

HHS Public Access

Author manuscript *J Perinat Med.* Author manuscript; available in PMC 2018 January 01.

Published in final edited form as:

J Perinat Med. 2017 January 01; 45(1): 63-70. doi:10.1515/jpm-2016-0207.

Risk of recurrent preterm birth among women according to change in partner

Rebecca J. Baer, MPH¹, Juan Yang, PhD², Christina D. Chambers, PhD¹, Kelli K. Ryckman, PhD³, Audrey F. Saftlas, PhD³, Vincenzo Berghella, MD⁴, Chris Dunkel Schetter, PhD⁵, Gary M. Shaw, DrPH⁶, David K. Stevenson, MD⁶, and Laura L. Jelliffe-Pawlowski, PhD⁷ ¹Department of Pediatrics, University of California San Diego, La Jolla, CA

Department of rediatines, oniversity of Gamornia Gan Diego, La Jona, Orr

²Genetic Disease Screening Program, California Department of Public Health, Richmond, CA

³Department of Epidemiology, University of Iowa, Iowa City, IA

⁴Department of Obstetrics and Gynecology, Sidney Kimmel Medical Center of Thomas Jefferson University, Philadelphia, PA

⁵Department of Psychology, University of California Los Angeles, Los Angeles, CA

⁶Department of Pediatrics, Stanford University, Stanford, CA

⁷Department of Epidemiology and Biostatistics, University of California San Francisco School of Medicine, San Francisco, CA

Abstract

There is a well-established literature indicating change in partner as a risk for preeclampsia, yet the research on the risk of preterm birth after a change in partners has been sparse and inconsistent. Using a population of California live born singletons, we aimed to determine the risk of preterm birth after a change in partner between the first and second pregnancy. Risk of preterm and early term delivery in the second pregnancy was calculated for mothers who did or did not change partners between births with the referent group as women who delivered both pregnancies at term and no change in partners. Adjusted odds ratios and their 95% confidence intervals were calculated. Relative to women who delivered at 39 weeks or later in the second pregnancy and did not change partners, preterm birth risks were somewhat lower for women who changed partners between first and second pregnancy compared to those women with same partner. For example, 10.6% of women who did not change partners and delivered their second pregnancy before 34 weeks also delivered their first pregnancy before 34 weeks, while 8.5% of women who changed partners did. Findings suggest partner change may alter the risk of preterm birth.

Keywords

adverse pregnancy outcome; change in partner; early term birth; interpregnancy interval; multipartner fertility; preterm birth; recurrent preterm birth

CORRESPONDING AUTHOR: Rebecca J. Baer, Department of Pediatrics, University of California San Diego, 9500 Gilman Drive, # 0828, La Jolla, CA, 92093, Telephone: (206) 351-0850; rjbaer@ucsd.edu.

CONFLICT OF INTEREST: The authors report no conflicts of interest or personal financial disclosures.

Introduction

Nearly one in five women between the ages of 41 and 49 in the United States have children with more than one partner. This percentage is higher among black and Hispanic women.(1, 2)

Partner change has been studied as a risk factor for adverse pregnancy outcomes such as preeclampsia, preterm birth, and birth defects.(3–10) There is a well-established literature indicating change in partners as a risk factor for preeclampsia,(3–7) while the research on the risk of preterm birth after a change in partners has been sparse and findings have been inconsistent.(3, 5, 8, 9) Studying pregnancy outcomes for women who change partners is complex, due to the many potential confounding factors that distinguish women who change partners from those who do not. Notable factors include maternal age, race/ethnicity, psychosocial and sociodemographic influences, interpregnancy interval and underlying medical conditions.(11, 12)

Low immunologic tolerance to new paternal antigens has been suggested as a mechanism to explain an association of partner change with increased risk of adverse pregnancy outcomes. Specifically, paternal antigen exposure is posited to facilitate maternal immune adaption during extended periods of sexual cohabitation.(13) Recent work demonstrating an increased risk of preeclampsia among women with low cumulative exposure to paternal seminal fluid supports such an immune-based mechanism.(14, 15) Additionally, paternal human leukocyte antigen (HLA) sharing has been suggested to have an influence on risk of preterm birth.(8, 15) Other mechanisms may be psychosocial such as increased level of stress, reduced support, or change in financial status or social capital.(3)

In this study, we analyzed the association of change in partner on the subsequent risk of preterm birth in a large sample of California births. We considered and adjusted for confounding factors such as race/ethnicity, maternal age, smoking status, maternal education, payment for delivery, hypertension, diabetes and interpregnancy interval and examined risk of preterm birth by gestational age at previous birth.

