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Exposure to community homicide during pregnancy and adverse birth outcomes: A within-community matched design

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Abstract

Background: Community violence is an understudied aspect of social context that may affect risk of preterm birth and small-for-gestational age (SGA).

Methods: We matched California mothers with live singleton births who were exposed to a homicide in their Census tract of residence in 2007–2011 to unexposed mothers within the same tract. We estimated risk differences with a weighted linear probability model, with weights corresponding to the matched data structure. We estimated the average treatment effect on the treated of homicide exposure on the risk of preterm birth and SGA during the pre-conception period, and first and second trimester.

Results: We found a small increase in risk of SGA associated with homicide exposure in the first trimester (0.14% [95% CI −0.01%, 0.30%]), but not for exposure during the pre-conception period (−0.01% [95% CI −0.17%, 0.15%]) or the second trimester (−0.06% [95% CI −0.23%, 0.11%]). Risk of preterm birth was not affected by homicide exposure. When women were exposed to homicides during all three exposure windows there was a larger increase in risk of SGA (1.09% [95% CI 0.15%, 2.03%]) but not PTB (0.14% [95% CI −0.74%, 1.01%]). Exposure to three or more homicides was also associated with greater risk of SGA (0.78% [95% CI 0.15%, 1.40%]). Negative controls indicated that residual confounding by temporal patterning was unlikely.

Conclusions: Homicide exposure during early pregnancy is associated with a small increased risk of SGA.

Keywords

homicide; violence; premature birth; small for gestational age; California

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Data and code availability: The code is available in the eAppendix. The data are confidential to protect patient privacy, but interested researchers can apply for access through California's Department of Public Health. The link to the application form is provided in the eAppendix.

The authors declare no conflicts of interest.

Introduction

Community violence is an aspect of the contextual environment that has been recognized as relevant to fetal and infant health (1), but has thus far been understudied in perinatal research. The adverse effects of direct violent injury on pregnant women and their infants are well documented (2–4), but violent events in one's community may have impacts beyond those directly harmed. Pregnant women can be indirectly affected by community violence through injury of friends or family, witnessing violence, or hearing about violence through neighbors or the media. Stress due to these experiences may have implications for both maternal and fetal health (5). Exposure to community violence may also play a role in the well-documented racial/ethnic disparities in pregnancy complications, gestational weight gain (6,7), growth restriction, and preterm birth (8–11), through disparities in exposure to violence and/or differential response (e.g., greater internalization of stress) to violence (12).

Community violence may be an important mechanism through which historical patterns of segregation and economic exclusion influence health outcomes and health disparities. Segregation of American cities has played an important role in neighborhood economic development in the 20th century (13–15), and patterns of investment associated with racial and economic segregation have implications for community health today (11,16–18), especially for lower income and minority women. Black women are more likely to live in economically neglected neighborhoods (13), to be exposed to violence during pregnancy (19,20), and to have elevated risk of preterm birth compared to white women (9,10,21). In-utero exposure to community violence may also have consequences for health and economic outcomes later in life, contributing to disparities in adult health (22–26).

Exposure to community violence may affect risk of adverse birth outcomes via two mechanisms. First, there may be behavioral changes associated with exposure that increase risk factors for preterm birth and intrauterine growth restriction. Some women may respond to community violence by adopting coping behaviors or altering activities in order to mitigate exposure to violence or to alleviate the psychological burden of the experience. Health behaviors linked to violence exposure include reduced physical activity, poor diet or nutrition, use of tobacco, alcohol, or other substances, and increased risky sexual behaviors (27–29). Each of these behaviors has either been directly linked or is related to pregnancy complications (e.g., preeclampsia, gestational diabetes, or infection) associated with adverse birth outcomes (30–37).

Second, there is evidence of direct biologic stress response mechanisms associated with community violence that may impact health during pregnancy (38,39), although some recent evidence has been mixed (40). Experiencing stress due to community violence can activate the hypothalamic-pituitary-adrenal axis, which secretes corticotropin-releasing hormone that stimulates the production of glucocorticoids like cortisol. The placenta also produces corticotropin-releasing hormone, and cortisol and other biological effectors of stress are associated with increased production of the hormone by the placenta, which may be responsible for instigating the events leading to parturition (5,21,41,42). Placental corticotropin-releasing hormone is thought to act as a marker of gestational time in pregnant women, regulating the timing of parturition, as its levels increase over the course of gestation and peak before initiation of labor (43,44). Therefore, stressful events that activate the maternal HPA axis early in pregnancy may lead to increased production of placental corticotropin-releasing hormone and overstimulation of the fetal hypothalamic-pituitary-adrenal axis, leading to preterm birth.

There has been substantial research in recent years about the contribution of the community environment to disparities in adverse pregnancy outcomes (8,10,45–50). However, literature assessing the relationship between community violence and gestational health is still in its infancy (51–55). One of the main challenges in studying community violence is structural confounding (56,57). Places with high and low levels of violence tend to differ in many ways that affect health, and these factors are too correlated for statistical adjustment to adequately disentangle. In this study, we leverage temporal variability in homicide to isolate the effect of violence exposure as an acute stressor or shock that could affect pregnancy outcomes. This allows us to address the issue of structural confounding by comparing women *within* communities who conceived at different times and therefore had different exposure to violence immediately before and during pregnancy. This within-community approach controls for any measured or unmeasured community-level factors that were constant over the study period. It also addresses the related problem of neighborhood selection, in which individuals are not comparable across neighborhoods due to patterns of residential mobility that replicate income and racial/ethnic inequalities (58).

