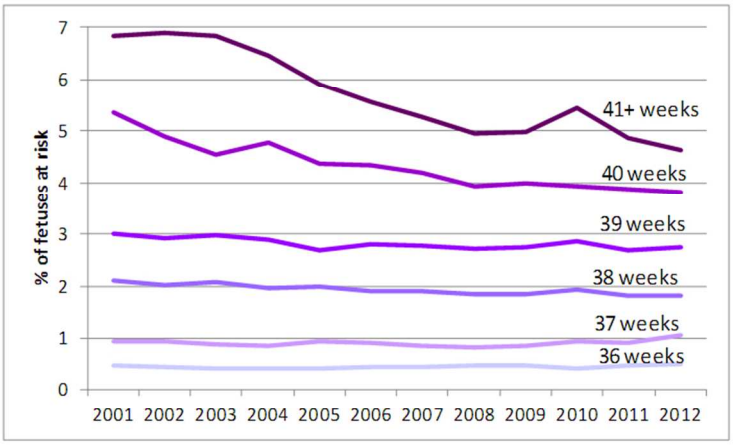


Figure 3: Distribution of gestational age among all deliveries 2001-2012, by year and labour onset.
190x254mm (96 x 96 DPI)



Supplementary Figure: Gestation specific rates of pregnancy hypertension at delivery among pregnancies-at-risk, 2001-2012
254x190mm (96 x 96 DPI)

STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cohort studies*

Section/Topic	Item #	Recommendation	Reported on BMJ Open page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1,2
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4-5
Objectives	3	State specific objectives, including any prespecified hypotheses	5
Methods			
Study design	4	Present key elements of study design early in the paper	4-5
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5-6
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up	5-6
		(*b*) For matched studies, give matching criteria and number of exposed and unexposed	NA
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	6-7
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	7-8
Bias	9	Describe any efforts to address potential sources of bias	8
Study size	10	Explain how the study size was arrived at	5
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	7-8
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	8-9
		(*b*) Describe any methods used to examine subgroups and interactions	8-9
		(*c*) Explain how missing data were addressed	8, 11 (Table 1.)
		(*d*) If applicable, explain how loss to follow-up was addressed	NA
		(*e*) Describe any sensitivity analyses	NA
Results			

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	9
		(b) Give reasons for non-participation at each stage	NA
		(c) Consider use of a flow diagram	-
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	9-11
		(b) Indicate number of participants with missing data for each variable of interest	8, 11
		(c) Summarise follow-up time (eg, average and total amount)	NA
Outcome data	15*	Report numbers of outcome events or summary measures over time	9,12
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	28, (Figure 1)
		(*b*) Report category boundaries when continuous variables were categorized	NA
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	12-13 (Table 2)
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	14-15
Discussion			
Key results	18	Summarise key results with reference to study objectives	15-16
Limitations			
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	17-18
Generalisability	21	Discuss the generalisability (external validity) of the study results	16
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	20

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

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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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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, preeclampsia and eclampsia) was the main outcome, preeclampsia was a secondary outcome

Results: From 2001 to 2012, pregnancy hypertension rates declined by 22% from 9.9% to 7.7% and preeclampsia 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% to 18.4%) and labour inductions by 10% (24.8% to 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 multi-fetal 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 (e.g. 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 inter-pregnancy interval, assisted reproductive technologies, family history of preeclampsia 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 anti-hypertensive 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, preeclampsia and eclampsia) has declined in Australia, Scotland, Sweden and New York (USA), but not in Alberta (Canada), Denmark or Norway although preeclampsia declined in Norway and Denmark.[5, 22, 23] In contrast, both pregnancy hypertension and preeclampsia 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 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 commencing 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, 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 is available free of charge to all women in public hospitals. 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 datasets: the New South Wales (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 grams 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 was available (from July 2000 to December 2012) and of birth records from 1994. As Australia does not have a unique registration number for citizens, the separate datasets were linked using probabilistic linkage and a best practice approach in 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. Ethics approval for the study was obtained from the NSW Population and Health Services Research Ethics Committee (2002/12/430).

