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Community violence and pregnancy: An understudied exposure in the etiology of adverse birth outcomes

by

Dana E. Goin

A dissertation submitted in partial satisfaction of the

requirements for the degree of

Doctor of Philosophy

in

Epidemiology

in the

Graduate Division

of the

University of California, Berkeley

Committee in charge:

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Associate Professor Maya Petersen
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Spring 2019

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Abstract

Community violence and pregnancy: An understudied exposure in the etiology of adverse birth outcomes

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Dana E. Goin

Doctor of Philosophy in Epidemiology

University of California, Berkeley

Associate Professor Jennifer Ahern, Chair

Understanding the etiology of adverse birth outcomes is of critical public health importance. Preterm delivery and small-for-gestational age births are especially problematic because infants with these conditions experience heightened risk for serious morbidities, mortality, and poorer health and economic outcomes over the life course. There is stark patterning by race/ethnicity and socioeconomic position in the prevalence of adverse birth outcomes, but individual-level risk factors have not adequately explained these disparities. Researchers therefore have expanded their focus upstream to potential fundamental causes like neighborhood poverty and structural racism. However, given that these causes do not necessarily map to realistic interventions, researchers have sought to find a middle ground between fundamental and proximal causes, where the links between levels of causation are clearly articulated, to acknowledge the source of the inequities, but to also identify plausible policy or clinical interventions that could reduce disparities in maternal and infant health. This dissertation contributes to this literature by examining the role of community violence in the etiology of adverse pregnancy outcomes and the perpetuation of inequities in reproductive health. Community violence is a social exposure that is associated with neighborhood and individual socioeconomic status, factors that have been linked to adverse birth outcomes around the world. In addition, Black women in the United States are disproportionately likely to experience both community violence and poor pregnancy outcomes; therefore, community violence may be an important factor for understanding disparities in reproductive health outcomes by race/ethnicity. Furthermore, given that violence in a community is often tied to histories of racial/ethnic segregation, economic exclusion, and social marginalization, exposure to community violence may be an important mechanism through which historical patterns of social inequity continue to shape health disparities today. The first chapter of this dissertation provides background on this question and details potential pathways that could connect community violence with pregnancy outcomes. The second chapter addresses how exposure to homicide in one's neighborhood during early pregnancy influences risk of preterm delivery and small-for-gestational-age birth. The third chapter examines whether

overall levels of firearm violence in a woman's community impact her risk of pregnancy complications and health behaviors during pregnancy and subsequent preterm birth. This work aims to assess the relationship between these factors, and to explore how they may be operating to produce disparities in preterm birth. The fourth chapter examines the relationship between exposure to fatal police violence during pregnancy on the hazard of early and late preterm delivery, and assesses whether there is effect modification by race/ethnicity. This chapter also addresses the fact that exposure to violence-related stress during pregnancy may be associated with an increased risk of spontaneous or induced abortion, and includes a simulation study to explore how using birth records that exclude these conceptions may affect the findings. The fifth chapter concludes. Overall, this work contributes new evidence about the relationship between community violence and adverse pregnancy outcomes, and improves our understanding of the etiology of inequities in reproductive health.

This work is dedicated to Chelsea Miller Goin.

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Chapter 1

Introduction

Social and economic disadvantage are well-known risk factors for adverse pregnancy outcomes [1–8]. In particular, infants born preterm are at the highest risk for morbidity and mortality [9], and evidence suggests preterm birth and intrauterine growth restriction are associated with elevated disease risk later in life [10–13]. While there is clear evidence that socioeconomic disadvantage is associated with increased risk of adverse pregnancy outcomes, the mechanisms underlying this phenomenon are not well understood [1, 3, 6, 14–20]. For example, Black women have increased risk of preterm birth compared to non-Hispanic white women, even after adjusting for individual and neighborhood socioeconomic position [2–4, 18]. Despite a recent increase in studies investigating links between structural factors and reproductive health outcomes [3, 15, 21], our understanding of the pathways that connect individual socioeconomic status, neighborhood environment, race/ethnicity, and adverse birth outcomes remains inadequate. Investigating how specific aspects of social, economic, and neighborhood disadvantage influence risk of poor pregnancy outcomes is critical to identifying interventions that could improve population health and reduce disparities in maternal and infant health in the United States.

This dissertation examines the relationship between exposure to community violence during pregnancy and a woman's risk of adverse pregnancy outcomes. Some evidence suggests that living in neighborhoods with high rates of violent crime increases the risk of preterm birth among both Black and white women [14, 22], but these studies do not adequately control for neighborhood environment, do not measure violence exposure specific to each woman's gestation, and likely suffer from structural confounding issues [5]. Nevertheless, community violence is an important factor to consider in the etiology of adverse birth outcomes because it is socially and geographically patterned in ways similar to adverse maternal and infant health outcomes. In addition, exposure to violence likely causes psychosocial stress among pregnant women, which has been identified as risk factor for preterm birth [23]. In addition, Black women are much more likely to experience community violence compared to non-Hispanic white women [24–26]. Thus exposure to community violence may play a key role in unexplained disparities of adverse maternal and infant health outcomes between Black and white women. This dissertation aims to contribute rigorous population-level evidence

to improve our understanding of how exposure to community violence influences the burden of adverse pregnancy outcomes and disparities in reproductive health in California.

Exposure to community violence is an environmental stressor that is likely to elicit a negative stress response in most women. Stress itself can be conceptualized as a challenge to a womans psychological or physiological system that her body is has difficulty managing or protecting itself from. The assessment of the severity of the stressor is known as appraisal, and can vary across individuals [27]. Some women may have increased resilience or reserve capacity (resources such as emotional management tools, social support, neighborhood or family social capital, etc.) to mitigate the effects of stressors, and therefore may experience attenuation of the stress response [28]. It is important to acknowledge the variation in possible stress responses because they may correspond to different plausible pathways between exposure to community violence and heightened risk for poor pregnancy outcomes.

Exposure to community violence may affect pregnancy outcomes via two mechanisms: first, there may be behavioral changes associated with exposure that influence other risk factors for adverse birth outcomes, and second, there may be direct physiological effects of stress that have implications for fetal development and the parturition process [29–31]. These are not mutually exclusive categories and there are likely feedback loops between them. However, this categorization is useful in order to think about the main mediating pathways that are likely to connect community violence and health during pregnancy.

Some women may respond to community violence by adopting coping behaviors in order to mitigate 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 [32–34]. Each of these behaviors has either been directly linked or is related to pregnancy complications associated with adverse pregnancy outcomes [35–43].

Evidence also suggests there are direct physiological mechanisms that could link stress related to community violence to disease risk and health during pregnancy [44, 45]. In response to a perceived threat, the sympathetic nervous system is activated and stimulates the sympatheticadrenal-medullary (SAM) axis [46]. The sympathetic nervous system has nerve fibers that extend to primary and secondary lymphoid tissues, and activation of the SAM axis results in secretion of catecholamines like epinephrine and norepinephrine from the adrenal glands [46]. These hormones are primarily responsible for the increased heart rate and blood flow associated with the "fight or flight" response. In addition, many immune cells have receptors for norepinephrine (called b2-adrenergic receptors), and once they bind to norepinephrine a process is initiated that increases production of cAMP (cyclic adenosine monophosphate), which can up-regulate transcription factors necessary for production of pro-inflammatory cytokines [46]. This is consistent with laboratory studies which have demonstrated that acute stressors tend to increase the number natural killer cells in peripheral blood and heighten secretion of proinflammatory cytokines [47]. A shift in the bias of the immune response from cell-mediated to humoral has been observed for other kinds of short-term naturalistic stressors as well [47]. This may be a result of an improved capacity for innate and humoral immune responses to address short-term challenges, while cell-mediated

responses take more time to develop and therefore may be maladaptive in situations of acute stress. Studies have suggested that acute stress events may play a role in preterm birth and intrauterine growth restriction as elevations in inflammatory cytokines and other markers of immune function associated with stress during early pregnancy have been linked to early delivery and babies born small for gestational age [19, 48–51].

Experiencing community violence as an acute stressor can also activate the hypothalamic-pituitary-adrenal (HPA) axis. The hypothalamus responds to stress signaling by secreting corticotropin-releasing hormone (CRH). This hormone stimulates the pituitary to secrete adrenocorticotrophin hormone (ACTH), which travels to the adrenal glands and instigates production of glucocorticoids like cortisol. Increasing levels of glucocorticoids is associated with suppression of inflammation and immune activity, as it is believed that glucocorticoids inhibit NF-κB, a transcription factor important in the production of pro-inflammatory cytokines like IL-1b, TNF-α, and IL-6 [46]. The placenta is also a source of CRH production. In normal human pregnancy, placental CRH 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 [16, 19]. It is thought that placental CRH production stimulates the fetal HPA-axis, which may be responsible for instigating the events leading to parturition [51]. In contrast to the inhibitory effects of cortisol on hypothalamic CRH, cortisol and other biological effectors of stress are associated with increased production of placental CRH, which may contribute to premature stimulation of the fetal HPA-axis and subsequent premature delivery [23, 51, 52].

Prolonged exposure to stress may also have detrimental health effects via dysregulation of the signaling between neuroendocrine and immune systems, with potentially either immunosuppressive or inflammatory effects [47, 53]. For example, studies indicate that severe and prolonged stress can cause immune suppression over time which is thought to be a result of heightened exposure to glucocorticoids like cortisol [19, 47, 54–58]. Evidence suggests that suppression of the maternal immune system can lead to increased risk of infection during pregnancy, which has been linked to preterm birth [59–62]. Dysregulated immune function may also be related to preeclampsia [63, 64] and asthma during pregnancy [65, 66]. However, chronic exposure to stress hormones can also cause immune cells to down-regulate expression of glucocorticoid receptors, resulting in desensitization to the immune-regulating effects of cortisol [53]. This may explain why some people experiencing chronic stress also experience exacerbations of autoimmune and allergic diseases whose symptoms we would expect to be alleviated in an immunosuppressive state [67, 68]. If chronic stress induces a heightened state of inflammation, we would expect the effects to be similar to the inflammatory effects instigated by exposure to acute stressors. In particular, elevated levels of inflammatory cytokines and dysregulation of the HPA axis may precipitate adverse birth outcomes.