We examined whether exposure to community homicide during pregnancy is associated with increased risk of preterm birth and small-for-gestational age (SGA). Because there may be different mechanisms affecting the maternal response to homicide during different periods of pregnancy (5,21), we examined the associations during the pre-conception period, the first trimester, and the second trimester. We also examined whether exposure in all three exposure windows or to three or more homicides at any point from pre-conception through the second trimester were associated with stronger effects to assess possible dose-response.

DATA & METHODS

Data description

This study used California birth and death records from 2007 to 2011 to estimate the association of homicide exposure during pregnancy with preterm birth and SGA among singleton gestations. We estimated the date of conception using the birthdate and estimate of gestational age, and restricted our analysis to women who conceived between 1 January 2007 and 1 March 2011 to ensure women of all gestational lengths were eligible to be included in the analysis to avoid fixed cohort bias (59).

We identified homicides from the death records using ICD-10 cause of death codes U01*, U02*, X85 – Y09, Y871, Y35*. Homicide victims' addresses were geocoded from the death records and maternal addresses at delivery were geocoded from the birth records. We defined preterm delivery as delivery of a live singleton birth before 37 weeks' gestation based on the estimate of gestational age in the birth record. We determined SGA using birthweight, gestational age, and infant sex from the birth record and classified according to the sex-specific reference from Talge et al (60). We obtained covariates from the birth record and included mother's age, race/ethnicity, parity, health insurance type used for prenatal care, conception year, and conception season. Covariate categories are listed in eAppendix 1.

We assessed three main windows of exposure to homicide during pregnancy: the pre-conception period (12 weeks prior to estimated conception date), the first trimester (weeks 0–12 of pregnancy) and a restricted second trimester (weeks 13–22). We used a restricted second trimester rather than the full second trimester (weeks 13–27) because 22 weeks is currently understood to be the minimum gestation time for infant viability (61). A challenge in studying the relationship of exposures that occur during pregnancy and preterm birth is those who deliver preterm have less time to accrue exposure. By restricting the exposure period to 22 weeks, we ensured that all gestations had an equal number of possible exposure-weeks. A mother was classified as exposed during the pre-conception period, the first trimester, or the second trimester if a homicide occurred during the corresponding weeks in her Census tract (Figure 1). To assess whether higher levels of exposure were associated with worse outcomes, we examined whether exposure to homicide in all three exposure windows (that is, at least one homicide occurred during the pre-conception period, first trimester, and second trimester), or exposure to three or more homicides at any point during the three exposure windows, was associated with preterm birth or SGA.

This study was approved by the Committees for the Projection of Human Subjects for the California Health and Human Services Agency and the University of California, Berkeley. Information for researchers interested in accessing the data used in this study is available in the supplemental material.

Matching and Statistical Analysis

We used general exact matching by Census tract of residence at the time of delivery to estimate within-tract associations. Our approach matched each exposed mother to all unexposed mothers with the same Census tract (62,63). The unexposed group was then weighted by dividing the number of exposed by the number of unexposed to account for the fact that there may be multiple controls for each exposed mother. Including these weights in a linear probability model estimates the average treatment effect on the treated within Census tracts (64,65). The regression model used in the analysis is as follows, with weights that estimate within-tract associations:

$$P\left(Y_{i,j}\right) = \beta_0 + \beta_1 x_j + \beta_2 conception\ \ year + \beta_3 conception\ \ season + \beta_4 race + \beta_5 health\ \ insurance + \beta_6 age + \beta_7 parity + \epsilon_{i,j}$$

Where $Y_{i,j}$ is birth outcome for individual i according to exposure window j and x_j is homicide exposure in pregnancy window j. Due to potential clustering by Census tract, we used Huber-White robust standard errors for inference (66).

We conducted falsification tests in the form of a negative control to assess whether our results could be due to systematic error. For this, we first created a pseudo-birth week, defined as 40 weeks after the conception week, which was estimated using the birth week and the reported gestational age at birth. We did so to ensure any seasonality in preterm deliveries did not contaminate the characterization of the exposure in the negative control. The first negative control was defined as homicide exposure in the 12 weeks (0–3 months) after the pseudo-birth week, and the second control period was the 13–24 weeks (3–6 months) after the pseudo-birth week. Since these exposures occurred after the birth, they could not affect the outcome of the pregnancy. An observed association in either of these analyses would suggest a biasing factor that could affect our results (e.g. residual effects of seasonality, autocorrelation in homicide exposure, or other temporal patterning leading to spurious results). It is important to note, however, that these falsification tests will not identify bias due to measurement error, another important form of systematic error (67). We also used randomization inference to assess the likelihood our results were due to chance. A detailed description of this procedure is available in the supplemental material.

We used R version 3.5 for all statistical analyses (code is available in the supplemental material).

RESULTS

There were 2,184,177 mothers with singleton conceptions between January 1, 2007, and March 1, 2011 who gave birth to a live infant in California. We had complete covariate values for 2,133,662 (97.7%) of these women (Figures 2 and 3). However, a substantial portion—349,077 (16.4%)—of these women were missing values for education. Given the large population size, we were unable to use multiple imputation or other missing data procedures due to the computational limitations. Therefore, our main analysis does not include educational attainment as a covariate. However, we include a sensitivity analysis in which we include educational attainment in the analysis with an indicator for missingness. These results are the same as those presented here, and are available in the supplemental material.