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 ICD10-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 (e.g. sensitivity 82%, positive predictive value [PPV] 92% in NSW).[38] Preeclampsia was a *secondary outcome* because reporting in population data is less accurate (e.g. sensitivity 71%, PPV 67% in NSW) due to misclassification between gestational hypertension and preeclampsia.[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 preeclampsia 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 (one 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 preeclampsia that was available for analysis included: maternal age at delivery (<20, 20-34, ≥35 years), nulliparity, Asian ethnicity (based on country of birth), multi-fetal pregnancy, smoking during pregnancy, summer birth and inter-pregnancy interval ≥5 years (with lookback to 1994). Information on maternal medical conditions and reproductive history was obtained (as reported versus not reported) from birth records from 1994, and non-pregnancy and pregnancy-related hospital admissions from 2000, including chronic hypertension, diabetes (pregestational or gestational), morbid obesity, previous

pregnancy hypertension, renal disease, autoimmune diseases (including rheumatoid arthritis, systemic lupus erythematosis 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 preeclampsia, 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% confidence interval (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 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 which 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%. Preeclampsia 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% to 10.1% and 5.0% to 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 preeclampsia (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).

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 yrs	15,222 (18.1)	22,556 (23.5)	<0.0001
Maternal age <20 yrs	3,797 (4.5)	3,158 (3.3)	<0.0001
Diabetes (any)	4000 (4.7)	8,812 (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)	2,971 (3.1)	<0.0001
Smoking	15,629 (18.5)	11,046 (11.5)	<0.0001
Multiple births	1,452 (1.7)	1,316 (1.4)	<0.0001

Autoimmune diseases	387 (0.5)	404 (0.4)	<0.0001
Previous pregnancy hypertension*	3,932 (8.0) †	3,929 (7.3)	<0.0001
Asian ethnicity	7,924 (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
Inter-pregnancy interval ≥5 yrs*	7,156 (15.9) †	7,461 (15.6)	0.62
Summer birth	20,143 (23.9)	23, 193 (24.2)	0.27
Obstetric factors			
Gestational age			
<37	5,457 (6.5)	6,649 (6.9)	<0.0001
37	4,215 (5.0)	6,544 (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
Pelabour 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
Socio-demographic factors			
Socio-economic 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	9,237 (11.0)	8,412 (8.8)	<0.0001
Private	19, 317 (22.9)	21,795 (22.7)	<0.0001

Percents 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, 45765 (4.3%) inter-pregnancy 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 (inter-pregnancy interval) allow a sufficient lookback period for previous pregnancies

In contrast changes in factors that would be consistent with a decrease in pregnancy hypertension rates included a significant decline in multi-fetal pregnancies, young maternal age, pregnancies complicated by maternal autoimmune diseases, previous pregnancy 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 both labour inductions and 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 to 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).

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

Risk factor	Pregnancy hypertension rates				
	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	8,356 (9.9)	7,433 (7.7)	<0.0001	-2.2	0.78 (0.76-0.80)
Maternal risk factors					
Nulliparity	4,827 (13.7)	4,362 (10.3)	<0.0001	-3.4	0.75 (0.72-0.78)
Maternal age ≥35 yrs	1,524 (10.0)	1,907 (8.5)	<0.0001	-1.6	0.84 (0.79-0.90)
Maternal age <20 yrs	394 (10.4)	304 (9.6)	0.18	-0.8	0.93 (0.80-1.07)
Diabetes (any)	623 (15.6)	1,058 (12.0)	<0.0001	-3.6	0.77 (0.70-0.84)
Morbid obesity	56 (45.2)	196 (24.2)	<0.0001	-20.9	0.54 (0.43-0.68)
ART	200 (15.1)	283 (9.5)	<0.0001	-5.6	0.63 (0.53-0.75)
Smoking	1,183 (7.6)	763 (6.9)	<0.0001	-0.7	0.91 (0.83-0.99)
Multiple births	292 (20.1)	226 (17.2)	0.0013	-2.9	0.85 (0.73-1.00)
Autoimmune diseases	67 (16.5)	53 (13.1)	0.007	-3.4	0.79 (0.57-1.11)
Previous pregnancy hypertension*	1144 (36.3)	1249 (31.8)	0.0001	-4.5	0.88 (0.82-0.94)
Asian ethnicity	558 (7.0)	935 (5.3)	<0.0001	-1.6	0.75 (0.68-0.83)
Chronic hypertension	132 (24.1)	160 (25.7)	0.48	1.6	1.07 (0.87-1.30)
Renal disease	49 (26.5)	52 (18.7)	0.009	-7.8	0.71 (0.50-1.00)
Summer birth	1,800 (8.9)	1,722 (7.4)	<0.0001	-1.5	0.83 (0.78-0.89)