Community violence therefore can act as both an acute and chronic stressor for pregnant women and may result in either immunosuppressive or inflammatory states that can impact risk for adverse pregnancy outcomes. The proposed mechanisms described here illustrate plausible biologic pathways that could link exposure to community violence to increased risk for poor birth outcomes, but it is unlikely that all individuals will experience the same cascade

of events nor will individuals with the same exposure necessarily have similar pregnancy outcomes. There are other factors that likely interact with these systems to modify risk. However, these pathways demonstrate the biologic plausibility of the relationship between exposure to community violence and increased risk of adverse birth outcomes.

Chapter 2 of this dissertation examines whether being exposed to homicide in a woman's Census tract immediately before conception and during early pregnancy is associated with increased risk of preterm birth and small-for-gestational-age births. Small-for-gestational age is a proxy measurement for intrauterine growth restriction that is based on infant sex-specific growth charts by gestational age at delivery [69]. Women are matched within Census tracts, so time-invariant characteristics of the neighborhood are controlled, while the variation in exposure according to when women conceive is leveraged to isolate the association between homicide exposure and these adverse outcomes. This addresses problems of structural confounding and adds important evidence about the role of acute violence exposure during pregnancy on infant health at birth.

Since chronic exposure to violence is likely to have different (and potentially stronger) effects on pregnancy health [45], Chapter 3 aims to assess how overall levels of firearm violence within a mother's zip code affect her risk of preterm delivery, and explores the role of pregnancy complications and health behaviors in mediating that association. This chapter utilizes propensity score matching to identify neighborhoods with similar characteristics but differing levels of firearm violence. Stochastic direct and indirect effects are estimated with early preeclampsia, asthma, gestational diabetes, infection, and substance use during pregnancy as potential mediators. Pre-pregnancy health characteristics that could affect both the mediator and outcome were accounted for in the estimation procedure, which is a strength of the stochastic mediation method. The inability to account for these types of variables has been a challenge to previous attempts to conduct mediation analyses in perinatal epidemiology.

Chapter 4 examines exposure to fatal police violence in one's Census tract during pregnancy on the hazard of early and late preterm birth. Fatal police violence is a form of community violence that is greatly understudied in terms of its impact on population health. This type of violence has been identified as a form of institutionalized racism that differentially affects men and women of color and is a barrier to achieving health equity [70]. Similar to Chapter 2, Chapter 4 compares women within Census tracts who conceived at different times and therefore had different exposures to fatal police violence during pregnancy. This chapter uses a survival analysis framework, which accounts for the fact that women who deliver preterm have less time to accrue exposure during pregnancy. Since men and women of color are more likely to die at the hands of police [71, 72], women of color may experience this stressor more acutely than white women. Therefore, this chapter examines whether or not there was effect modification by race/ethnicity in the association of exposure to fatal police violence and preterm birth.

Finally, each of the chapters in this dissertation utilizes birth records from the state of California. One limitation of using birth records to study social stressors and adverse pregnancy outcomes is that while birth records are exhaustive and complete for live births,

they exclude early pregnancy loss. In the event that exposure to violence induces a stress response that indicates an unsafe environment for the fetus, it is possible weaker fetuses are selectively aborted. This would result in left truncation, which could bias results if only the healthiest fetuses survive long enough to be observed in the birth record. However, measuring rates of spontaneous abortion is notoriously difficult because many women who experience early pregnancy loss do not report it and in some cases may not even know themselves that they were pregnant. Furthermore, women who are exposed to community violence may decide they do not want to bring a child into the world as a result of stress and worry from the exposure. To understand the role of left-truncation due to spontaneous and induced abortion in studies of community violence and preterm birth, Chapter 4 includes a simulation study that investigates the scenarios in which bias may be introduced as a result of this phenomenon.

Untangling the associations between stressors associated with the social environment and adverse pregnancy outcomes is required before new prevention efforts can be effectively identified, organized, and deployed. This dissertation evaluates whether community violence, as a plausibly modifiable exposure that is a consequence of other fundamental causes of adverse reproductive health outcomes, meaningfully contributes to the patterns of adverse birth outcomes we see in California. Connecting the dots between these historical, community-level, and individual-level factors and their roles in perpetuating health inequities is important in order to understand their joint and independent relationships with health behaviors and disease etiology, and to identify as many places for intervention as possible. This dissertation aims to advance the field's understanding of how stress, pregnancy complications, and health behaviors related to community violence influence disparities in reproductive health.

Chapter 2

Homicide and Adverse Birth Outcomes

2.1 Introduction

Community violence is an aspect of the contextual environment that has been recognized as relevant to fetal and infant health [73], but which has thus far been understudied in perinatal research. The adverse effects of direct violent injury on pregnant women and their infants are well documented [74–76]; however, violent events in ones community may have impacts beyond those directly harmed. Pregnant women may 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 [77]. Exposure to community violence may also play a role in the well-documented racial/ethnic disparities in pregnancy complications, gestational weight gain [78, 79], growth restriction, and preterm birth [2, 4, 6, 80], through disparities in exposure to violence and/or differential response (e.g., greater internalization of stress) to violence [81].

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 a significant role in neighborhood economic development in the 20th century [82–84], and patterns of investment associated with racial and economic segregation have implications for community health today [6, 7, 85, 86], especially for lower income and minority women. Black women are more likely to live in economically neglected neighborhoods [82], to be exposed to violence during pregnancy [24, 26], and to have elevated risk of preterm birth compared to white women [2, 23, 80]. In-utero exposure to community violence may also have consequences for health and economic outcomes later in life, contributing to disparities in adult health [87–91].

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 [32–34]. 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 [35, 37–42, 92].

Second, there is evidence of direct biological stress response mechanisms associated with community violence that may impact health during pregnancy [44, 45], although some recent evidence has been mixed [93]. Experiencing stress due to community violence can activate the hypothalamic-pituitary-adrenal (HPA) axis, which secretes corticotropin-releasing hormone (CRH) that stimulates the production of glucocorticoids like cortisol. The placenta also produces CRH, and cortisol and other biological effectors of stress are associated with increased production of placental CRH, which may be responsible for instigating the events leading to parturition [23, 52, 77, 94]. Placental CRH 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 [16, 19]. Therefore, stressful events that activate the maternal HPA axis early in pregnancy may lead to increased production of placental CRH and overstimulation of the fetal HPA 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 [2, 4, 15, 18, 95–98]. However, literature assessing the relationship between community violence and gestational health is still in its infancy [14, 22, 99–101]. One of the main challenges in studying community violence is structural confounding [5, 102]. 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 [103].

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 [23, 77], 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.

2.2 Data and 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 January 1, 2007 and March 1, 2011 to ensure women of all gestational lengths were eligible to be included in the analysis to avoid fixed cohort bias [104].

Homicides were identified 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. SGA was determined using birthweight, gestational age, and infant sex from the birth record and classified according to the sex-specific reference from Talge et al [69]. Covariates were obtained from the birth record and included mothers age, race/ethnicity, parity, health insurance type used for prenatal care, conception year, and conception season. We categorized maternal age as younger than 20, 20-24, 25-29, 30-34, and 35 or older. Educational attainment was categorized as less than high school, high school graduate, some college or associates degree, bachelors degree, graduate degree, or missing. Insurance types were no prenatal care, Medi-Cal without Comprehensive Perinatal Services Program (CPSP) services, other government insurance, private insurance, self-pay, Medi-Cal with CPSP services, and other. Parity was categorized as 1, 2, or 3 or more. 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 [105]. 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 2.1). 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 [106, 107]. 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 (ATT) within Census tracts [108, 109]. The regression model used in the analysis is as follows, with weights that estimate within-tract associations:

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

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 [110].

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 [111].

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

2.3 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 2.2 and 2.3). However, a significant portion349,077 (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 1). 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 (ATT: 0.09% [95% CI -0.06%, 0.24%]) and the first trimester (ATT: 0.07% [95% CI -0.09%, 0.22%]). There was also no association of preterm birth with exposure during the second trimester (ATT: -0.02% [95% CI: -0.19%, 0.14%]) (Figure 2.4). The relationship between SGA and homicide exposure in the pre-conception period was ATT: -0.01% [95% CI -0.17%, 0.15%], the first trimester association was ATT: 0.14% [95% CI -0.01%, 0.30%], and the second trimester association was ATT: -0.06% [95% CI -0.23%, 0.11%] (Figure 4). For both preterm birth and SGA, the results of the negative controls were null. For example, the estimated 0-3 months negative control ATT 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 ATT estimate was -0.04% (95% CI -0.10%, 0.12%) for preterm birth and-0.04% (95% CI -0.20%, 0.12%) for SGA. Randomization inference suggests the observed associations between homicide exposure and preterm birth are likely due to chance (Supplemental Figures 1-3). 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).

Exposure to one or more homicides in each of the three exposure windows was associated with elevated risk of SGA (ATT: 1.09% [95% CI 0.15%, 2.03%]) (Figure 2.5), but not preterm birth (ATT: 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 (ATT: 0.78% [95% CI 0.15%, 1.40%]), but not preterm birth (ATT: 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).

2.4 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 [100]. A 2014 study found no association between homicide exposure and gestational age among mothers in Mexico [101]. 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 contextfor 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 Californias 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 [112]. 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 [5, 103]. 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 [113]. 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 [114], 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.

