The final version of this paper was published in *Annals of Rheumatic Diseases* 2013;72:547–551. doi:10.1136/annrheumdis-2011-201210

Second pregnancy outcomes for women with systemic lupus erythematosus

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ABSTRACT

Background Systemic lupus erythematosus (SLE) is associated with adverse pregnancy outcomes overall.

Objective To examine the outcomes for women with SLE in a pregnancy subsequent to a first birth with an adverse outcome.

Methods A population-based cohort study was carried out of 794 577 deliveries to 532 612 women giving birth in New South Wales, Australia from 2001 to 2009. Data were obtained from longitudinally linked birth records and hospital records.

Results 675 women had a diagnosis of SLE in the study period (prevalence 127 per 100 000 childbearing women). Of 177 women who had a first nulliparous birth and subsequent pregnancy, 10 (5.6%) had a perinatal death in the first pregnancy, and of these women, 9 (90%) had a baby discharged home alive in the second pregnancy. Of the 167 women whose first-birth infants survived, second pregnancy outcomes included: 18 (11%) admission for spontaneous abortion, 1 perinatal death (0.6%) and 148 (89%) infants discharged home. Two women had a thromboembolic event in their first pregnancy but had no thromboembolic event in the second pregnancy on pregnancy but had no thromboembolic event in the second pregnancies only.

Conclusion Women with SLE are at high risk of adverse pregnancy outcomes. However, those who have a perinatal death in their first pregnancy can expect a live birth for a subsequent pregnancy.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a multisystem

autoimmune connective tissue disease that predominantly affects women of childbearing age. A review of prevalence studies, mainly from the 1990s, reported rates for Caucasian populations ranging from 50 to 130/100 000 women.1

A systematic review and meta-analysis of pregnancy outcomes in women with SLE published in 2010 included 37 studies of 2751 pregnancies in 1842 women.2 The majority of studies (n=29) were small case series, with the largest including 203 women with 267 pregnancies.3 Compared with women without SLE, women with SLE have greater rates of pregnancy complications, including higher rates of pre-eclampsia, gestational hypertension, eclampsia and lupus fl are.2 These findings have been confirmed in large studies of hospital records; however, hospital database studies have been limited by an inability to track longitudinally individuals with one or multiple hospital admissions.3 4 The hospitalisation studies also suggest that women with SLE have higher rates of caesarean section and higher rates and duration of hospital admission.3 4 Fetal or neonatal complications in women with SLE compared with those without SLE have been widely reported and include spontaneous abortion, intrauterine growth restriction, preterm birth, stillbirth and neonatal death, as well as congenital heart block in women with autoantibodies to anti- SSA/Ro and anti-SSB/La.2 More recent studies have suggested improved fetal and neonatal outcomes compared with earlier studies.5

Outcomes for women with SLE in a pregnancy subsequent to a first birth with an adverse outcome have not been reported. This lack of information for counselling arose in discussions subsequent to a woman with SLE presenting with a first pregnancy stillbirth. Therefore the aim of our study was to determine the second pregnancy incidence and recurrence of perinatal mortality and maternal morbidity among women with SLE.

SUBJECTS AND METHODS

Study population

The study population included all women who gave birth in New South Wales (NSW), Australia during 2001–8. NSW is the most populous Australian state with a population of ~ 6.9 million and one-third of all Australian births (~95 000 a year) in over 100 hospitals, ranging from small rural hospitals to tertiary centres. Australian healthcare includes a mixture of public and private care; all women are covered by national health insurance, which provides access to hospital and specialist care but about one-third take out private insurance for obstetric care.