Obstetric factors					
Gestational age					
<37	1,005 (18.4)	1,203 (18.1)	0.35	-0.3	0.98 (0.89-1.07)
37	716 (17.0)	929 (14.2)	<0.0001	-2.8	0.84 (0.76-0.91)
38	1,562 (12.5)	1,524 (8.6)	<0.0001	-3.9	0.69 (0.65-0.74)
39	1,805 (9.5)	1,788 (6.3)	<0.0001	-3.2	0.66 (0.62-0.71)
40	2,218 (8.3)	1,404 (5.8)	<0.0001	-2.5	0.70 (0.66-0.75)
≥41	1,047 (6.4)	584 (4.6)	<0.0001	-1.8	0.72 (0.65-0.80)
Planned deliveries (any)	5,719 (18.0)	5,604 (12.8)	<0.0001	-5.2	0.71 (0.69-0.74)
Prelabour CS	1,484 (13.6)	1,819 (10.3)	<0.0001	-3.3	0.76 (0.71-0.81)
Labour inductions	4,235 (20.3)	3,785 (14.5)	<0.0001	-5.8	0.72 (0.69-0.74)
Planned delivery at 37-39 weeks	2,932 (19.5)	3,281 (12.6)	<0.0001	-7.0	0.64 (0.62-0.67)

* among multipara

† Among women with the specified risk factor in 2012 compared with 2001

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 1.56-1.59), multiple pregnancy (RR=1.76, 95%CI 1.71-1.81), diabetes (RR=2.07, 95%CI 2.04-2.10), chronic hypertension (RR=1.95, 95%CI 1.87-2.03), morbid obesity (RR=1.94, 95%CI 1.83-2.06), renal disease (RR=1.21, 95%CI 1.12-1.32), autoimmune diseases (RR=1.81, 95%CI 1.72-1.91), use of assisted reproductive technology (RR=1.63, 95%CI 1.60-1.67), and pregnancy hypertension in a previous pregnancy (RR=1.62, 95%CI 1.59-1.65). 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 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, 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 (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, both inductions and prelabour caesarean sections have increased from 36 – 39 weeks with fewer women reaching 40 weeks and going into spontaneous labour. These patterns were also observed for preeclampsia 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 preeclampsia 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 preeclampsia 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 preeclampsia 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 have 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 preeclampsia, 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 which 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 the most marked decline in rates of pregnancy hypertension occur from 40 weeks (Figure 2, 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 preeclampsia in the induction group.[53] Furthermore, a program 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 preeclampsia (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 multi-organ disease in the clinical diagnosis of preeclampsia.[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 are no significant short or long-term harms for the baby. However, recent research is 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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COMPETING INTERESTS STATEMENT

Competing interests: All authors have completed the ICMJE uniform disclosure form at http://www.icmje.org/coi_disclosure.pdf and declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.

CONTRIBUTION TO AUTHORSHIP

CR and JF conceived the study. CA undertook data preparation and CR provided statistical analysis, with CA providing statistical and JM clinical oversight. CR, CA, JM and JF 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, drafting the manuscript, approve and take responsibility for the final manuscript.

TRANSPARENCY DECLARATION

The lead author (CR) affirms that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned have been explained.