Materials and methods

In this retrospective cohort study, the sample included mothers who delivered their first two singleton live births in California from 2005 through 2011. All women were nulligravida in their first delivery and did not report a termination or fetal demise between pregnancies. Pregnancies after the first two live births, even in the same study period, were not considered in these analyses. The sample contained linked baby birth certificate and maternal and baby hospital discharge records available through the California Office of Statewide Health Planning and Development. Sample selection proceeded by first identifying women based on vital statistics birth certificate files, using linkage algorithms that leveraged identifiers and other data including first and last name (married or maiden), date and place of maternal birth, address, phone number, and reported month and year of birth of the first pregnancy as reported in the second. The sample was restricted to both pregnancies delivering between 20 to 44 weeks gestation and excluded women with missing information for name or birth date

of infant's father, length of interpregnancy interval, or who had an infant with a major malformation. (Figure 1).

To determine the risk of preterm birth after a partner change between a first and second pregnancy, we began by subgrouping the women by gestation at birth in the second pregnancy (< 34, 34–36, 37–38, and 39 weeks). We then analyzed the risk of short gestation length of the second pregnancy (<34, 34–36, and 37–38 weeks) in association with partner change with gestational length in the first pregnancy as an effect modifier (p < 0.05 based on test for heterogeneity). Length of gestation in the first pregnancy was divided into these same four categories of gestation as the length of gestation in their second pregnancy. Adjusted logistic regression models were used to examine the relationship between a change in partners between pregnancies and short gestational age in the second pregnancy (<34, 34–36, 37–38 weeks) among women who had gestational age at < 34, 34–36, and 37–38 weeks versus 39 weeks in the first pregnancy by calculating odds ratios (ORs) and their 95% confidence intervals (CIs).

Factors considered as potential covariates included maternal characteristics and obstetric factors derived from birth certificate and hospital discharge records including race/ethnicity, maternal age, smoking status, maternal education, Medi-Cal payment for the delivery (California's low income health insurance), hypertension during pregnancy, and diabetes. Smoking was coded as yes if reported on the birth certificate or the hospital discharge record. Coding for hypertension (any, preexisting with and without preeclampsia, gestational with and without preeclampsia), diabetes (any, preexisting, and gestational), was based on the International Classification of Disease, 9th Revision, Clinical Modification (ICD-9-CM) (16) four digit codes contained in the hospital discharge files. Interpregnancy interval was estimated as months from the birthdate of first pregnancy to conception of second pregnancy (calculated by birthdate and gestational length of second pregnancy).

Partner change was determined by comparing the identifiers for the father (name and date of birth) on the birth certificate in the first pregnancy with those in the second. We compared maternal characteristics and obstetric factors among women who had the same partner for their first and second pregnancies versus women who changed partners between their first and second pregnancies using a chi-square analysis. Factors that were significantly (p < 0.05) different between women who changed partners and those who did not were adjusted for all preterm birth risk calculations. The referent group for all comparisons was women who delivered both infants at 39 weeks and did not change fathers between pregnancies.

All analyses were performed using Statistical Analysis Software (SAS) version 9.3 (Cary, NC) and were based on data received by the Genetic Disease Screening Program at the California Department of Public Health as of February 1st, 2015. Methods and protocols for the study were approved by the Committee for the Protection of Human Subjects within the Health and Human Services Agency of the State of California.

Results

Of the 186,724 mothers included in the cohort, 7,192 (3.9%) had a change in partner between first and second pregnancy. Women who had births associated with change in partner between their first and second pregnancy differed from the women who did not change partners: 64.7% of women who changed partners were Hispanic, compared with 43.9% of women who did not change partners. Similarly, 5.5% of women who changed partners and 1.8% of those who did not change partners were black. Women who changed partners were younger, more likely to smoke, were less educated and more likely to pay for both deliveries using Medi-Cal (Table 1).