Data sources

Data were obtained from longitudinally linked population datasets for births during the years 2001–9: the NSW Perinatal Data Collection (PDC), a population-based surveillance system of all births (\geq 20 weeks' gestation or \geq 400 g birth weight) and the NSW Admitted Patient Data Collection (APDC), an administrative database of all public and private hospital admissions in NSW. The separate datasets were linked using probabilistic linkage methods and were made available to the researchers as deidentified data.6 Longitudinal linkage allows identification of individuals with multiple hospital records and consecutive pregnancies. The linkage and use of the data was approved by the NSW Population and Health Services Research Ethics Committee. The PDC records were used to identify nulliparous women with a first birth of \geq 20 weeks' gestation during 2001 to 2008 and to identify subsequent pregnancies and births up until 31 December 2009.

SLE diagnosis

Women with SLE were identified from the APDC, based on a diagnosis of SLE made in the medical records by an attending doctor and subsequently coded into the discharge summary. More than 20 diagnoses and procedures for each hospital admission can be

recorded, and are coded according to the International Classifi \cation of Diseases version 10 Australian modifi \cation (ICD10). SLE (ICD10 code M32) could have appeared as the primary admitting diagnosis or as a comorbid condition for nonpregnancy or pregnancy-related admissions.

SLE is reliably identified in hospital data when the diagnosis is recorded in the medical record. A US validation of 234 inpatients with SLE found administrative data to ascertain 89% of cases with few false positives (positive predictive value 89%).7 This is consistent with three local validation studies of hospital data identification of connective tissue disorders, which found reporting sensitivities of 85–100% and specificities of 99.9–100%.8–10

Pregnancy factors

Linked APDC discharge summaries were available from July 2000 to December 2009 and included pregnancy and non-pregnancy admissions. Only spontaneous abortions which result in admission to hospital can be identified in the APDC. The APDC was also used to identify conditions representing potential nephritis or renal involvement during pregnancy, including glomerular disorders, tubulo-interstitial disorders, pre-eclampsia or eclampsia, renal biopsy, renal failure or dialysis. The APDC and PDC were used to identify women who had pregnancy hypertension (including gestational hypertension, pre-eclampsia and eclampsia), diabetes (pre-existing and gestational) and obstetric haemorrhage (antepartum or postpartum) and thromboembolic events (puerperal deep vein thrombosis, pulmonary embolism or cerebral venous thromboembolism), antenatally and up to 42 days after delivery. Transfusion was identified from procedure codes recorded in the discharge summaries, and included blood products such as fresh frozen plasma or platelets, as well as packed red blood cell transfusion. Gestational age at delivery and infant birthweight were recorded in the PDC. Infant discharge status (stillbirth, neonatal death or discharge alive) was ascertained from the PDC and the APDC. Stillbirth was defined as a fetal death of at least 20 weeks' gestation or 400 g birth weight. Neonatal death was defined as a death during the first 28 days or where the infant was never discharged home alive. Perinatal deaths include both stillbirths and neonatal deaths. Small for gestational age was defined as below the third birth weight centile for gestational age and infant sex.11 Only factors that we have previously shown to be reliably reported in the population health data were included in the analyses.8 9 12-15

Analysis

The initial analysis compared pregnancy outcomes in mothers ever diagnosed with SLE with mothers without SLE. Further analyses were limited to nulliparous women with a record of SLE before their second consecutive birth, as our primary aim is to enable counselling of women with SLE and a second pregnancy. We used frequency distributions and contingency table analyses to describe the pregnancies and outcomes experienced by women with and without SLE. The relationship between SLE and maternal factors and pregnancy outcomes is reported as relative risk (RR) with 95% CI.

RESULTS

From 2001 through 2009 there were 794 577 deliveries to 532 612 women, including 1058 deliveries to 675 women with an SLE diagnosis recorded during the study period (Figure 1). This gives a prevalence rate of SLE of 127 per 100 000 childbearing women. Compared with women without SLE, the rate of adverse outcomes in women with SLE ever recorded was increased (table 1). There were no maternal deaths among women with SLE. Figure 1 is a schematic diagram of how the study population was derived. There were 350 nulliparous women with a first birth and who ever had an admission with SLE in the study period; for 234 (67%) a diagnosis of SLE was recorded before their first delivery. The rate of stillbirth in that first birth was higher in the women whose diagnosis was not recorded by the time of that first birth than in those who already had a diagnosis (7% vs 3%) but the risk was not statistically significant: RR=2.7 (0.95–7.6). The neonatal death rate was 1% both for diagnosed and