Prevalence of homicide exposure was 8.2% during the pre-conception period, 8.7% during the first trimester, and 6.8% during the second trimester (Table). Approximately 0.9% of mothers were exposed to three or more homicides during the three exposure windows, and 0.2% of mothers were exposed to one or more homicides in each of the three exposure windows. Prevalence of preterm birth was 8.9% and prevalence of SGA was 9.3%. The proportion exposed to homicide and risk of adverse birth outcomes varied across racial/ethnic groups. In the pre-conception, first, and second trimesters, prevalence of homicide exposure was lowest for non-Hispanic white mothers (4.9%, 5.2%, and 4.0%, respectively) and highest for non-Hispanic black mothers (14.0%, 14.7%, and 11.6%, respectively). The prevalence of preterm birth was lowest for Hispanic mothers (8.5%) and highest for non-Hispanic black mothers (13.0%) over the study period. SGA was also more common among non-Hispanic black mothers (15.3%), and least common among non-Hispanic white mothers (7.4%).

In the adjusted analyses of the matched data, we did not find meaningfully increased risk of preterm birth among mothers exposed to a homicide during the pre-conception period (average treatment effect on the treated 0.09% [95% CI −0.06%, 0.24%]) and the first trimester (average treatment effect on the treated 0.07% [95% CI −0.09%, 0.22%]). There was also no association of preterm birth with exposure during the second trimester (average treatment effect on the treated −0.02% [95% CI: −0.19%, 0.14%]) (Figure 4). Randomization inference suggests the observed associations between homicide exposure and preterm birth are likely due to chance (Supplemental Figures 1–3). The relationship between SGA and homicide exposure in the pre-conception period was −0.01% [95% CI −0.17%, 0.15%], the first trimester association was 0.14% [95% CI −0.01%, 0.30%], and the second trimester association was −0.06% [95% CI −0.23%, 0.11%] (Figure 4). The randomization inference suggests the relationship of first trimester homicide exposure and SGA was not due to chance (Supplemental Figure 4 and Supplemental Table 1).

For both preterm birth and SGA, the results of the negative controls were null. For example, the estimated 0–3 months negative control average treatment effect among the treated was −0.02% (95% CI −0.17%, 0.14%) for preterm birth and −0.03% (95% CI −0.19%, 0.12%) for SGA. The 3–6 months negative control average treatment effect on the treated estimate was −0.04% (95% CI −0.10%, 0.12%) for preterm birth and-0.04% (95% CI −0.20%, 0.12%) for SGA.

Exposure to one or more homicides in each of the three exposure windows was associated with elevated risk of SGA (average treatment effect on the treated: 1.09% [95% CI 0.15%, 2.03%]) (Figure 5), but not preterm birth (average treatment effect on the treated: 0.14% [95% CI −0.74%, 1.01%]). Similarly, exposure to three or more homicides at any point during the three exposure windows was associated with increased risk of SGA (average treatment effect on the treated: 0.78% [95% CI 0.15%, 1.40%]), but not preterm birth (average treatment effect on the treated: 0.32% [95% CI −0.28%, 0.92%]). The randomization inference suggests that the results for SGA were unlikely to be due to chance (Supplemental Figures 8 and 9, and Supplemental Table 1).

DISCUSSION

In this study, exposure to homicide during the first trimester of pregnancy was associated with a small increased risk of SGA among mothers in California. We did not find increases in risk of preterm birth associated with homicide exposure during the pre-conception period, the first trimester, or the second trimester. Exposure to multiple homicides was associated with greater elevations in risk of SGA, but not preterm birth. We used a design that matched on community to avoid issues related to structural confounding and neighborhood selection, and found our results were robust to several falsification tests.

Previous studies that have examined homicide exposure and gestational outcomes have found mixed results. A 2016 study of mothers in Brazil found a 1.5% increase in risk of preterm birth associated with one standard deviation increase in homicide rates during the first trimester, with stronger associations found among mothers with lower educational attainment (54). A 2014 study found no association between homicide exposure and gestational age among mothers in Mexico (55). Both studies used fixed effects to estimate within community associations, and the study of Brazilian mothers found the strongest associations for exposures during the first trimester. We utilized a similar approach by matching on communities and also found that the associations were strongest in the first trimester. Differences in results between these studies may be attributable to differences in context—for example, consequences of maternal homicide exposure in Mexico, Brazil, and California may differ based on a variety of factors, including family and community social support, access to prenatal health care, or political empowerment.

There were several limitations to our approach. In particular, our use of a population-level database of birth records had several potential sources of measurement error. We only had access to the mothers' addresses at the time of delivery; however, it is likely some portion of women moved Census tracts during their pregnancy, and their exposure may be incorrectly assigned. The homicides were geocoded from California's death records based on the address of the decedent rather than the location where the homicide occurred. While homicide locations can be captured with crime records, both approaches have some tradeoffs. Our approach does not capture women exposed to a homicide in their neighborhood if the decedent lived elsewhere, or women exposed to homicide in a neighborhood that differs from their own. However, homicides captured in death records are more complete than law enforcement data and can be identified with more granular temporal and geographic resolution. Furthermore, the experienced reaction to violence may have stronger implications for women who know someone who was killed, which is more likely if the victim lived in her neighborhood; there may also be stronger behavioral implications for pregnant women who live in the same neighborhood as the deceased, especially related to diet and exercise, if they modify activities due to fear or grief.