Significantly more women who changed partners between their first and second pregnancy developed gestational hypertension (with or without preeclampsia) and fewer developed gestational diabetes in either pregnancy. However, there was no significant difference in preexisting hypertension (with or without preeclampsia) or preexisting diabetes between women who did or did not change partners between pregnancies. Interpregnancy intervals for women who changed partners were longer than for women who did not (Table 2).

Relative to women who delivered at 39 weeks in the second pregnancy and did not change partners, preterm birth risks were somewhat lower for women who changed partners between first and second pregnancy compared to those women with same partner. Compared with women who delivered both infants at 39 weeks and did not change partners between pregnancies, women who delivered <34 weeks in their first pregnancy were more likely to deliver <34 weeks in their second pregnancy: the adjusted odds were 16.9-fold (95% CI 13.9 to 20.5) higher for women who did not change partners and 6.5-fold (95% CI 2.5 to 15.7) higher for women who changed partners between pregnancies. For the women who delivered 34–36 weeks for their first pregnancy, those who did not change partners were at an adjusted 6.0-fold (95% CI 5.2 to 7.1) higher risk of delivering < 34 weeks in their second pregnancy and women who changed partners were at an adjusted 3.4-fold higher risk (95% CI 1.7 to 6.7). After adjustment for confounders, women who delivered between 37 and 38 weeks for their first pregnancy and did not change partners were 2.3-times (95% CI 2.0 to 2.6) more likely to deliver before 34 weeks in their second pregnancy while women who changed partners were not at a statistically significant increased risk (aOR 1.3, 95% CI 0.8 to 2.3). Women who gave birth before 39 weeks in their first pregnancy and changed partners for their second pregnancy were at 1.2 to 1.7-fold more likely to deliver at 39 weeks or later than women who did not change partners (Table 3).

Discussion

Women in the population who changed partners were, in general, at lower risk of delivering before 39 weeks than women who did not change partners. Additionally, women who gave birth to a preterm infant in their first pregnancy and changed partners were 1.2 to 1.6-fold more likely to deliver a baby at 39 weeks or later compared with women who did not change partners.

This study represents one of the largest and most detailed examinations of the potential risks of change in partner on preterm birth and to our knowledge is the first to find a decreased risk of recurrent preterm birth among women who changed partners.(8) However, our study's findings must be interpreted while being mindful of its limitations. The study's administrative database did not allow for verification of paternity, but instead relied on the name and date of birth of the father listed on the birth certificate. Therefore, there are likely occurrences of inadvertent misclassification regarding partner change between pregnancies. Furthermore, there are some variables we would have liked to control for, had we been able to obtain the information, such as length of cohabitation with partners. The premise of the theory of immunologic tolerance suggests that the longer a woman is sexually cohabiting with her partner, the more likely she is to have tolerance to his antigens. Without these data, we are unable to further explore how this theory may have related to our observed results. Similarly, we did not have data on certain psychosocial or socioeconomic variables potentially related to life changes involving change in partner as either mediators or covariates, such as changes in economic status, marital status and social support. This information is important for considering social and demographic reasons for why change in partner may decrease risk of preterm birth. Four percent of the women in our study had a change of partner, while other studies report 9% to 19%.(1-3) It is likely that our observed percentages were lower than other studies due to our restriction to first and second pregnancies during a shorter seven-year time period.

We did not distinguish between spontaneous and iatrogenic preterm births in this study. Women who change partners have been shown to be more likely to develop preeclampsia and preeclampsia which increases the occurrence of iatrogenic preterm birth.(3, 5, 6) For this reason alone, one might expect change in partner to be a risk factor for preterm birth. In fact, in a large Norwegian population, Vatten and Skjaerven(5) demonstrated women who changed partners were at increased risk of preterm birth after adjusting for maternal age, birth interval and year of birth. However, our findings are consistent with other studies that have shown no effect or a decreasing effect.(8, 9) This finding is particularly important as it suggests that the partner change may be related primarily to reduced risk of spontaneous rather than iatrogenic preterm birth.

The consideration of interpregnancy interval in this type of analysis is complex. Women who change partners between pregnancies tend to have longer interpregnancy intervals, and many studies adjust for this interval in their risk analyses.(5, 8) However, since interpregnancy interval can result from two or more variables (e.g., partner change and subfertility), others have suggested stratifying or controlling for it might introduce bias into the analysis.(11, 12) To adequately consider interpregnancy interval in our analyses, we calculated our risks by either adjusting for interpregnancy interval or removing it from the model. We also stratified our analyses by interpregnancy interval (not tabled). In each of these analyses, our findings were quite similar. Women who changed partners between their first and second pregnancy were at somewhat lower risk of delivering before 39 weeks than women who did not change partners – regardless of how interpregnancy interval was considered analytically.