undiagnosed women. For 37 of these women the earliest recorded diagnosis of SLE was after a second birth; they were excluded from further analyses. These excluded women were older at the time of their first recorded diagnosis of SLE (31.3 years (SD 4.7)) compared with the 313 women who remained in the analysis (29.8 years (SD 5.3)). The 37 women who were not diagnosed until after their second pregnancy also had a spontaneous abortion rate of 30% for that second pregnancy.

Maternal and infant characteristics of the 313 women who remained in the analysis are shown in table 2. The median period between first and second pregnancies was 2.3 years (IQR 1.6–3.0); however, women with a first birth in the latter part of the study period had less time in follow-up for a second birth. Among the subgroup of women whose first birth was in 2001–4, 119/172 (69%) recorded a second pregnancy.

The birth outcomes for the 177 women who had a second pregnancy are shown in table 3. Of the 10 women who had a stillbirth or neonatal death in their first pregnancy, nine (90%) had a live-born infant who was discharged home in their second pregnancy while one woman had a spontaneous abortion at \geq 14 weeks. Among the 167 mothers whose firstborn was discharged home alive there was only one stillbirth and no neonatal deaths in the second pregnancy but there were 18 spontaneous abortions (11%). None of the spontaneous abortions were coded as \geq 14 weeks' gestation, although two were of unknown gestation. None of these first-born infants, but two of the second-born infants, were diagnosed with atrioventricular block.

Table 4 shows the occurrence and recurrence of serious maternal adverse events in the first and second pregnancies. The persistence or recurrence of renal impairment in a second pregnancy was common but obstetric haemorrhage recurrence was not.

DISCUSSION

This is the largest cohort study of SLE in pregnancy that includes data on infant outcomes such as gestational age and size at birth; the infants of mothers with SLE had significantly increased risks for growth restriction, preterm birth and perinatal mortality. We report second birth outcomes for women with SLE, and the outlook for a baby surviving to discharge home is generally favourable. However, women who had only one birth were more likely to have had renal impairment than the women who went on to have a second birth, which may indicate differential severity and selective fertility.

The rate of perinatal death in first births (4.8% in the cohort of 313 women) and in the subsequent pregnancies (<1%) in our study are lower than the 6.1% perinatal death rate for SLE pregnancies calculated in a recent meta-analysis.2 A majority of the births in that metaanalysis occurred in previous decades and our results may partly reflect improvements in the management of SLE, and of neonates generally, for our recent cohort. Only 11% of the second pregnancies in our study resulted in a spontaneous abortion, compared with 16.0% in that same meta-analysis. In the Johns Hopkins first trimester cohort study, 9% of the pregnancies resulted in spontaneous abortion, of which 5% were before 10 weeks.16 Coding for spontaneous abortions was limited to before and after 14 weeks in our study, so we cannot make a direct comparison by gestation. Our results are consistent with a high rate of early spontaneous abortion, as all but one of the spontaneous abortions assigned a gestational range occurred before 14 weeks. The women in our study had similar rates of pregnancy hypertension and stroke to women in the large US study of over 13 000 hospitalisations which used both SLE and pregnancy-related discharge codes, but we found higher rates of thromboembolic events and obstetric haemorrhage.17 However, our event counts included all those that occurred in antenatal admissions and admissions up to 6 weeks postpartum, not just admissions coded as pregnancy related.17

In women with SLE, stillbirth or neonatal morbidity may be caused by complete atrioventricular heart block, secondary to autoantibodies to SSA/Ro or SSB/La. The rate of congenital heart block in the infants of women with autoantibodies is reported to be 1–5%.18 There were three infants with neonatal heart block within the entire study cohort which is a rate of 0.6%; however, the rate or level of autoantibodies in the study population is not known. The optimal screening and or treatment strategy for women with autoantibodies to SSA/Ro or SSB/La is also not known, and although some authors recommend weekly fetal echocardiography ultrasound surveillance, with treatment with steroids for those with a prolonged PR interval, the screening and treatment strategy in NSW during the time period is also not known.19