We defined the community of interest as the Census tract in this analysis. We expect that residents would be aware of homicides that occurred within this geographic unit; however, it is possible that smaller units would better align with how women conceive of their neighborhood and who they think of as their neighbors. In general, studies that rely on definitions of neighborhood units may be sensitive to the choice of geographic unit, a problem that has been discussed as the modifiable areal unit problem (68). Our use of within community comparisons, matched on Census tract, avoids comparisons across communities in which other characteristics of communities are likely to differ substantially and lead to problems with structural confounding (57,58). We additionally controlled for year and season of conception. Characteristics of communities that remain constant over the study period are thus controlled by design. However, the approach relies on the assumption that within these short time windows (year and season) there are no time varying confounders that affect homicide occurrence and preterm delivery.

In addition, gestational age is an estimate, and previous validation studies have suggested the clinical estimate we used in this study may underestimate preterm birth compared to early ultrasound (69). Gestational age is most accurate when measured early in pregnancy, and it is possible women who have their first prenatal visit later in their pregnancy could also be more likely to be exposed to homicides in their communities, which may create systematic error associated with the mismeasurement of gestational age. One goal of matching on tract and controlling for individual characteristics was to mitigate this type of error. Finally, because this analysis used birth records that only capture live births or gestations that persist until at least 22 weeks, we were unable account for possible fetal loss or induced abortion due to homicide exposure. An indirect way to address this would be to examine the relationship between homicides and birth rates, but given the small population size of Census tracts, monthly birth rates were too variable to model reliably. Bias due to fetal loss may have especially affected our estimates for second trimester exposure, as the fetus must have survived past the first trimester in order to be exposed. An ongoing research interest for our group is to address the issues of reproductive decision-making, fetal loss, and immortal time bias in studies of violence exposure and birth outcomes.

In this study, we found exposure to homicide during the first trimester of pregnancy was associated with a small increased risk of SGA among mothers in California. Exposure to homicide during the pre-conception period and the second trimester were not associated with SGA. It is possible that the physiologic stress response to homicide exposure is more relevant for adverse birth outcomes earlier in pregnancy, or that behavior changes due to homicide exposure have less time to influence the outcome of the pregnancy when exposure happens in the second trimester. Future studies could consider how these possible mechanisms may differ based on the timing of violence exposure.

Exposure to more than three homicides and exposure during all three exposure windows was associated with larger elevations in risk for SGA. These findings suggest that chronic exposure to homicide, especially during the pre-conception and early pregnancy period, is likely to have a more pronounced impact on risk of SGA compared to a single homicide. Additional research that focuses on creative identification strategies to study chronic violence exposure and reproductive health outcomes is needed to better understand these differences.

These findings add to the growing awareness of violence as a public health issue and the ties between the marginalization of certain communities and health disparities. However, given the small magnitudes of the estimates in this study, it is clear that the disparities in birth outcomes that exist in California can only be explained in very small part by acute variations in fatal community violence exposure. Non-fatal forms of community violence are much more prevalent (70), and therefore more work should be done to investigate the many potential sources of disadvantage, including community and interpersonal violence, that may contribute to disparities in adverse pregnancy outcomes. In order to advance our understanding of how stressful experiences during pregnancy affect maternal health and fetal development, future research should examine other forms of community violence and the biologic and behavioral mechanisms that explain how homicide exposure could increase risk for adverse birth outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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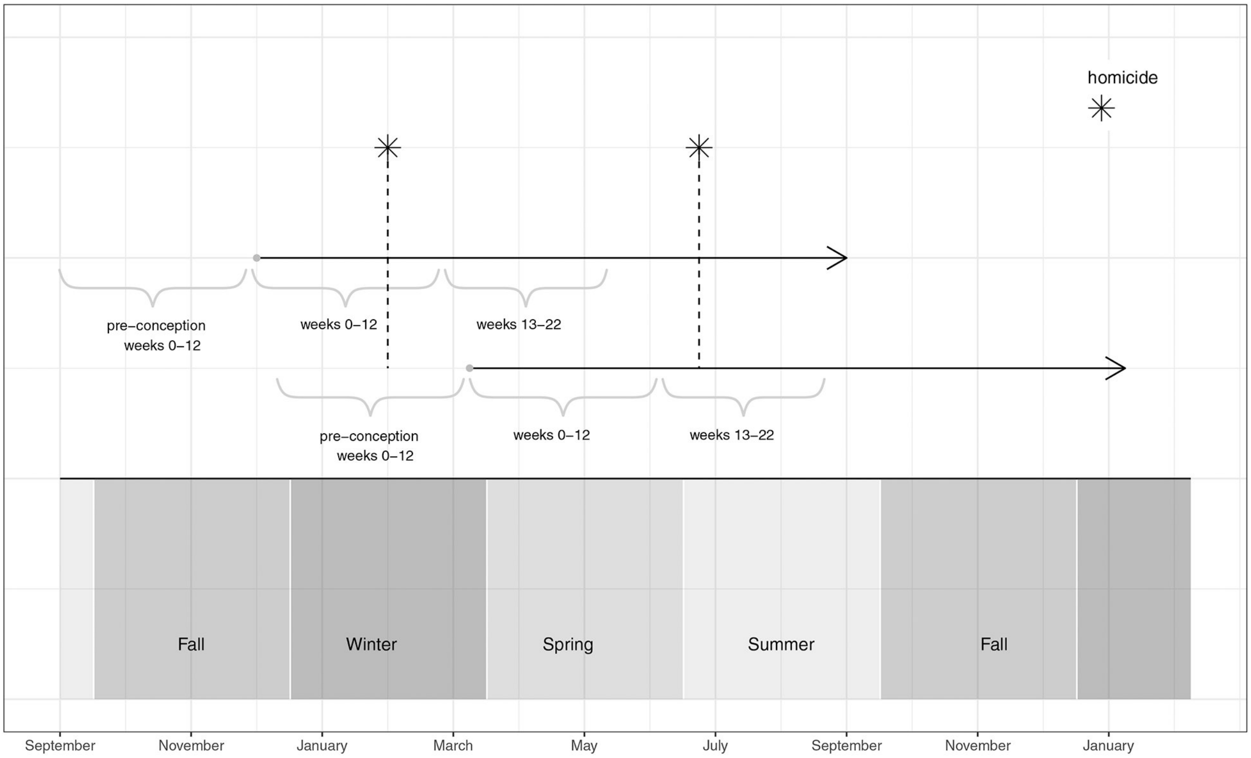