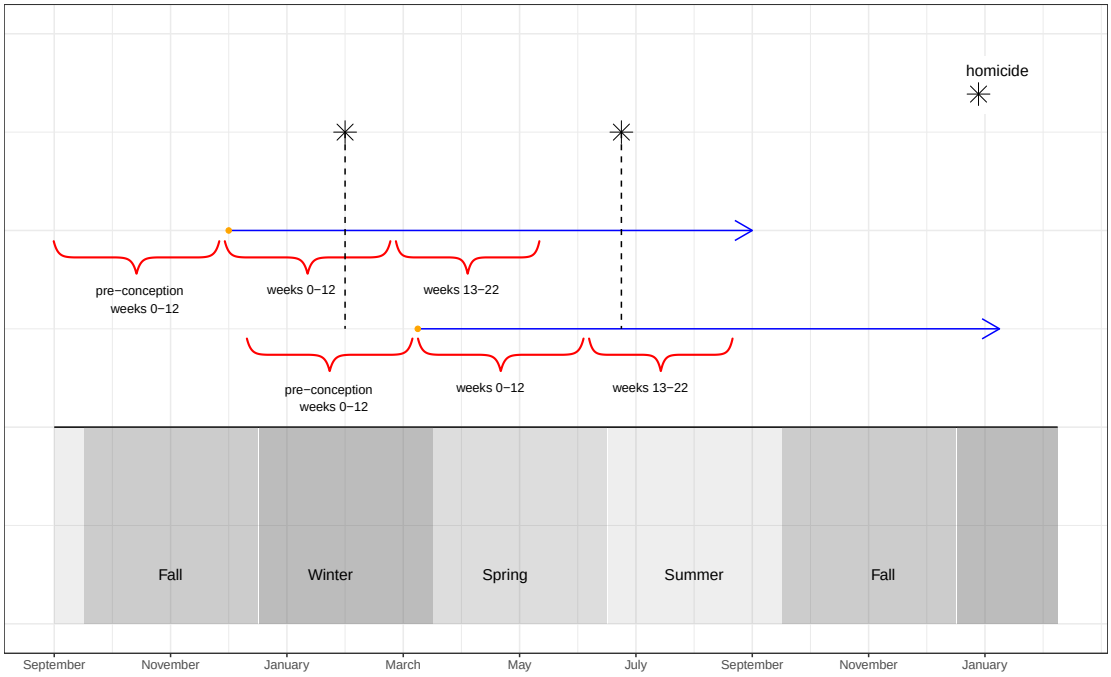


Figure 2.1: Diagram illustrating how homicides are mapped to gestations according to the three exposure windows.

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.

	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	8.20%	175,078	12.50%	173,977	12.50%	172,036	12.40%
White, NH	28,801	4.90%	28,801	9.70%	28,524	9.60%	27,974	9.50%
Black, NH	16,333	14.00%	16,333	17.30%	16,286	17.30%	16,196	17.30%
Asian, NH	14,452	5.50%	14,452	10.20%	14,307	10.10%	14,066	10.00%
Hispanic	110,960	10.00%	110,959	13.40%	110,360	13.40%	109,356	13.30%
Exposed to first trimester homicide								
All	185,489	8.70%	184,534	13.20%	185,489	13.30%	184,249	13.30%
White, NH	30,217	5.20%	29,907	10.10%	30,217	10.20%	29,900	10.10%
Black, NH	17,171	14.70%	17,131	18.10%	17,171	18.20%	17,123	18.30%
Asian, NH	15,199	5.80%	15,058	10.70%	15,199	10.80%	15,043	10.70%
Hispanic	118,120	10.70%	117,692	14.20%	118,120	14.30%	117,429	14.30%
Exposed to second trimester homicide								
All	144,324	6.80%	142,148	10.20%	143,583	10.30%	144,324	10.40%
White, NH	23,225	4.00%	22,618	7.60%	23,007	7.80%	23,225	7.90%
Black, NH	13,500	11.60%	13,414	14.20%	13,478	14.30%	13,500	14.40%
Asian, NH	11,866	4.50%	11,560	8.20%	11,758	8.30%	11,866	8.50%
Hispanic	91,990	8.30%	90,885	11.00%	91,621	11.10%	91,990	11.20%
Exposed to one or more homicides in all 3 exposure windows								
All	5,233	0.20%	5,233	0.40%	5,233	0.40%	5,233	0.40%
White, NH	346	0.10%	346	0.10%	346	0.10%	346	0.10%
Black, NH	907	0.80%	907	1.00%	907	1.00%	907	1.00%
Asian, NH	282	0.10%	282	0.20%	282	0.20%	282	0.20%
Hispanic	3,572	0.30%	3,572	0.40%	3,572	0.40%	3,572	0.40%
Exposed to 3 or more homicides								
All	20,225	0.90%	20,218	1.40%	20,225	1.40%	20,206	1.50%
White, NH	1,855	0.30%	1,851	0.60%	1,855	0.60%	1,853	0.60%
Black, NH	3,144	2.70%	3,144	3.30%	3,144	3.30%	3,141	3.40%
Asian, NH	1,298	0.50%	1,297	0.90%	1,298	0.90%	1,298	0.90%
Hispanic	13,450	1.20%	13,448	1.60%	13,450	1.60%	13,437	1.60%
Preterm								
All	189,704	8.90%	126,419	9.00%	126,083	9.00%	125,488	9.00%
White, NH	50,859	8.70%	26,191	8.80%	26,083	8.80%	25,921	8.80%
Black, NH	15,183	13.00%	12,460	13.20%	12,442	13.20%	12,398	13.20%
Asian, NH	23,225	8.90%	12,886	9.10%	12,862	9.10%	12,754	9.10%
Hispanic	94,710	8.50%	71,088	8.60%	70,900	8.60%	70,670	8.60%
SGA								
All	198,196	9.30%	132,530	9.50%	132,194	9.50%	131,475	9.50%
White, NH	43,516	7.40%	22,670	7.60%	22,568	7.60%	22,453	7.60%
Black, NH	17,935	15.30%	14,900	15.80%	14,885	15.80%	14,768	15.80%
Asian, NH	34,275	13.10%	18,626	13.20%	18,647	13.20%	18,479	13.20%
Hispanic	96,967	8.70%	72,751	8.80%	72,552	8.80%	72,267	8.80%

Table 2.1: Descriptive statistics of homicide exposure and birth outcomes overall and by race/ethnicity

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.

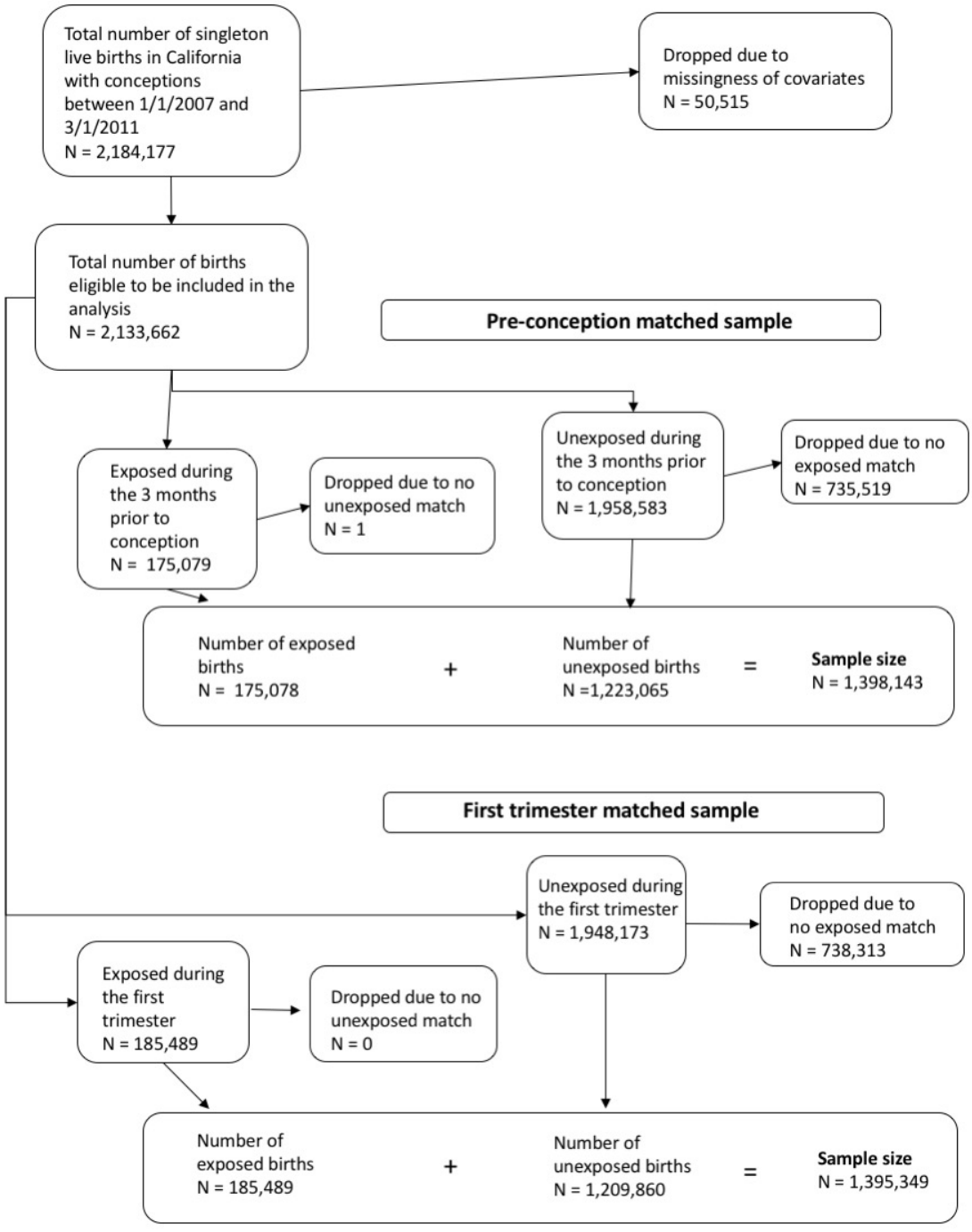


Figure 2.2: Illustration of the creation of the matched samples for the pre-conception and first trimester analyses.