Some studies report that women with SLE have lower live birth rates and lower pregnancy rates than women without SLE.20 21 Women with SLE in our population were more likely to have used assisted reproduction methods (RR=1.42) but the actual proportion was not large (4.8% vs 3.4% in women without SLE). Our study does not capture women with SLE who never carried a pregnancy to \geq 20 weeks' gestation. It is not known whether a lower pregnancy rate among women with SLE might be related to adverse pregnancy outcomes, infertility, spontaneous abortion, personal relationship problems and/or a deliberate decision to limit family size, possibly because of the women's quality of life due to the disease. The rate of second pregnancy within the study period was 69%, comparable to that of the general population.

The recurrence analysis was limited to women with first birth and second pregnancy, in order to have a homogeneous study population.22 For some women, their first recorded diagnosis of SLE was after their first birth, rather than before. Poor fetal outcomes have been seen in pregnancies complicated by SLE before the clinical diagnosis of the disease, as was found in this study.23 For those attempting a second pregnancy, renal impairment was common, obstetric haemorrhage was higher than in the general maternity population and thromboembolic events and perinatal death did not reoccur, although numbers were small.

The strength of this study is that it is a recent, large, population- based study in which perinatal factors including perinatal mortality, gestational age and birth weight are reliably reported. 8 9 14 15 Data were collected as part of a population surveillance system, and thus the potential for bias is reduced, as all childbearing women are included, regardless of outcome. The linkage allowed us to track longitudinally women rather than simply tabulate admissions, which can result in multiple counting of those who have many hospital admissions. Furthermore, validation studies find SLE to be reliably identified in hospital data7–10 and the SLE prevalence in our study of 127 per 100 000 childbearing women is consistent with expectation.1 Of note, however, is that the timing of the SLE diagnosis is based on the first hospital admission when the SLE diagnosis was recorded. One of the limitations in this study is the lack of detailed clinical data, including treatment information. A prospective cohort study presented in abstract form, of 333 women with SLE in pregnancy, found that the following baseline variables were associated with poor pregnancy outcome: higher SLE disease activity index, high titre antiphospholipid antibody and higher median uric acid levels and an increase over baseline of the SLE disease activity index.24 Other studies have found that women with lupus nephritis have worse pregnancy outcomes.2 25 We could not determine the degree of underlying renal impairment or hypertension, or SLE disease activity or the presence of antiphospholipid antibodies.

Spontaneous abortion rates may be underestimated in our study, as only spontaneous abortions that resulted in hospitalisation can be identified. Additionally, women who never carried a pregnancy to 20 weeks or more were not included in our data. The study's focus is on the prospects for pregnancy in women with SLE after a first birth; women with more severe forms of SLE may never achieve a first birth of \geq 20 weeks' gestation.

CONCLUSIONS

In this recent cohort, 69% of women with SLE with at least 5 years' follow-up went on to have a second pregnancy. In second pregnancies to women diagnosed with SLE, 89% resulted in infants being discharged home alive, with no recurrence of

REFERENCES

1. Danchenko N, Satia JA, Anthony MS. Epidemiology of systemic lupus erythematosus: a comparison of worldwide disease burden. *Lupus* 2006;15: 308–18.

2. Smyth A, Oliveira GH, Lahr BD, *et al.* A systematic review and meta-analysis of pregnancy outcomes in patients with systemic lupus erythematosus and lupus nephritis. *Clin J Am Soc Nephrol* 2010;5:2060–8.

3. Clowse ME, Magder LS, Witter F, *et al.* The impact of increased lupus activity on obstetric outcomes. *Arthritis Rheum* 2005;52:514–21.