Figure 1.

Diagram illustrating how homicides are mapped to gestations according to the three exposure windows.

Below this figure should be the following note:

The arrows represent two distinct pregnancies for women living in the same neighborhood. The start of the arrow corresponds to the week of conception, and the head of the arrow represents the week of birth. The brackets demonstrate each exposure window corresponding to the two gestations. The stars are homicides that occurred in the neighborhood, and the dotted line illustrates which exposure window each homicide would correspond to for the two different women.

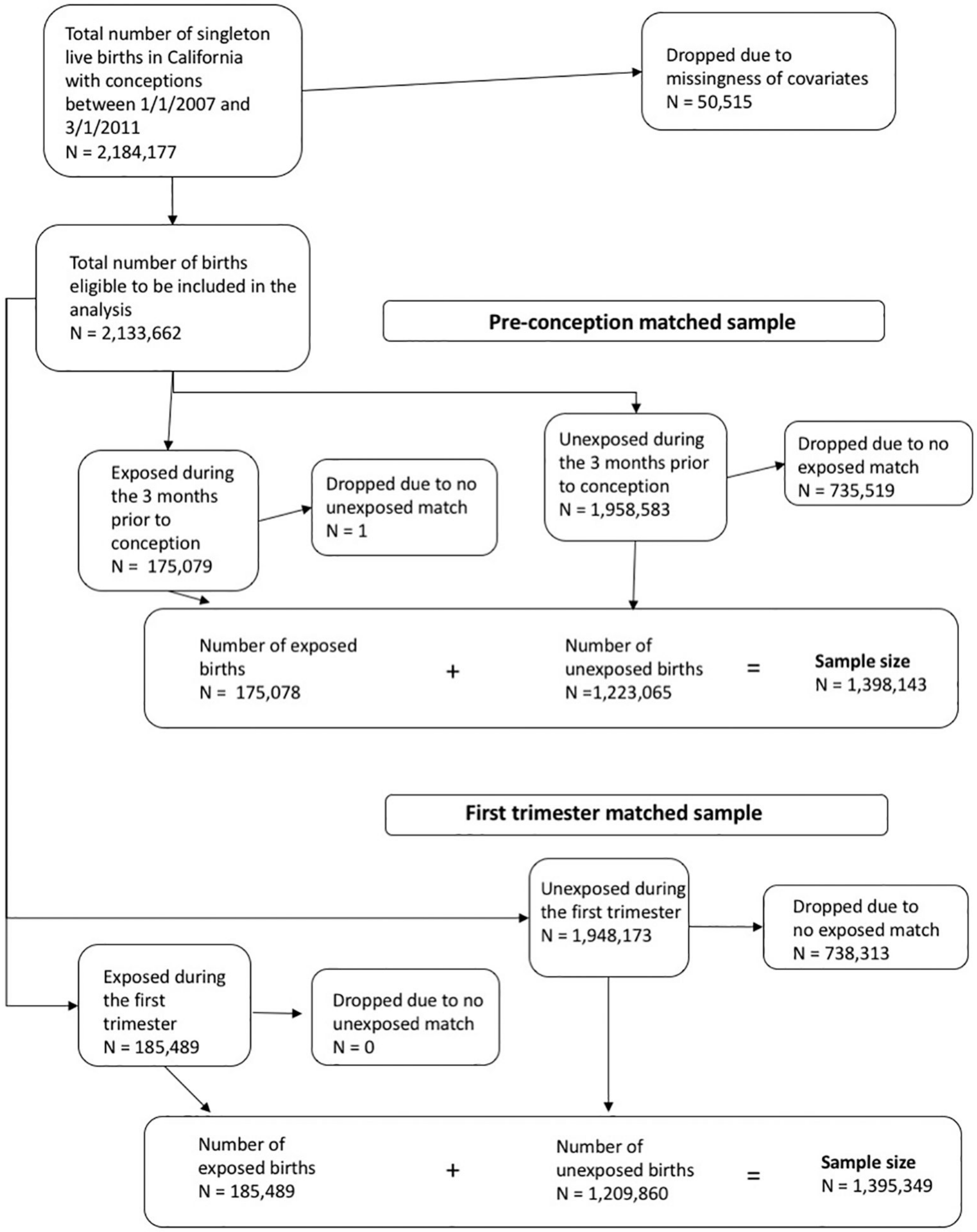

Figure 2.
Illustration of the creation of the matched samples for the pre-conception and first trimester analyses.