Our findings present a challenge to derive a pathophysiological explanation, considering that observed results were opposite of what we may have expected *a priori* based on other

outcomes of pregnancy. The causes of preterm birth have yet to be identified, however they are thought to be complex and multifactorial.(17, 18) One well described risk factor is a previous preterm birth. After adjusting for maternal factors, women who changed partners between their first and second pregnancy were at somewhat lower risk of delivering before 39 weeks than women who did not change partners. This supports the human leukocyte antigen (HLA) sharing theory which suggests that sufficient dissimilarity in HLA genotype between the mother and the father is needed in order for the mother to develop the appropriate immune tolerance for her fetus and "non-self".(8) Specifically, if preterm birth is due in part to a similar HLA genetic profile in the woman and her partner, then a change in partner would by chance be expected to result in less HLA similarity between partners in some cases, and thus lead to a reduced risk of recurrence of preterm birth. Our findings do not support the immunologic tolerance theory of risk, i.e., change in partner introduces new paternal antigen in the context of a woman having developed immunologic tolerance to the previous partner's antigens. However, as noted, we were not able to address the length of sexual cohabitation after a change in partner or the use of barrier methods of contraception. These factors relate to length of a woman's exposure to the new partner's novel paternal antigen and therefore the length of time a woman has to develop immunologic tolerance.

Although several studies have suggested the genetic risk of preterm birth lies with the mother,(19) our findings suggest the need to further investigate a possible paternal role in preterm birth. One such investigation was made by Alio and colleagues,(20) who showed advanced paternal age (> 45 years) as a risk factor for preterm birth. Because our population of women who changed partners differed in many characteristics from those who had the same partner, it is difficult to determine the paternal role in the risk of preterm birth in this population.

Further, ongoing study into the psychosocial and sociodemographic influences on preterm birth also deserves attention. Our population of women who changed partners were more likely to be Hispanic or black, were younger, more likely to smoke, less educated and more likely to pay for both deliveries using Medi-Cal. Psychosocial factors such as reduced stress or increased support, or socioeconomic factors such as improved financial status or social capital may have influenced the modified risk we demonstrated after partner change.

Further study into the effect of change in partner on preterm birth is warranted. Studies should be undertaken where more information is available on length of sexual cohabitation, marital status, psychosocial, and socioeconomic data. Further work should investigate the risk of spontaneous and iatrogenic preterm birth separately. Additionally, more research into the genetic and immune influences of change in partner and preterm birth is needed.

Acknowledgments

An abstract of these findings was presented as a poster at the 36th Annual Pregnancy Meeting of the Society of Maternal Fetal Medicine, February 1 – 4, 2016 in Atlanta, Georgia. This study was supported, in part, by funding from the Preterm Birth Initiative - California, University of California San Francisco School of Medicine; March of Dimes Prematurity Research Center at Stanford University; the Stanford Child Health Research Institute and the Stanford Clinical and Translational Science Award (CTSA) to Spectrum (UL1 TR001085). The CTSA program is led by the National Center for Advancing Translational Sciences (NCATS) at the National Institutes of Health (NIH). Also supported in part by the March of Dimes Prematurity Research Center Ohio Collaborative, the Bill and Melinda Gates Foundation, and The Eunice Kennedy Shriver National Institute of Child Health and Development

(R00-HD65786). The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIH.