4. Chakravarty EF, Nelson L, Krishnan E. Obstetric hospitalizations in the United States for women with systemic lupus erythematosus and rheumatoid arthritis. *Arthritis Rheum* 2006;54:899–907.

5. Clark CA, Spitzer KA, Laskin CA. Decrease in pregnancy loss rates in patients with systemic lupus erythematosus over a 40-year period. *J Rheumatol* 2005;32: 1709–12.

6. Centre for Health Record Linkage. CHeReL Quality Assurance Procedures for Record Linkage: http://www.cherel.org.au/downloads.html (accessed March 2011).

7. Chibnik LB, Massarotti EM, Costenbader KH. Identifi cation and validation of lupus nephritis cases using administrative data. *Lupus* 2010;19:741–3.

8. Hadfi eld RM, Lain SJ, Cameron CA, *et al.* The prevalence of maternal medical conditions during pregnancy and a validation of their reporting in hospital discharge data. *Aust N Z J Obstet Gynaecol* 2008;48:78–82.

Taylor LK, Travis S, Pym M, et al. How useful are hospital morbidity data for monitoring conditions occurring in the perinatal period? Aust NZJ Obstet Gynaecol 2005;45:36–41.
 Henderson T, Shepheard J, Sundararajan V. Quality of diagnosis and procedure coding in ICD-10 administrative data. Med Care 2006;44:1011–9.

11. Roberts CL, Lancaster PA. Australian national birthweight percentiles by gestational age. *Med J Aust* 1999;170:114–8.

12. Lain SJ, Roberts CL, Hadfi eld RM, *et al.* How accurate is the reporting of obstetric haemorrhage in hospital discharge data? A validation study. *Aust N Z J Obstet Gynaecol* 2008;48:481–4.

Roberts CL, Bell JC, Ford JB, *et al.* The accuracy of reporting of the hypertensive disorders of pregnancy in population health data. *Hypertens Pregnancy* 2008;27:285–97.
 Roberts CL, Bell JC, Ford JB, *et al.* Monitoring the quality of maternity care: how well are labour and delivery events reported in population health data? *Paediatr Perinat Epidemiol* 2009;23:144–52.

15. Taylor LK, Pym M, Bajuk B, *et al.* Validation study: NSW midwives data collection 1998. *NSW Mothers and Babies* 1998 2000;9 (S-2):97–9.

16. Clowse ME, Magder LS, Witter F, *et al.* Early risk factors for pregnancy loss in lupus. *Obstet Gynecol* 2006;107:293–9.

17. Clowse ME, Jamison M, Myers E, *et al*. A national study of the complications of lupus in pregnancy. *Am J Obstet Gynecol* 2008;199:127.e1–6.

18. Hutter D, Silverman ED, Jaeggi ET. The benefi ts of transplacental treatment of isolated congenital complete heart block associated with maternal anti-Ro/SSA antibodies: a review. *Scand J Immunol* 2010;72:235–41.

19. Mevorach D, Elchalal U, Rein AJ. Prevention of complete heart block in children of mothers with anti-SSA/Ro and anti-SSB/La autoantibodies: detection and treatment of first-degree atrioventricular block. *Curr Opin Rheumatol* 2009;21:478–82.

20. Ekblom-Kullberg S, Kautiainen H, Alha P, *et al.* Reproductive health in women with systemic lupus erythematosus compared to population controls. *Scand J Rheumatol* 2009;38:375–80.

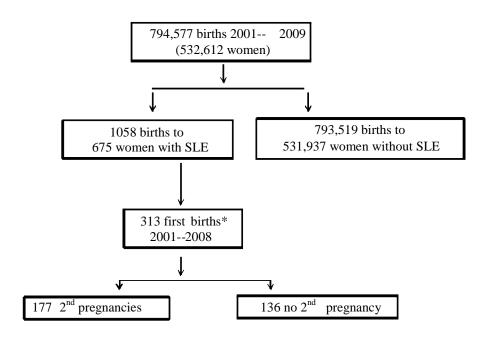
21. Vinet E, Labrecque J, Pineau CA, *et al.* A population-based assessment of live births in women with systemic lupus erythematosus. *Ann Rheum Dis* 2012;71:557–9.