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DATA SHARING

Data are not available for sharing.

ETHICAL APPROVAL

Ethical approval was obtained from the NSW Population and Health Services Research Ethics Committee (Reference No. 2012-12-430).

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List of Figures

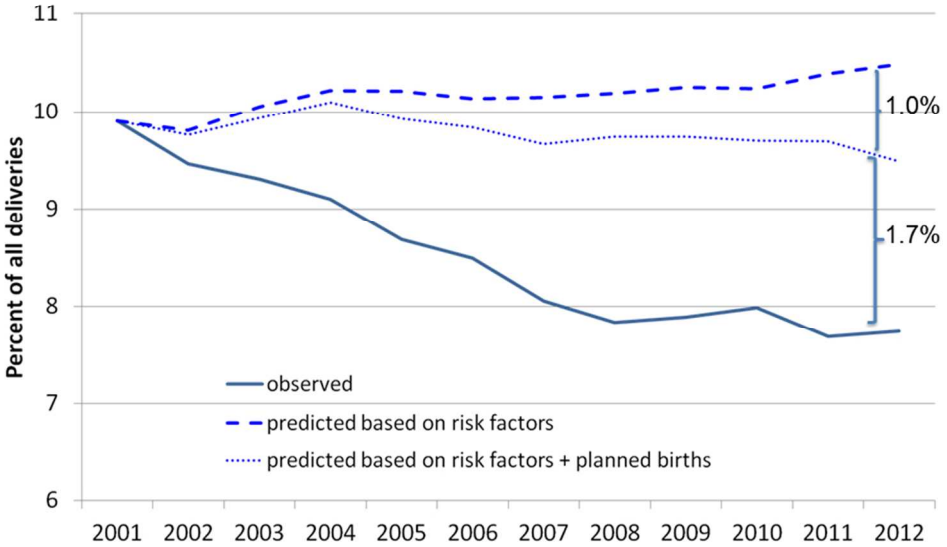

Figure 1: Observed and predicted trends in pregnancy hypertension, NSW 2001-2012
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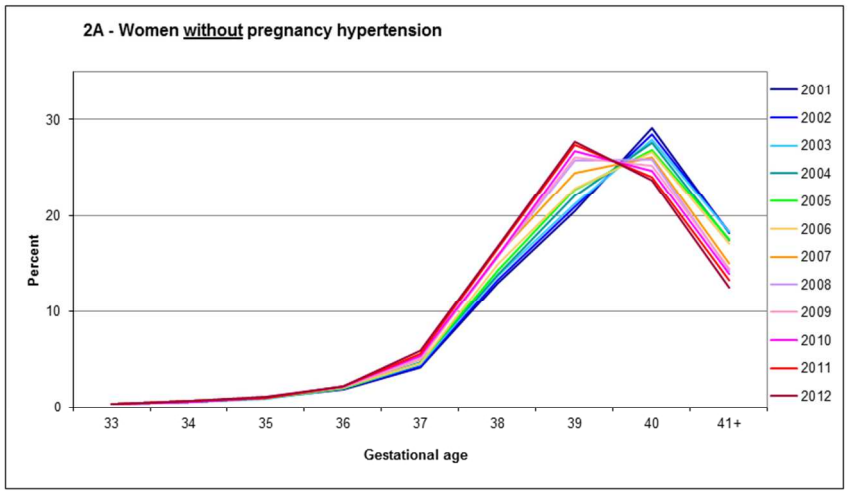