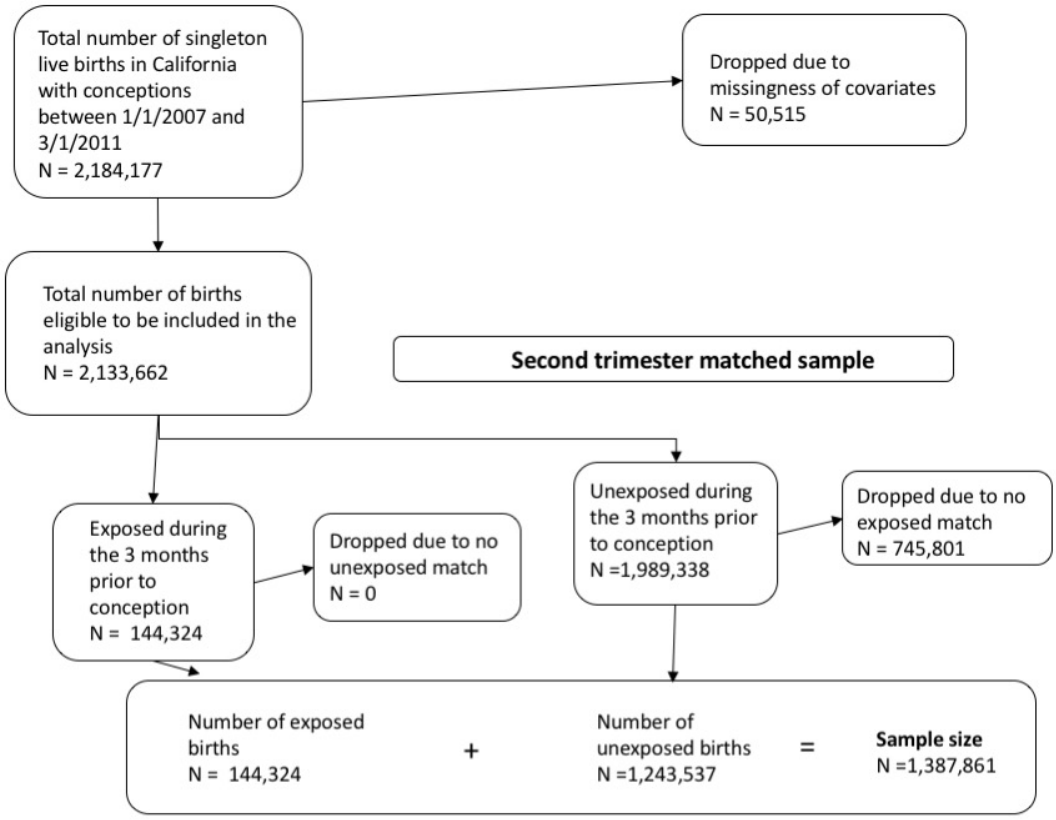


Figure 2.3: Illustration of the creation of the matched samples for the second trimester analyses.

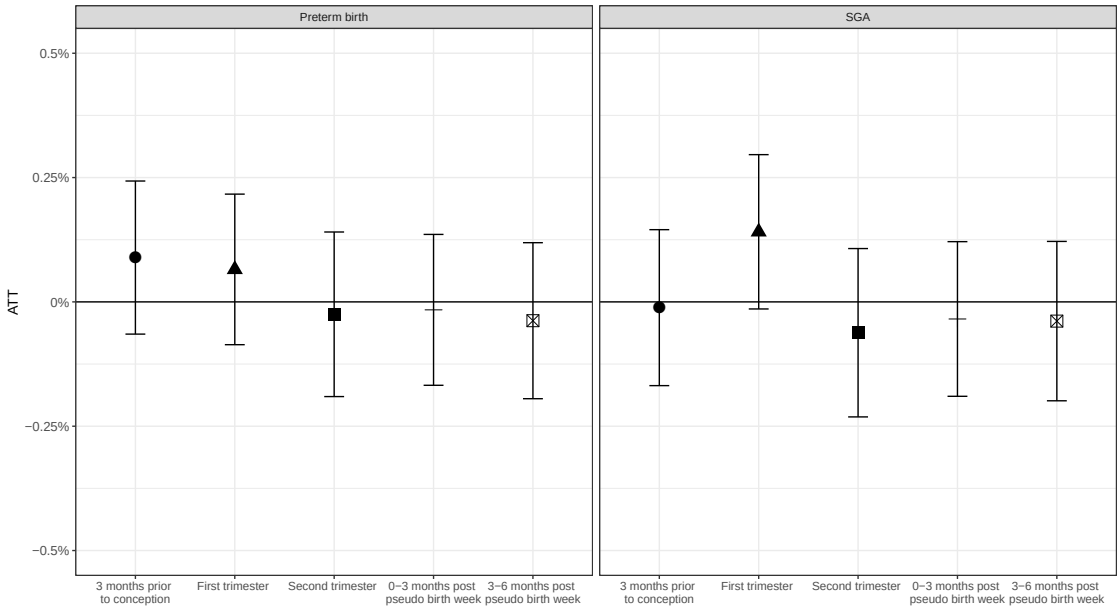


Figure 2.4: Estimated average treatment effect on the treated (ATT) of homicide exposure during the pre-conception period (1-12 weeks before conception), first trimester (weeks 0-12), second trimester (weeks 13-22), and the negative controls (0-12 weeks and 13-24 weeks after the pseudo-birth week) on risk of preterm birth and SGA in California, 2007-2011.

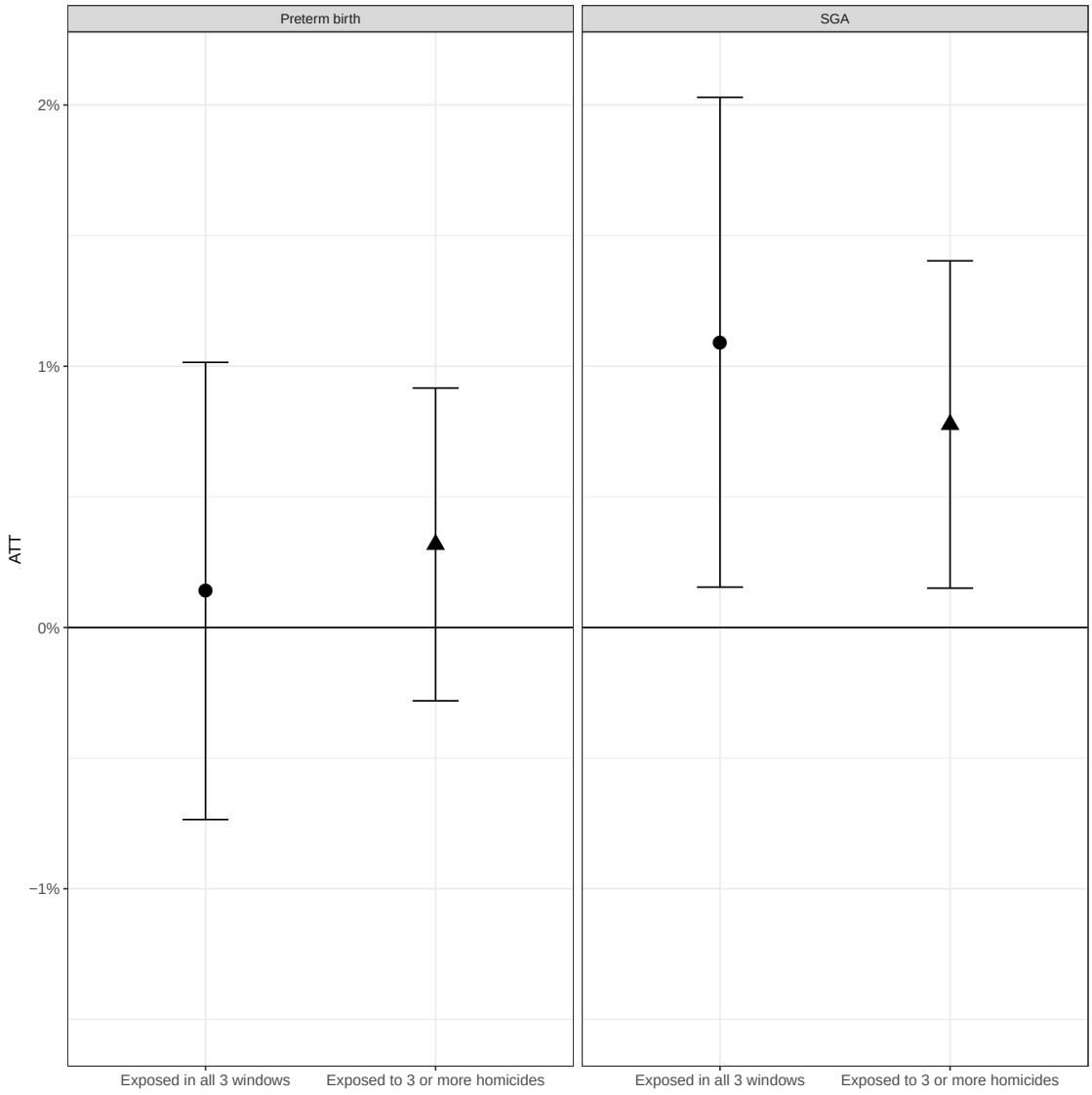


Figure 2.5: Estimated average treatment effect on the treated (ATT) of homicide exposure during all three exposure windows and exposed to at least three homicides on risk of preterm birth and SGA in California, 2007-2011.