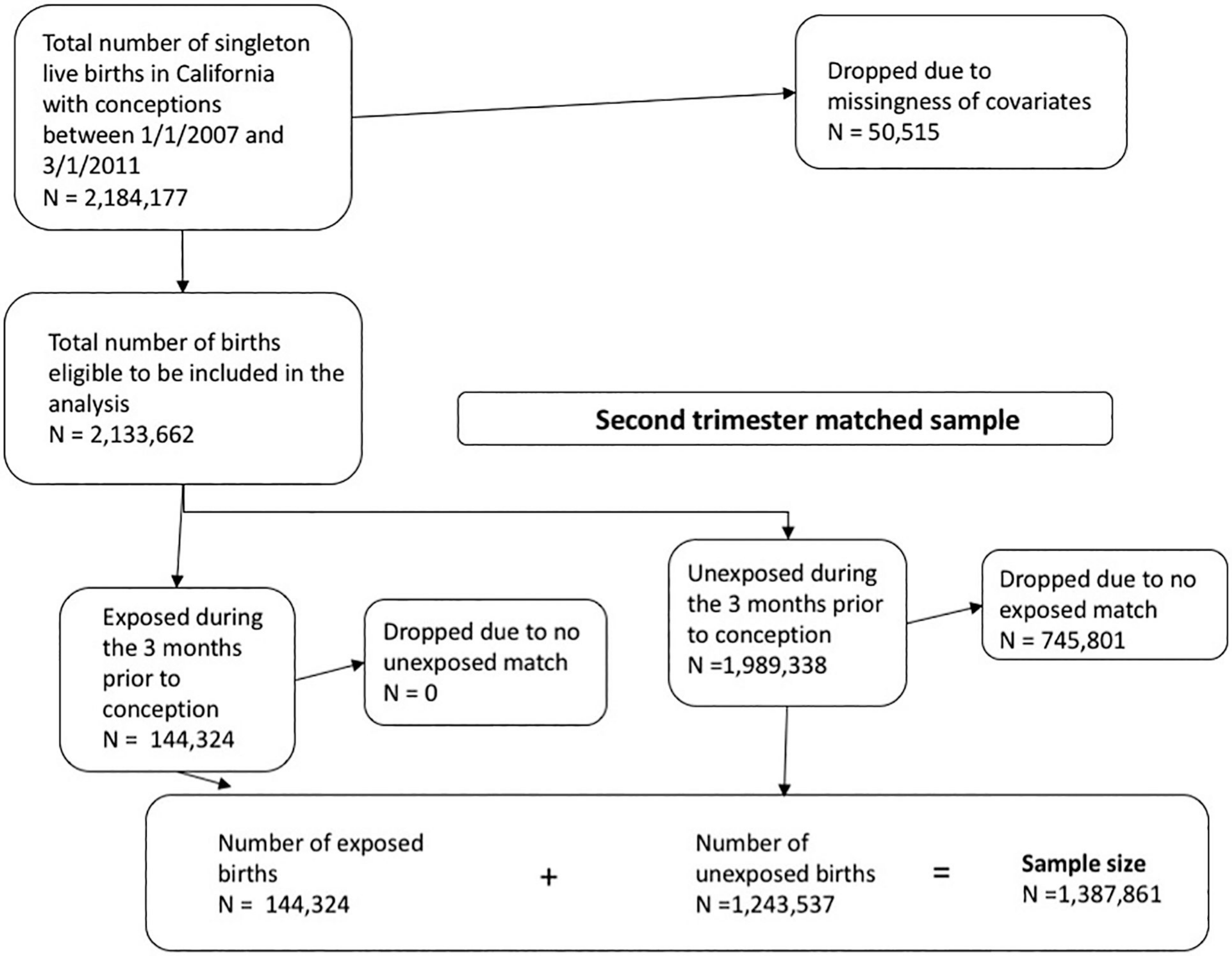

Figure 3.

Illustration of the creation of the matched samples for the second trimester analyses.