Figure 2: Annual distribution of gestational age 2001-2012 for women A) without pregnancy hypertension at delivery
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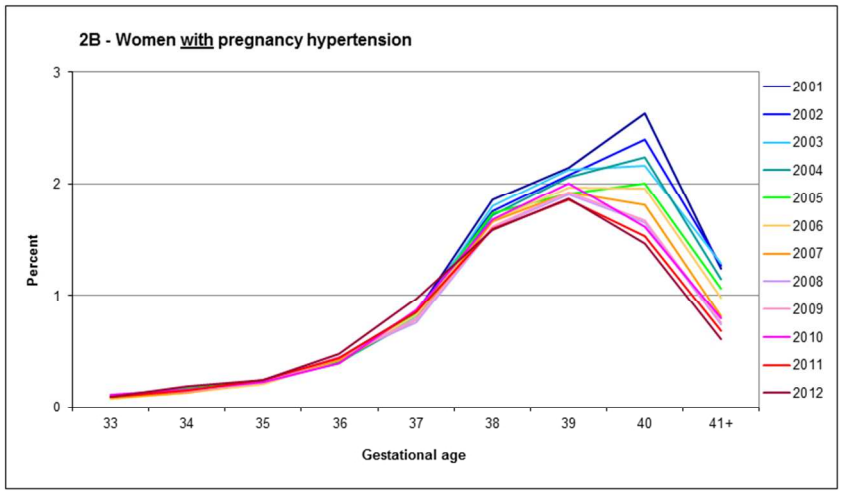

Figure 2: Annual distribution of gestational age 2001-2012 for women with pregnancy hypertension at delivery B) with pregnancy hypertension at delivery
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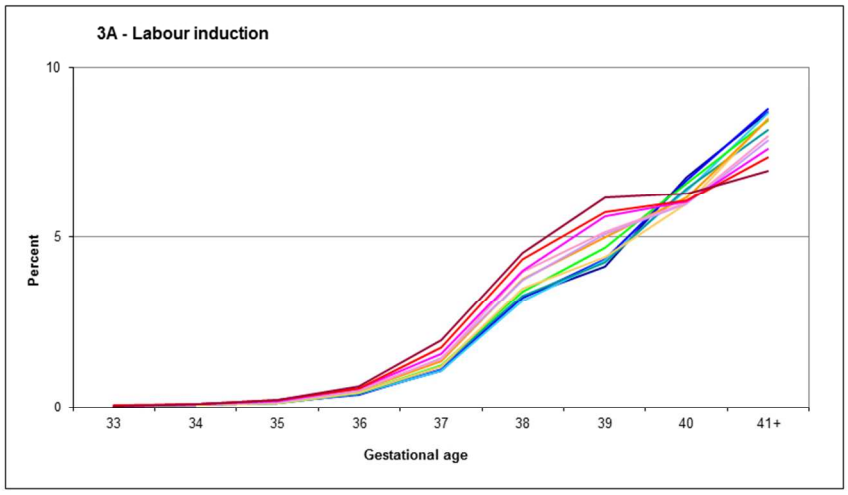

Figure 3: Distribution of gestational age among all deliveries 2001-2012, by year and labour onset A) Labour inductions
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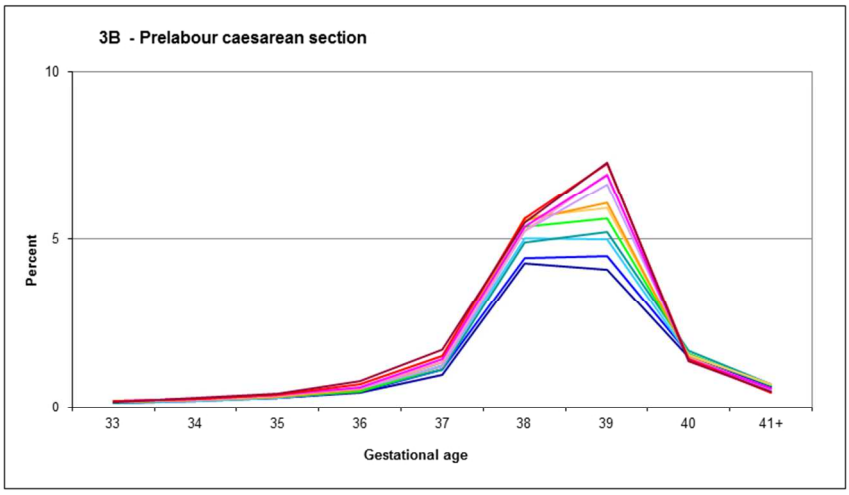