2.5 Supplemental Material

Randomization inference

Randomization inference was used as an additional check to assess the likelihood of our results occurring due to random error. Randomization inference is a type of permutation test in which the exposure values are randomly shuffled across individuals, allowing us to compare the observed association with the distribution of associations that occur due to chance [115]. We performed the randomization inference by permuting the homicide exposure values within Census tracts, rerunning the weighting and regression analysis, and recording the t-statistic. This was done 1,000 times to generate a distribution of the t-statistic under the null hypothesis of no relationship between homicide exposure and adverse birth outcomes. We then compared the observed t-statistic to the null distribution to assess the likelihood that our results could be attributed to random error.

Exposure period	Outcome	P-value from randomization inference
Pre-conception period	Preterm birth	0.11
	SGA	0.57
First trimester	Preterm birth	0.18
	SGA	0.04
Second trimester	Preterm birth	0.63
	SGA	0.75
Exposure to three or more homicides during the pre-conception period, first trimester, and second trimester	Preterm birth	0.15
	SGA	0.01
Exposure to homicide in the pre-conception period, the first trimester, and the second trimester	Preterm birth	0.37
	SGA	0.02

Table 2.2: Randomization inference p-values for each outcome and exposure window.

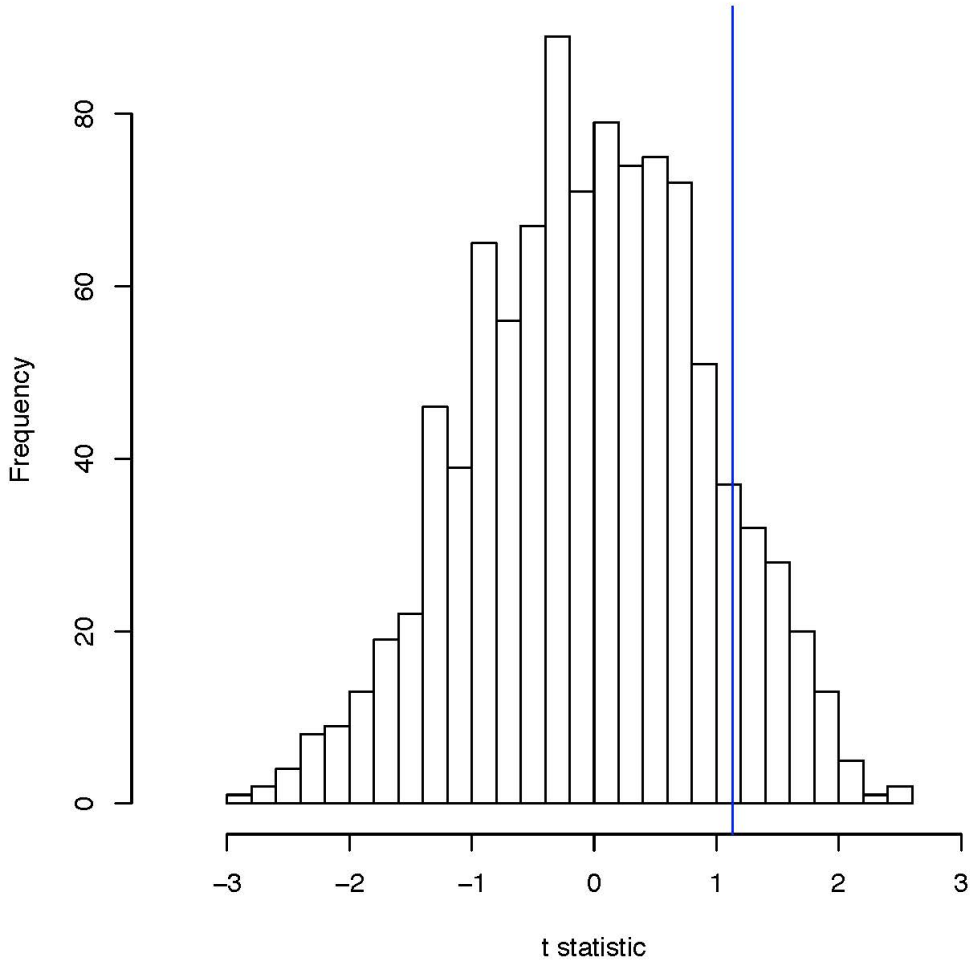


Figure 2.6: Randomization inference for homicide exposure during the pre-conception period on risk of preterm birth.

Note: The blue vertical line is the t statistic for the observed association of pre-conception homicide exposure and risk of preterm birth.

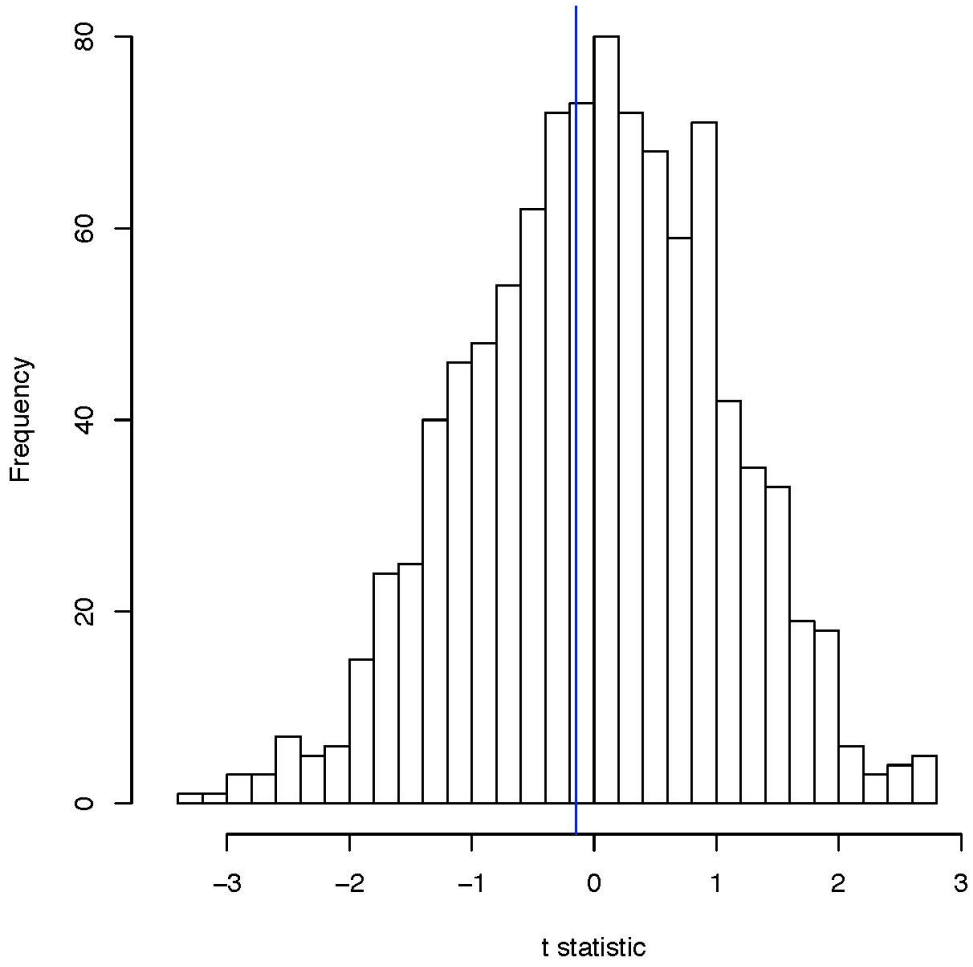


Figure 2.7: Randomization inference for homicide exposure during the pre-conception period on risk of SGA.

Note: The blue vertical line is the t statistic for the observed association of pre-conception homicide exposure and risk of SGA.

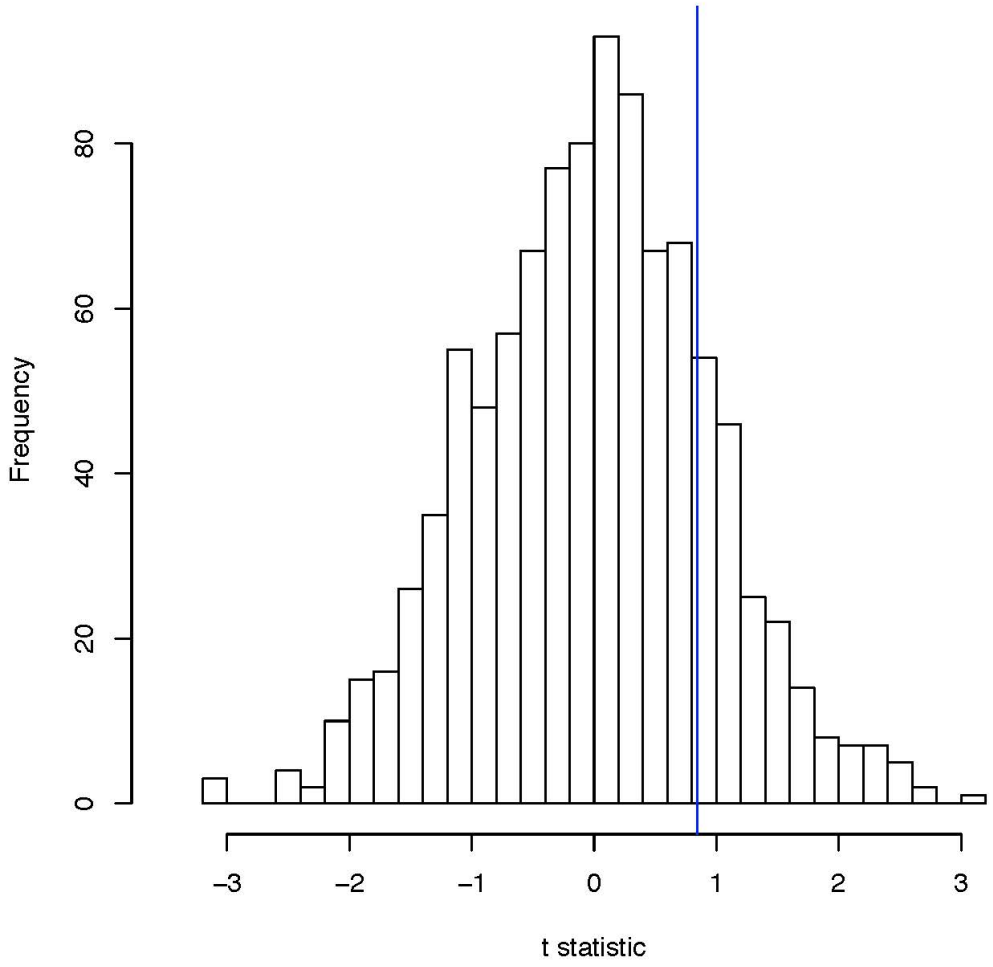


Figure 2.8: Randomization inference for homicide exposure during the first trimester on risk of preterm birth.