References

- Guzzo KB. New Partners, More Kids: Multiple-Partner Fertility in the United States. The Annals of the American Academy of Political and Social Science. 2014; 654(1):66–86. [PubMed: 25284822]
- 2. Dorius, C. Multipartnered fertility at midlife. National Center for Family and Marriage Research Counting Couples, Counting Families Conference; 19–20 July; Bethesda, MD. 2011.
- Bandoli G, Lindsay S, Johnson DL, Kao K, Luo Y, Chambers CD. Change in paternity and select perinatal outcomes: causal or confounded? Journal of obstetrics and gynaecology : the journal of the Institute of Obstetrics and Gynaecology. 2012; 32(7):657–62. [PubMed: 22943712]
- Saftlas AF, Levine RJ, Klebanoff MA, Martz KL, Ewell MG, Morris CD, et al. Abortion, changed paternity, and risk of preeclampsia in nulliparous women. American journal of epidemiology. 2003; 157(12):1108–14. [PubMed: 12796047]
- Vatten LJ, Skjaerven R. Change of partner between births and adverse pregnancy outcomes. Tidsskrift for den Norske laegeforening : tidsskrift for praktisk medicin, ny raekke. 2003; 123(24): 3546–8.
- Krulewitch CJ, Herman AA, Yu KF, Johnson YR. Does changing paternity contribute to the risk of intrauterine growth retardation? Paediatr Perinat Epidemiol. 1997; 11(Suppl 1):41–7. [PubMed: 9018714]
- Li DK, Wi S. Changing paternity and the risk of preeclampsia/eclampsia in the subsequent pregnancy. American journal of epidemiology. 2000; 151(1):57–62. [PubMed: 10625174]
- Li DK. Changing paternity and the risk of preterm delivery in the subsequent pregnancy. Epidemiology. 1999; 10(2):148–52. [PubMed: 10069250]
- Basso O, Olsen J, Christensen K. Study of environmental, social, and paternal factors in preterm delivery using sibs and half sibs. A population-based study in Denmark. Journal of epidemiology and community health. 1999; 53(1):20–3. [PubMed: 10326048]
- Chambers CD, Chen BH, Kalla K, Jernigan L, Jones KL. Novel risk factor in gastroschisis: change of paternity. American journal of medical genetics Part A. 2007; 143A(7):653–9. [PubMed: 17163540]
- 11. Basso O, Christensen K, Olsen J. Higher risk of pre-eclampsia after change of partner. An effect of longer interpregnancy intervals? Epidemiology. 2001; 12(6):624–9. [PubMed: 11679788]
- Zhang J, Patel G. Partner change and perinatal outcomes: a systematic review. Paediatr Perinat Epidemiol. 2007; 21(Suppl 1):46–57. [PubMed: 17593197]
- Robillard PY, Dekker G, Chaouat G, Hulsey TC, Saftlas A. Epidemiological studies on primipaternity and immunology in preeclampsia--a statement after twelve years of workshops. Journal of reproductive immunology. 2011; 89(2):104–17. [PubMed: 21543120]
- Saftlas AF, Rubenstein L, Prater K, Harland KK, Field E, Triche EW. Cumulative exposure to paternal seminal fluid prior to conception and subsequent risk of preeclampsia. Journal of reproductive immunology. 2014; 101–102:104–10.
- 15. Triche EW, Harland KK, Field EH, Rubenstein LM, Saftlas AF. Maternal-fetal HLA sharing and preeclampsia: variation in effects by seminal fluid exposure in a case-control study of nulliparous women in Iowa. Journal of reproductive immunology. 2014; 101–102:111–9.
- Association AM. International Classification of Diseases: ICD-9-CM 2008. 2008. Vol. 2007. Chicago, IL: American Medical Association; 2007.
- Romero R, Dey SK, Fisher SJ. Preterm labor: one syndrome, many causes. Science. 2014; 345(6198):760–5. [PubMed: 25124429]
- Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. Lancet. 2008; 371(9606):75–84. [PubMed: 18177778]
- Boyd HA, Poulsen G, Wohlfahrt J, Murray JC, Feenstra B, Melbye M. Maternal contributions to preterm delivery. Am J Epidemiol. 2009; 170(11):1358–64. [PubMed: 19854807]
- 20. Alio AP, Salihu HM, McIntosh C, August EM, Weldeselasse H, Sanchez E, et al. The effect of paternal age on fetal birth outcomes. Am J Mens Health. 2012; 6(5):427–35. [PubMed: 22564913]

Singleton live b	irth cohort, 2005-2011, California N=3,767,337
	Women with two or more singletons
	N=700,634 women with 1,247,434 births
	Linked to be arital discharge data
	N=489,787 women with 1,036,587 births
	Nulligravida and nullipara at the first programov and primigravid and
	primipara at the second pregnancy N=234 020 women and 234 020 sibling pairs
	Gestational age 20 to 44 weeks in both pregnancies
	N=221,776 women and 221,776 sibling pairs
	Infant had no major malformations
	N=213,212 women and 213,212 sibling pairs
	Not missing paternity or interpregnancy interval information N=186,724 women and 186,724 sibling pairs