22. Ford JB, Roberts CL, Bell JC, *et al.* Postpartum haemorrhage occurrence and recurrence: a population-based study. *Med J Aust* 2007;187:391–3.

23. Dhar JP, Essenmacher LM, Ager JW, *et al.* Pregnancy outcomes before and after a diagnosis of systemic lupus erythematosus. *Am J Obstet Gynecol* 2005;193:1444–55.

24. Buyon JP, Garabet L, Kim M, *et al.* Favorable prognosis in a large, prospective multicenter study of lupus pregnancies (abstract). *Arthritis Rheum* 2011;63:1707.
25. Kwok LW, Tam LS, Zhu T, *et al.* Predictors of maternal and fetal outcomes in pregnancies of patients with systemic lupus erythematosus. *Lupus* 2011;20:829–36.

Figure 1: Study population derivation



 * Among women with an SLE diagnosis recorded before their 2^{nd} birth

Table 1. Characteristics of pregnancies to mothers with and without a record of SLE (all pregnancies	
\geq 20 weeks gestation), NSW 2001-2009.	

	SLE pregnancies* n=1058 (%)	Pregnancies without SLE n=793,519 (%)	Relative risk (95% CI)
Maternal factors		(70)	
Age \geq 35 years	25.0	21.0	1.19 (1.07-1.32)
Smoking during pregnancy	10.7	14.3	0.75 (0.63-0.89)
Assisted reproduction (any record)	4.8	3.4	1.42 (1.09-1.86)
Pregnancy hypertension	19.8	8.7	2.28 (2.02-2.57)
Diabetes (gestational or pre-existing)	7.4	6.1	1.22 (0.98-1.51)
Renal impairment	13.2	2.8	4.64 (3.97-5.41)
One or more antenatal admissions	38.8	21.5	1.81 (1.67-1.95)
Pregnancy outcomes			
Twins, other multiple pregnancy	1.3	1.6	0.84 (0.50-1.42)
Preterm (<37 weeks)			
33-36 weeks	14.7	5.0	2.92 (2.51-3.40)
<33 weeks	8.5	1.7	5.03 (4.07-6.23)
Onset of labour			
spontaneous	33.7	59.0	1.00 (referent)
induced	37.6	25.1	1.77 (1.65-1.89)
prelabour caesarean	28.6	15.8	2.17 (2.00-2.36)
Any caesarean birth	42.3	27.6	1.53 (1.43-1.64)
Delivery at tertiary referral hospital	48.2	27.2	1.77 (1.66-1.89)
Antepartum haemorrhage	6.6	3.7	1.78 (1.42-2.23)
Postpartum haemorrhage	9.4	7.6	1.23 (1.02-1.49)
Any Thromboembolic event‡	1.51	0.17	9.11 (5.19-14.9)
Pulmonary embolism			
antepartum	0.47	0.04	13.2 (5.45-31.8)
puerperal†	0.76	0.06	13.5 (6.72-27.1)
Cerebral event	0.28	0.01	30.8 (9.73-97.6)
Puerperal† deep vein thrombosis	0.47	0.08	6.08 (2.53-14.6)
Transfusion of blood product	3.9	1.3	3.03 (2.24-4.09)
Infant outcomes			
Small for gestational age (<3 rd percentile)	6.4	4.0	1.60 (1.27-2.02)
Perinatal mortality	3.0	0.8	3.59 (2.55-5.06)
5-minute Apgar <7	4.7	2.0	2.36 (1.80-3.10)
Heart block	0.38	0.01	37.5 (13.8-102)

* Includes diagnosis after second or later birth
 † Up to 6 weeks postpartum
 ‡ pulmonary embolism, cerebral event (cerebral ischemia or infarction) or puerperal DVT