Note: The blue vertical line is the t statistic for the observed association of first trimester homicide exposure and risk of preterm birth.

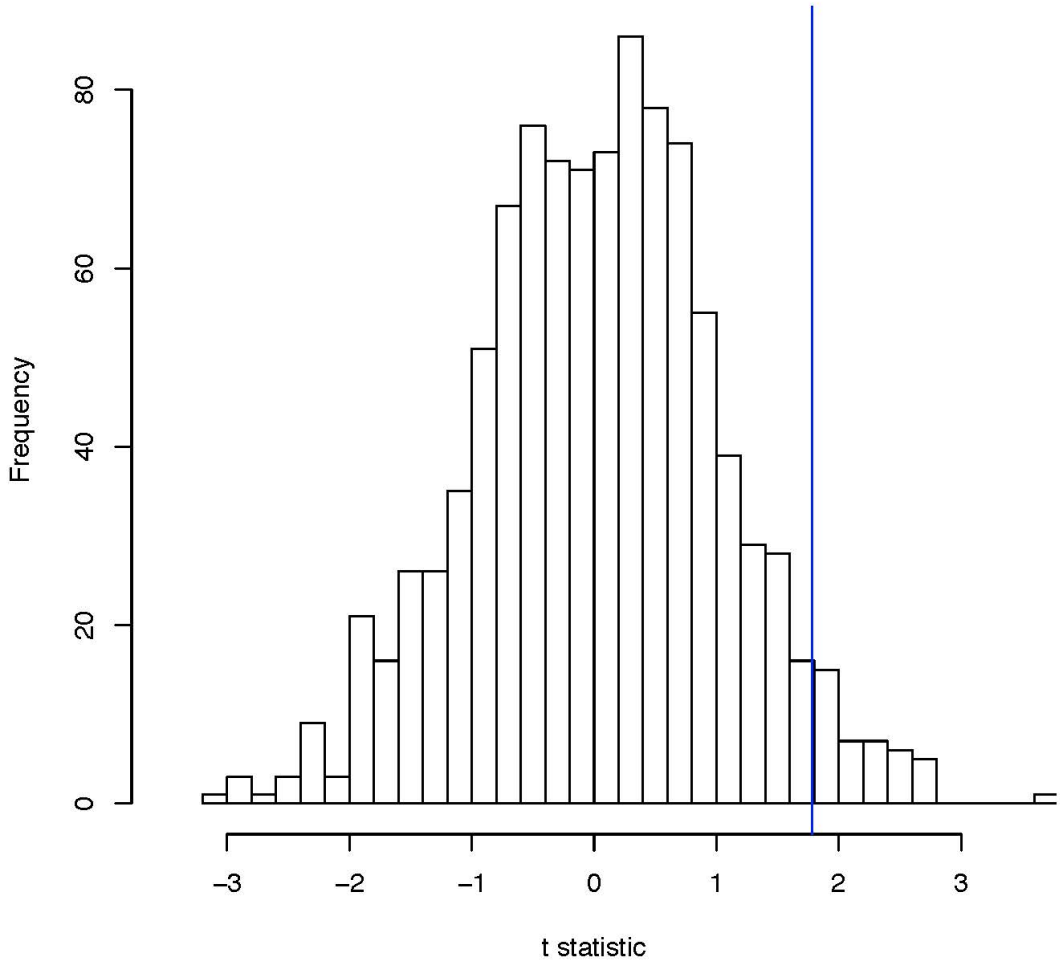


Figure 2.9: Randomization inference for homicide exposure during the first trimester on risk of SGA.

Note: The blue vertical line is the t statistic for the observed association of first trimester homicide exposure and risk of SGA.

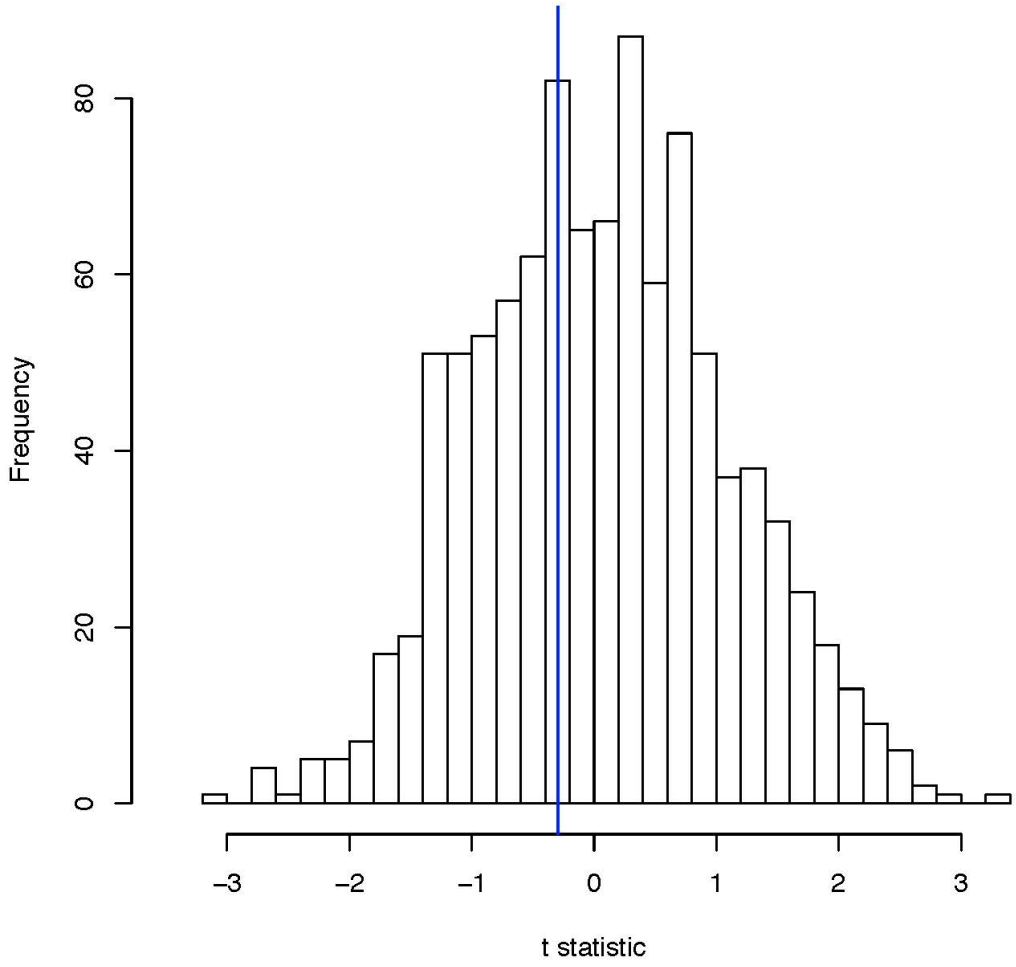


Figure 2.10: Randomization inference for homicide exposure during the second trimester on risk of preterm birth.

Note: The blue vertical line is the t statistic for the observed association of second trimester homicide exposure and risk of preterm birth.