Figure 3: Distribution of gestational age among all deliveries 2001-2012, by year and labour onset B) Prelabour caesarean sections
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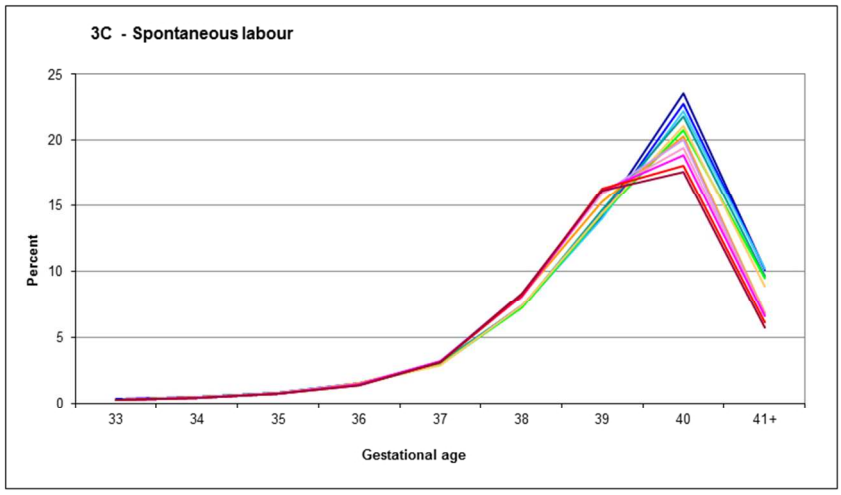

Figure 3: Distribution of gestational age among all deliveries 2001-2012, by year and labour onset C) Spontaneous labour
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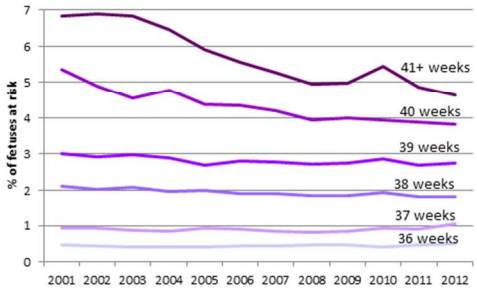

Supplementary Figure: Gestation specific rates of pregnancy hypertension at delivery among pregnancies-at-risk, 2001-2012
254x190mm (96 x 96 DPI)

STROBE 2007 (v4) Statement—Checklist of items that should be included in reports of *cohort studies*

Section/Topic	Item #	Recommendation	Reported on BMJ Open page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1,2
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4-5
Objectives	3	State specific objectives, including any prespecified hypotheses	5
Methods			
Study design	4	Present key elements of study design early in the paper	4-5
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	5-6
Participants	6	(*a*) Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up	5-6
		(*b*) For matched studies, give matching criteria and number of exposed and unexposed	NA
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	6-7
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	7-8
Bias	9	Describe any efforts to address potential sources of bias	8
Study size	10	Explain how the study size was arrived at	5
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	7-8
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	8-9
		(*b*) Describe any methods used to examine subgroups and interactions	8-9
		(*c*) Explain how missing data were addressed	8, 11 (Table 1.)
		(*d*) If applicable, explain how loss to follow-up was addressed	NA
		(*e*) Describe any sensitivity analyses	NA
Results			

Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	9
		(b) Give reasons for non-participation at each stage	NA
		(c) Consider use of a flow diagram	-
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	9-11
		(b) Indicate number of participants with missing data for each variable of interest	8, 11
		(c) Summarise follow-up time (eg, average and total amount)	NA
Outcome data	15*	Report numbers of outcome events or summary measures over time	9,12
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	28, (Figure 1)
		(*b*) Report category boundaries when continuous variables were categorized	NA
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	12-13 (Table 2)
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	14-15
Discussion			
Key results	18	Summarise key results with reference to study objectives	15-16
Limitations			
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	17-18
Generalisability	21	Discuss the generalisability (external validity) of the study results	16
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	20

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.