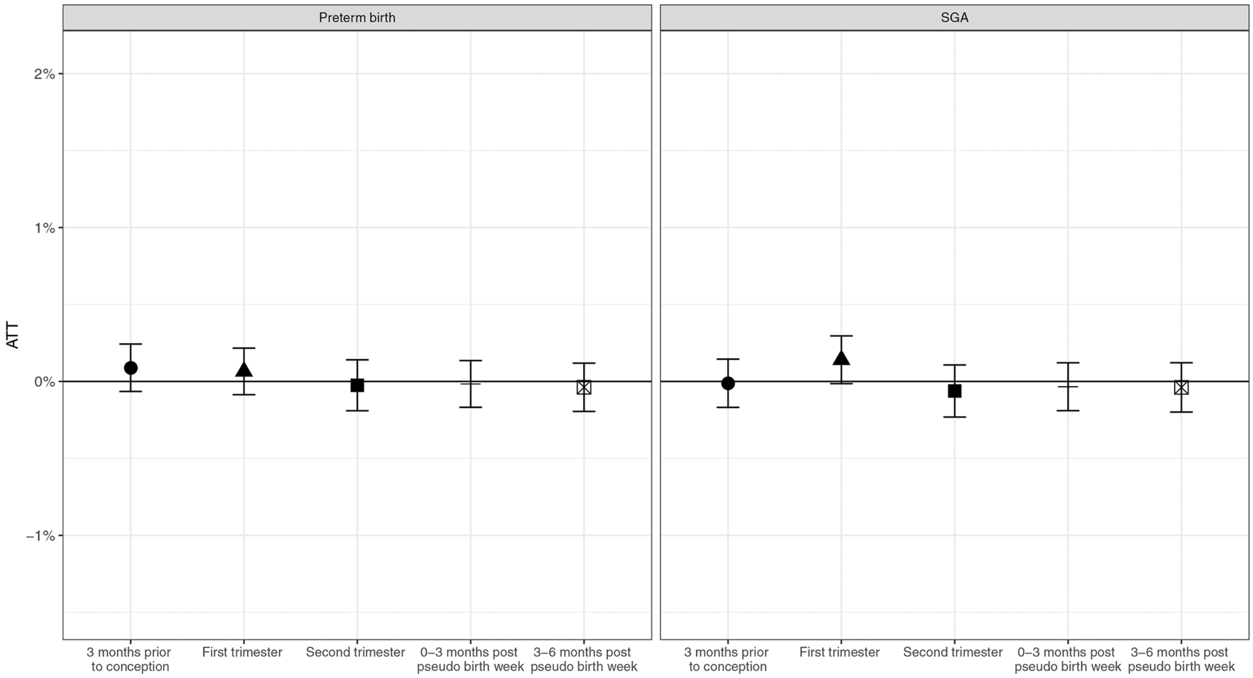

Figure 4.

Estimated average treatment effect on the treated (ATT) of homicide exposure during the pre-conception period (1–12 weeks before conception), first trimester (weeks 0–12), second trimester (weeks 13–22), and the negative controls (0–12 weeks and 13–24 weeks after the pseudo-birth week) on risk of preterm birth and SGA in California, 2007–2011.

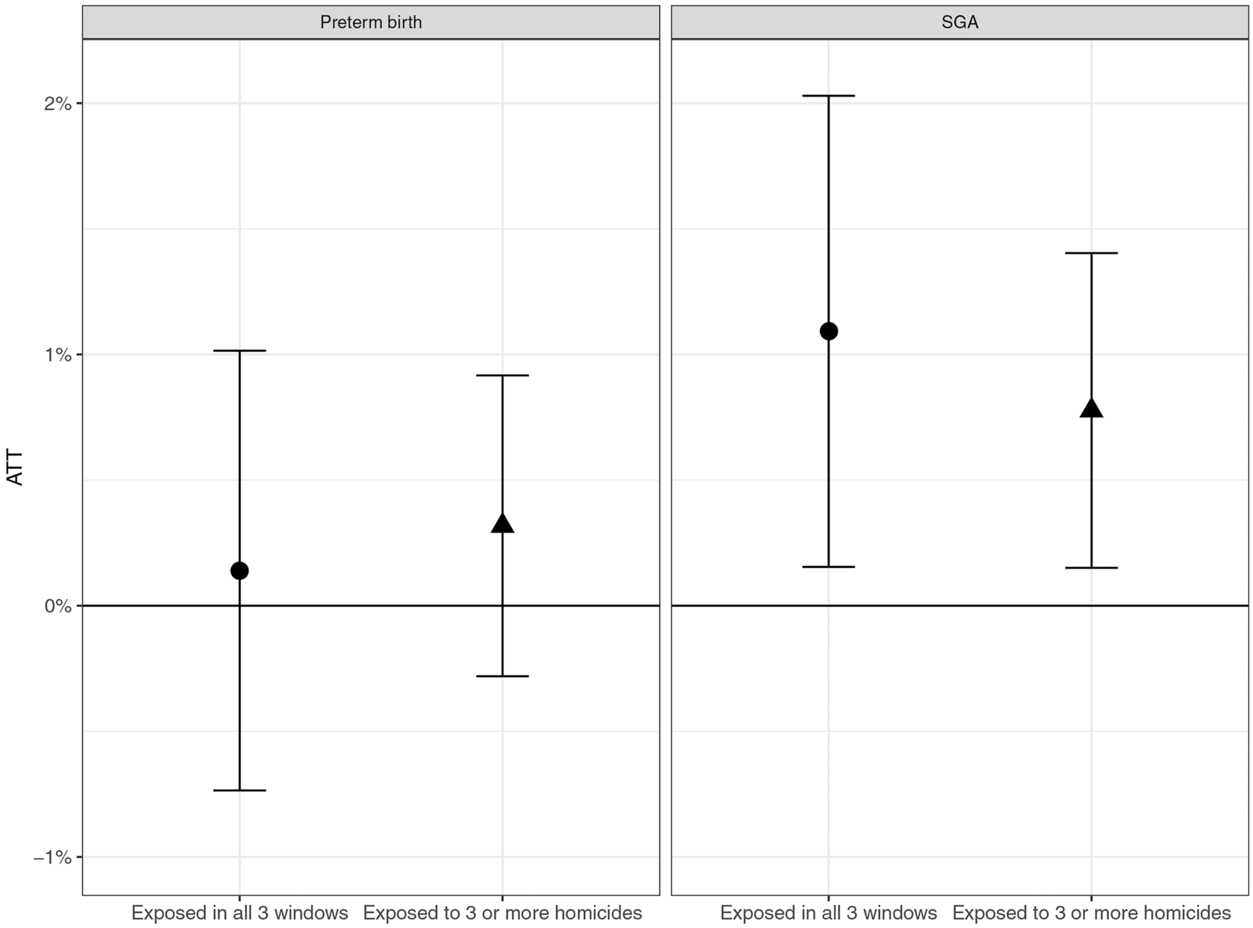

Figure 5.