Figure 1. Sample selection

Table 1

Maternal characteristics by change in partner

	Changed	partner
	No	Yes
Maternal characteristic	n (%)	n (%)
Sample	179,532	7,192
Race/ethnicity ¹		
American Indian/Alaska Native	509 (0.3)	32 (0.4)
Asian	32,637 (18.5)	511 (7.2)
Black	3,095 (1.8)	394 (5.5)
Hawaiian/Pacific Islander	616 (0.3)	34 (0.5)
Hispanic	77,526 (43.9)	4,617 (64.7)
Other	1,361 (0.8)	64 (0.9)
Two or more	3,677 (2.1)	224 (3.1)
White, not Hispanic	57,262 (32.4)	1,261 (17.7)
Mother's age (years) ¹		
<18, both pregnancies	1,110 (0.6)	107 (1.5)
<18 1st pregnancy, > 18 – 34 2nd pregnancy	6,560 (3.7)	1,375 (19.1)
18 – 34, both pregnancies	140,355 (78.2)	5,350 (74.4)
18 – 34 1st pregnancy, > 34 2nd pregnancy	17,970 (10.0)	240 (3.3)
Smoked during pregnancy ¹		
Neither pregnancy	175,102 (97.5)	6,779 (94.3)
1 st pregnancy, not 2 nd	1,727 (1.0)	118 (1.6)
2 nd pregnancy, not 1 st	2,264 (1.3)	251 (3.5)
Both pregnancies	439 (0.2)	44 (0.6)
Maternal education ¹		
< 12 years, both pregnancies	25,887 (16.3)	1,823 (29.3)
< 12 years 1 st pregnancy, 12 years 2 nd	7,950 (5.0)	931 (15.0)
< 12 years 1 st pregnancy, > 12 years 2 nd	1,725 (1.1)	257 (4.1)
12 years, both pregnancies	21,009 (13.2)	1,240 (19.9)
12 years 1^{st} pregnancy, > 12 years 2^{nd}	7,338 (4.6)	591 (9.5)
> 12 years, both pregnancies	95,007 (59.8)	1,376 (22.1)
Medi-Cal payment for delivery ¹		
Neither pregnancy	106,010 (59.2)	1,679 (23.5)
1 st pregnancy, not 2 nd	10,050 (5.6)	760 (10.6)
2^{nd} pregnancy, not 1^{st}	9,278 (5.2)	858 (12.0)
Both pregnancies	53 740 (30 0)	3 860 (53 9)

¹Indicates p < 0.05 by chi-square

Table 2

Obstetric factors by change in partner

	Changed	Partner
	No	Yes
	n (%)	n (%)
Sample	179,532	7,192
Hypertension disorder		
No hypertension disorder in either pregnancy ¹	164,360 (91.5)	6,482 (90.1)
Pre-existing hypertension without preeclampsia		
Neither pregnancy	164,360 (99.4)	6,482 (99.5)
2 nd pregnancy, not 1 st	353 (0.2)	17 (0.3)
Both pregnancies	702 (0.4)	17 (0.3)
Pre-existing hypertension with preeclampsia		
Neither pregnancy	164,360 (99.9)	6,482 (99.9)
2 nd pregnancy, not 1 st	59 (0.0)	4 (0.1)
Both pregnancies	155 (0.1)	3 (0.1)
Gestational hypertension without preeclampsia		
Neither pregnancy	164,360 (97.0)	6,482 (96.3)
1 st pregnancy, not 2 nd	3,689 (2.2)	170 (2.5)
2 nd pregnancy, not 1 st	972 (0.6)	72 (1.1)
Both pregnancies	379 (0.2)	10 (0.1)
Gestational hypertension with preeclampsia ¹		
Neither pregnancy	164,360 (96.1)	6,482 (95.0)
1 st pregnancy, not 2 nd	4,987 (2.9)	247 (3.6)
2 nd pregnancy, not 1 st	1,108 (0.6)	65 (1.0)
Both pregnancies	569 (0.3)	31 (0.5)
Diabetes		
Pre-existing diabetes		
Neither pregnancy	162,492 (99.2)	6,725 (99.2)
2 nd pregnancy, not 1 st	379 (0.2)	20 (0.3)
Both pregnancies	1,011 (0.6)	34 (0.5)
Gestational diabetes ¹		
Neither pregnancy	162,492 (91.1)	6,725 (94.0)
1 st pregnancy, not 2 nd	4,302 (2.4)	115 (1.6)
2 nd pregnancy, not 1 st	7,391 (4.1)	249 (3.5)
Both pregnancies	4,235 (2.4)	69 (1.0)
Interpregnancy interval*		
< 6 months	13,258 (7.4)	304 (4.2)
6 – 12 months	34,038 (19.0)	735 (10.2)
12 – 17 months	41,939 (23.4)	990 (13.8)