Characteristics of first pregnancy ≥ 20 weeks gestation	Women 1 st and 2 nd pregnancies n=171 n (%)	Women with only 1 st pregnancies n=145 n (%)
Maternal factors and outcomes		
Maternal age <30 years 30-34 years ≥ 35 years	99 (57.9) 52 (30.4) 20 (11.6)	68 (46.9) 37 (25.5) 40 (27.6)*
Smoking during pregnancy Antenatal admissions	11 (6.4)	13 (9.0)
one two or more	38 (22.2) 25 (14.6)	39 (26.9) 22 (15.2)
In vitro fertilisation	10 (5.8)	16 (11.0)
Twin pregnancy	3† (1.8)	4 (2.8)
Chronic hypertension	3 (1.8)	7 (4.8)
Gestational hypertension	35 (20.5)	42 (29.0)
Preeclampsia	15 (8.8)	24 (16.6)*
Pregnancy renal impairment††	19 (11.1)	35 (24.1)*
Diabetes	10 (5.9)	9 (6.2)
Lupus anticoagulant	7 (4.1)	8 (5.5)
Antepartum/postpartum haemorrhage	17 (9.9)	27 (18.6)*
Thromboembolic event‡	1 (0.6)	4 (2.8)
Mother transferred to ICU	2 (1.2)	2 (1.4)
Infant outcomes		
Gestational age at birth <34 weeks 34-36 weeks ≥37 weeks	20 (11.6) 20 (11.6) 131 (76.6)	16 (11.0) 24 (16.6) 105 (72.4)
Spontaneous preterm birth	14 (8.2)	11 (7.6)
Birthweight (grams: median, IQR)	3110 (2700-3480)	2980 (2410-3420)
SGA <3rd percentile	14 (8.2)	18 (12.5)
Perinatal death	11 (6.4)	5 (3)

Table 2. Maternal and infant characteristics of women with SLE and a first pregnancy≥ 20 weeks gestation 2001-2008

* P<0.05 for chi-squared test of comparison between women with second pregnancies and women without

one twin pregnancy resulted in the neonatal death of one twin
preeclampsia, admission with glomerular or tubulo-interstitial disorder, or renal failure

SGA Small for gestational age

‡ pulmonary embolism, puerperal DVT, cerebral event

		Next pregnancy outcome			
First pregnancy (of ≥ 20 weeks) infant outcome	Miscarriage n= 9 n (row %)	Perinatal death (n=1) n (row %)	Baby discharged home (n=161) n (row %)		
Stillbirth (n=8)	1 (12)	0 (0)	7 (88)		
Neonatal death (n=3)	0 (0)	0 (0)	3 (100)		
Baby discharged home (n=160)	8 (5)	1 (1)	151 (94)		
Gestation <32 weeks (n=16)	2 (12)	0 (0)	14 (88)		
SGA < 3 rd percentile (n=14)	0 (0)	1 (7)	13 (93)		

Table 3. Infant outcomes of the first pregnancy compared to the second pregnancy among 171 women with a diagnosis of SLE.

SGA Small for gestational age

	Adverse event at next birth?	
Maternal adverse events in first	Yes	No
pregnancy (of ≥ 20 weeks)	n (row %)	n (row %)
Pregnancy renal impairment†		
Yes (n=19)	9 (47)	10 (53)
No (n=152)	10 (7)	142 (94)
Antepartum/postpartum		
haemorrhage		
Yes (n=17)	1 (6)	16 (94)
No (n=164)	16 (10)	138 (94)
Transfusion		
Yes (n=4)	0 (0)	4 (100)
No (n=167)	6 (4)	161 (96)
Thromboembolic event‡		
Yes (n=1)	0 (0)	1 (100
No (n=170)	2 (1)	169 (99)

Table 4. Maternal adverse events among 171 women with a subsequent pregnancy

† defined as preclampsia, admission with glomerular or tubulo-interstitial disorder, or renal failure
 ‡ pulmonary embolism, puerperal DVT, cerebral event