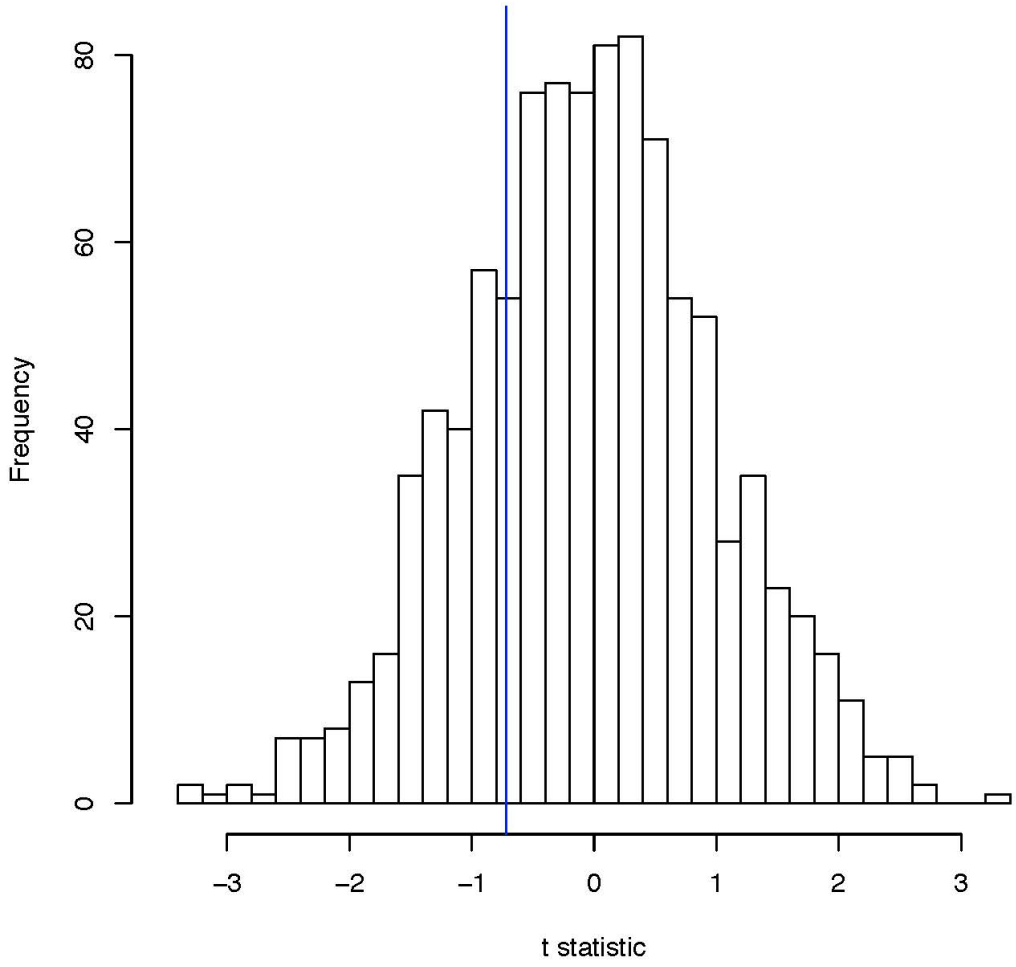


Figure 2.11: Randomization inference for homicide exposure during the second trimester on risk of SGA.

Note: The blue vertical line is the t statistic for the observed association of second trimester homicide exposure and risk of SGA.

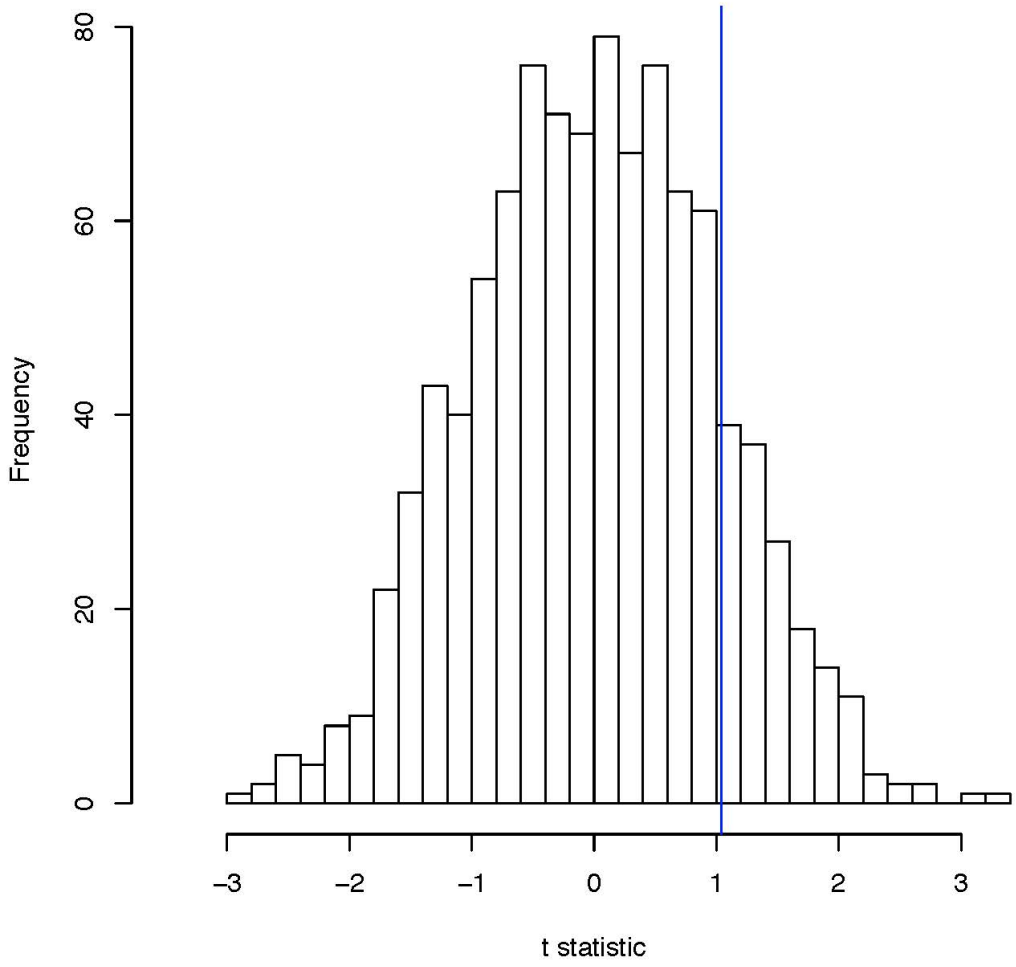


Figure 2.12: Randomization inference for exposure to three or more homicides during the pre-conception period, first trimester, and second trimester on risk of preterm birth.

Note: The blue vertical line is the t statistic for the observed association of exposure to three or more homicides during the pre-conception period, first trimester, and second trimester and risk of preterm birth.

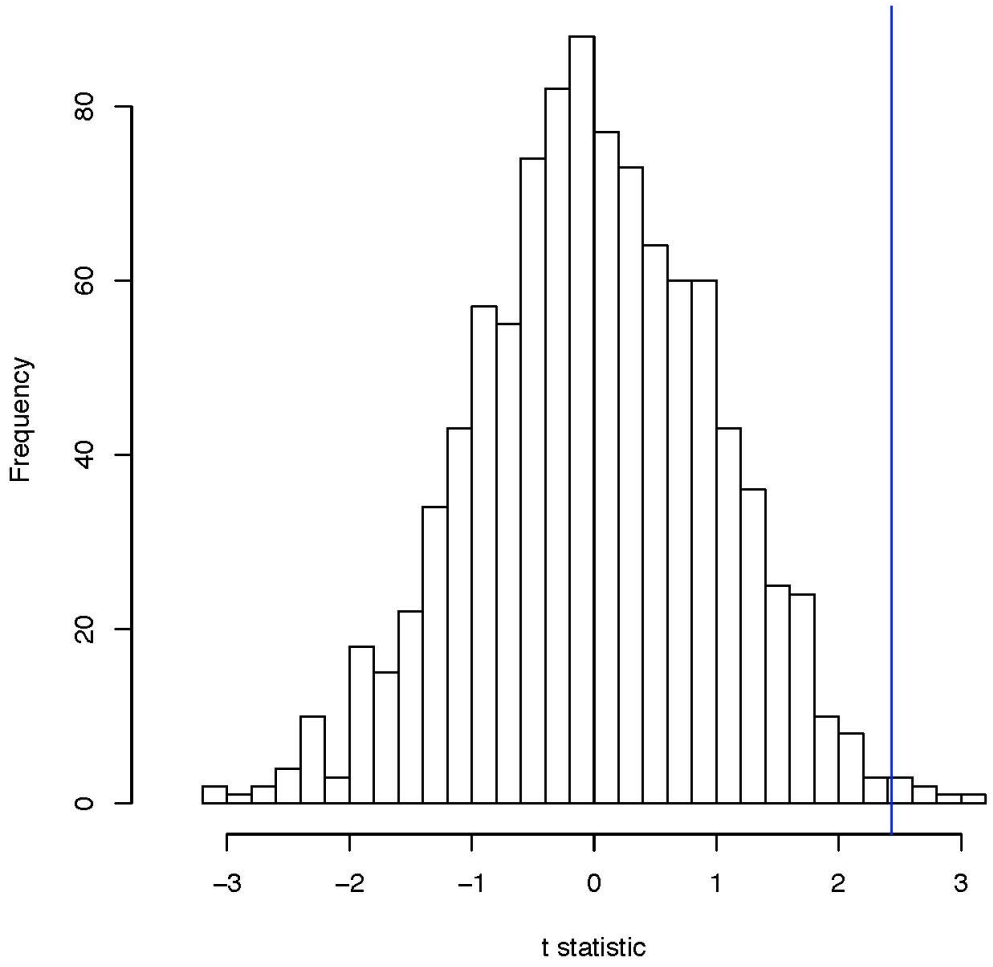


Figure 2.13: Randomization inference for exposure to three or more homicides during the pre-conception period, first trimester, and second trimester on risk of SGA.

Note: The blue vertical line is the t statistic for the observed association of exposure to three or more homicides during the pre-conception period, first trimester, and second trimester and risk of SGA.