-

	Changed	Partner
	No	Yes
	n (%)	n (%)
18 – 35 months	68,213 (38.0)	2,631 (36.6)
36 – 47 months	14,962 (8.3)	1,366 (19.0)
48 months^2	7,122 (4.0)	1,166 (16.2)

 1 Indicates p < 0.05 by chi-square

 2 Study population included 7 years of births

				Gestation at 2 ⁿ	^d birth (weeks)			
	Ŷ	< 34	34	- 36	37	- 38	39	- 44
	Same partner	Changed partner	Same partner	Changed partner	Same partner	Changed partner	Same partner	Changed partner
	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	u (%)	u (%)
	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)	aOR (95% CI)
Sample	1,457	82	6,646	330	50,129	1,978	121,300	4,802
Gestation at 1 st birth (week	(S							
< 34	155 (10.6)	7 (8.5)	322 (4.8)	15 (4.5)	740 (1.5)	59 (3.0)	942 (0.8)	87 (1.8)
	22.7 (18.8, 27.3)	14.8 (6.7, 32.8)	10.5 (9.2, 12.0)	8.8 (5.2, 15.0)	2.5 (2.2, 2.7)	5.0(3.8, 6.5)		2.5 (2.0, 3.1)
	16.9 (13.9, 20.5)	6.5 (2.7, 15.7)	8.9 (7.8, 10.2)	5.0 (2.9, 8.6)	2.3 (2.1, 2.6)	3.3 (2.5, 4.4)		1.7 (1.3, 2.1)
34 - 36	217 (14.9)	10 (12.2)	988 (14.9)	42 (12.7)	3,706 (7.4)	154 (7.8)	4,152 (3.4)	236 (4.9)
	7.2 (6.2, 8.4)	4.8 (2.4, 9.5)	7.3 (6.8, 7.9)	5.6 (4.0, 7.9)	2.8 (2.7, 2.9)	2.9 (2.5, 3.5)		1.5 (1.4, 1.8)
	6.0 (5.2, 7.1)	3.4 (1.7, 6.7)	6.5 (6.0, 7.1)	4.2 (3.0, 6.0)	2.7 (2.6, 2.8)	2.4 (2.0, 2.9)		1.3 (1.1, 1.5)
37 – 38	406 (27.9)	18 (22.0)	2,286 (34.4)	104 (31.5)	15,862 (31.6)	586 (29.6)	22,623 (18.7)	1,034 (21.5)
	2.5 (2.2, 2.8)	1.6 (0.9, 2.7)	3.1 (2.9, 3.3)	2.6 (2.0, 3.3)	2.2 (2.3, 2.3)	2.1 (1.9, 2.3)		1.2 (1.2, 1.3)
	2.3 (2.0, 2.6)	1.3 (0.8, 2.3)	2.9 (2.7, 3.1)	2.2 (1.8, 2.9)	2.1 (2.1, 2.2)	1.9 (1.7, 2.1)		1.2 (1.1, 1.3)
39 - 44	679 (46.6)	47 (57.3)	3,050 (45.9)	169 (51.2)	29,821 (59.5)	1,179 (59.6)	93,583 (77.2)	3,445 (71.7)
							Reference	
							Reference	

pregnancy), maternal education (12 years in both pregnancies as reference), Medi-Cal payment for delivery (neither pregnancy as reference), no hypertension disorder in either pregnancy (yes as reference), gestational hypertension without preeclampsia (either vs. neither pregnancy), gestational hypertension with preeclampsia (either vs. neither pregnancy), gestational diabetes (either vs. neither pregnancy), and interpregnancy interval (18 to 47 months as reference).

J Perinat Med. Author manuscript; available in PMC 2018 January 01.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 3