Estimated average treatment effect on the treated (ATT) of homicide exposure during all three exposure windows and exposed to at least three homicides on risk of preterm birth and SGA in California, 2007–2011.

Table 1.

Descriptive statistics of homicide exposure and birth outcomes overall and by race/ethnicity

	Complete case full data		Matched data for pre-conception exposure		Matched data for first trimester exposure		Matched data for second trimester exposure	
Total sample size	**2,133,662**		**1,398,143**		**1,395,349**		**1,387,861**	
	N	**(%)**	**N**	**(%)**	**N**	**(%)**	**N**	**(%)**
Exposed to pre-conception homicide								
All	175,079	8.2%	175,078	12.5%	173,977	12.5%	172,036	12.4%
White, NH	28,801	4.9%	28,801	9.7%	28,524	9.6%	27,974	9.5%
Black, NH	16,333	14.0%	16,333	17.3%	16,286	17.3%	16,196	17.3%
Asian, NH	14,452	5.5%	14,452	10.2%	14,307	10.1%	14,066	10.0%
Hispanic	110,960	10.0%	110,959	13.4%	110,360	13.4%	109,356	13.3%
Exposed to first trimester homicide								
All	185,489	8.7%	184,534	13.2%	185,489	13.3%	184,249	13.3%
White, NH	30,217	5.2%	29,907	10.1%	30,217	10.2%	29,900	10.1%
Black, NH	17,171	14.7%	17,131	18.1%	17,171	18.2%	17,123	18.3%
Asian, NH	15,199	5.8%	15,058	10.7%	15,199	10.8%	15,043	10.7%
Hispanic	118,120	10.7%	117,692	14.2%	118,120	14.3%	117,429	14.3%
Exposed to second trimester homicide								
All	144,324	6.8%	142,148	10.2%	143,583	10.3%	144,324	10.4%
White, NH	23,225	4.0%	22,618	7.6%	23,007	7.8%	23,225	7.9%
Black, NH	13,500	11.6%	13,414	14.2%	13,478	14.3%	13,500	14.4%
Asian, NH	11,866	4.5%	11,560	8.2%	11,758	8.3%	11,866	8.5%
Hispanic	91,990	8.3%	90,885	11.0%	91,621	11.1%	91,990	11.2%
Exposed to one or more homicides in all 3 exposure windows								
All	5,233	0.2%	5,233	0.4%	5,233	0.4%	5,233	0.4%
White, NH	346	0.1%	346	0.1%	346	0.1%	346	0.1%
Black, NH	907	0.8%	907	1.0%	907	1.0%	907	1.0%
Asian, NH	282	0.1%	282	0.2%	282	0.2%	282	0.2%
Hispanic	3,572	0.3%	3,572	0.4%	3,572	0.4%	3,572	0.4%
Exposed to 3 or more homicides								
All	20,225	0.9%	20,218	1.4%	20,225	1.4%	20,206	1.5%
White, NH	1,855	0.3%	1,851	0.6%	1,855	0.6%	1,853	0.6%
Black, NH	3,144	2.7%	3,144	3.3%	3,144	3.3%	3,141	3.4%
Asian, NH	1,298	0.5%	1,297	0.9%	1,298	0.9%	1,298	0.9%
Hispanic	13,450	1.2%	13,448	1.6%	13,450	1.6%	13,437	1.6%
Preterm								
All	189,704	8.9%	126,419	9.0%	126,083	9.0%	125,488	9.0%
White, NH	50,859	8.7%	26,191	8.8%	26,083	8.8%	25,921	8.8%

	Complete case full data		Matched data for pre-conception exposure		Matched data for first trimester exposure		Matched data for second trimester exposure	
Total sample size	**2,133,662**		**1,398,143**		**1,395,349**		**1,387,861**	
	N	**(%)**	**N**	**(%)**	**N**	**(%)**	**N**	**(%)**
Black, NH	15,183	13.0%	12,460	13.2%	12,442	13.2%	12,398	13.2%
Asian, NH	23,225	8.9%	12,886	9.1%	12,862	9.1%	12,754	9.1%
Hispanic	94,710	8.5%	71,088	8.6%	70,900	8.6%	70,670	8.6%
SGA								
All	198,196	9.3%	132,530	9.5%	132,194	9.5%	131,475	9.5%
White, NH	43,516	7.4%	22,670	7.6%	22,568	7.6%	22,453	7.6%
Black, NH	17,935	15.3%	14,900	15.8%	14,885	15.8%	14,768	15.8%
Asian, NH	34,275	13.1%	18,626	13.2%	18,647	13.2%	18,479	13.2%
Hispanic	96,967	8.7%	72,751	8.8%	72,552	8.8%	72,267	8.8%

Note: The racial/ethnic groups for which we present separate categories have sufficient sample size to be presented on their own. There are mothers of other race/ethnicities in the "all" category.