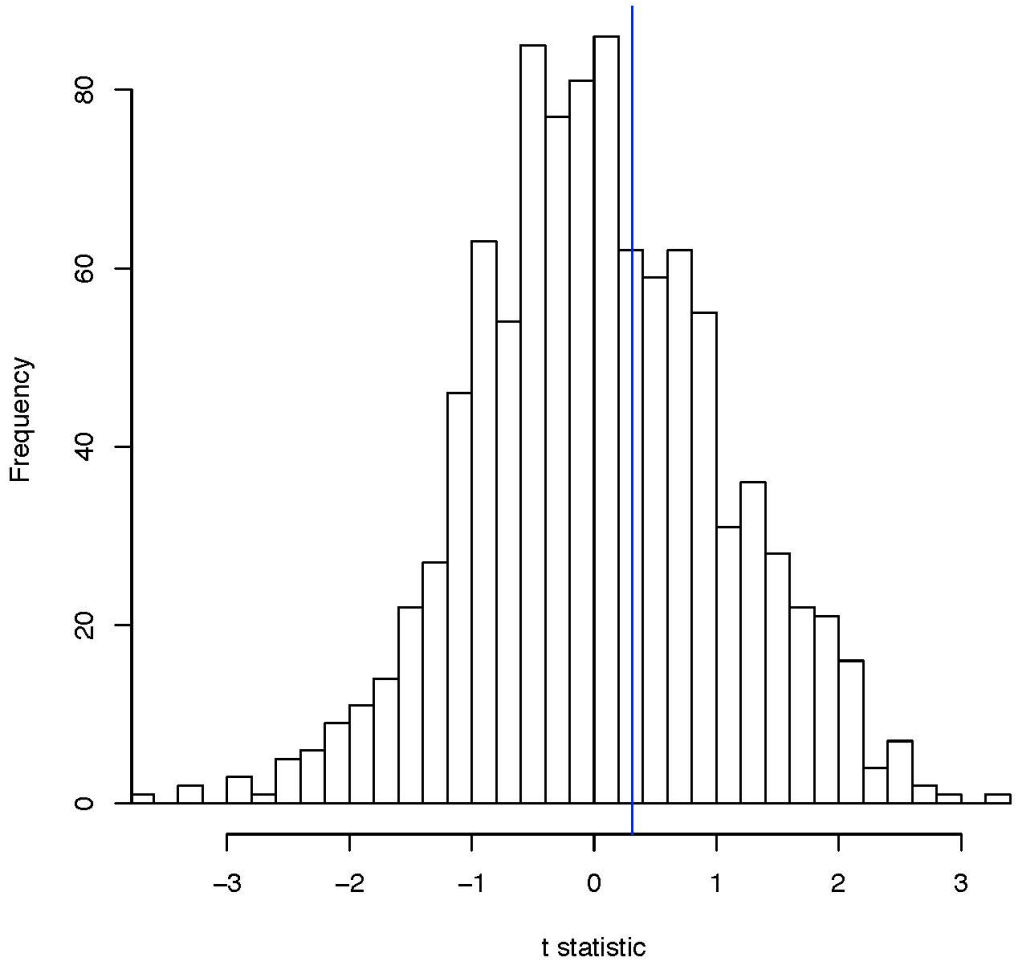


Figure 2.14: Randomization inference for exposure during the pre-conception period, first trimester, and the second trimester on risk of preterm birth.

Note: The blue vertical line is the t statistic for the observed association of exposure to homicides during all three exposure windows and risk of preterm birth.

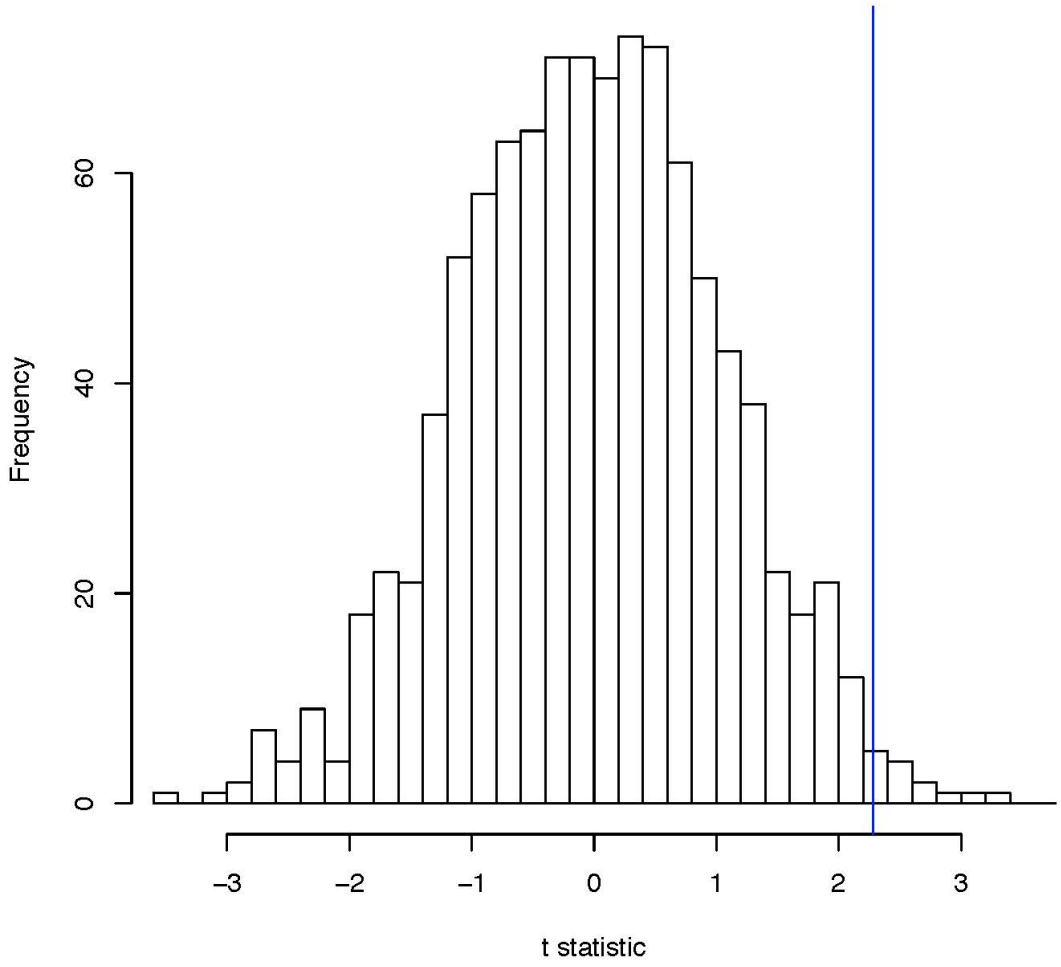


Figure 2.15: Randomization inference for exposure during the pre-conception period, first trimester, and the second trimester on risk of SGA.

Note: The blue vertical line is the t statistic for the observed association of exposure to homicides during all three exposure windows and risk of SGA.

Sensitivity analysis results

Due to the high level of missingness of education attainment in the birth record, we excluded it as a covariate from the main analysis. However, we reran our results while controlling for educational attainment, with a missingness indicator.

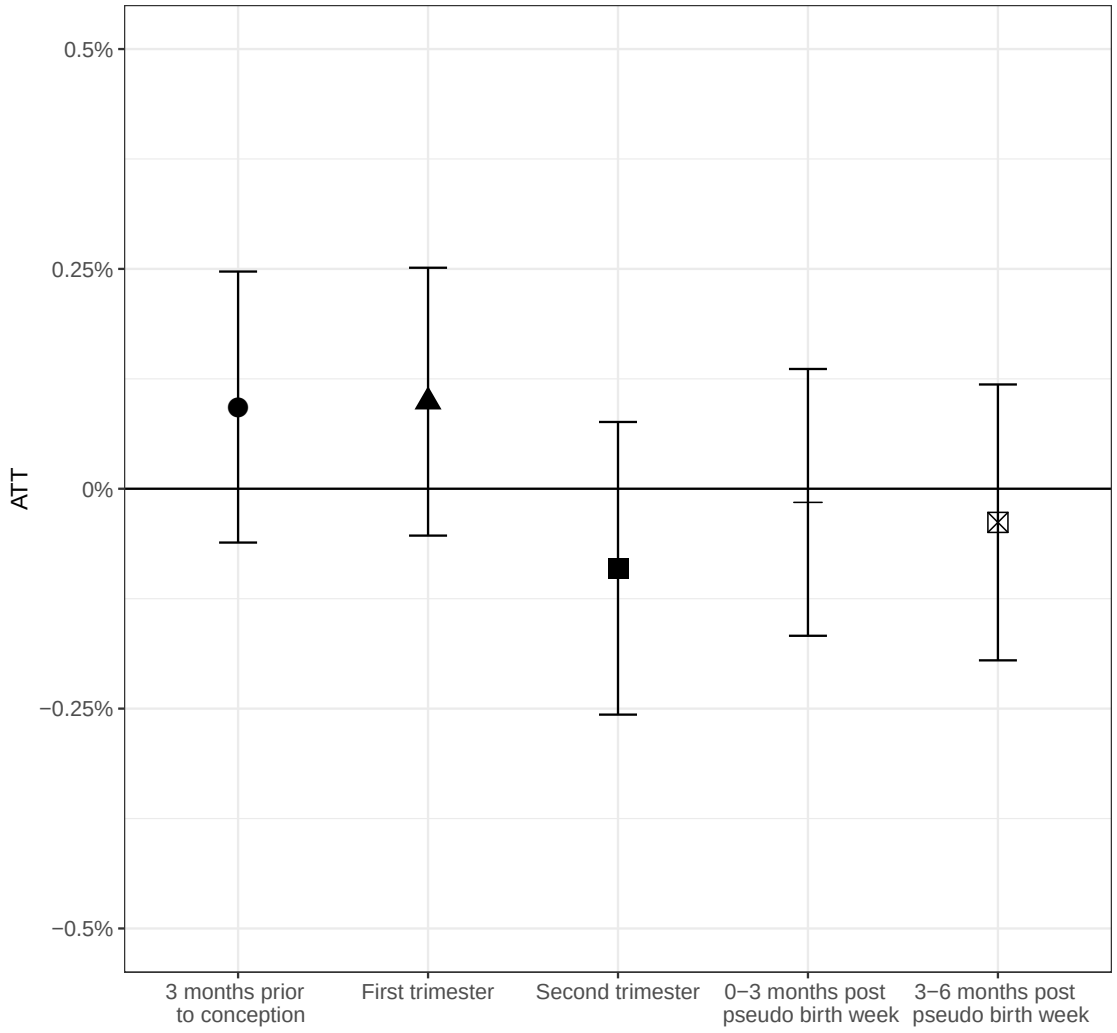


Figure 2.16: Estimated average treatment effect on the treated (ATT) of homicide exposure (adjusting for educational attainment with a missing category) on risk of preterm birth during the pre-conception period (1-12 weeks before conception), first trimester (weeks 0-12), second trimester (weeks 13-22), and the negative controls (0-12 weeks and 13-24 weeks after the pseudo-birth week) in California, 2007-2011.

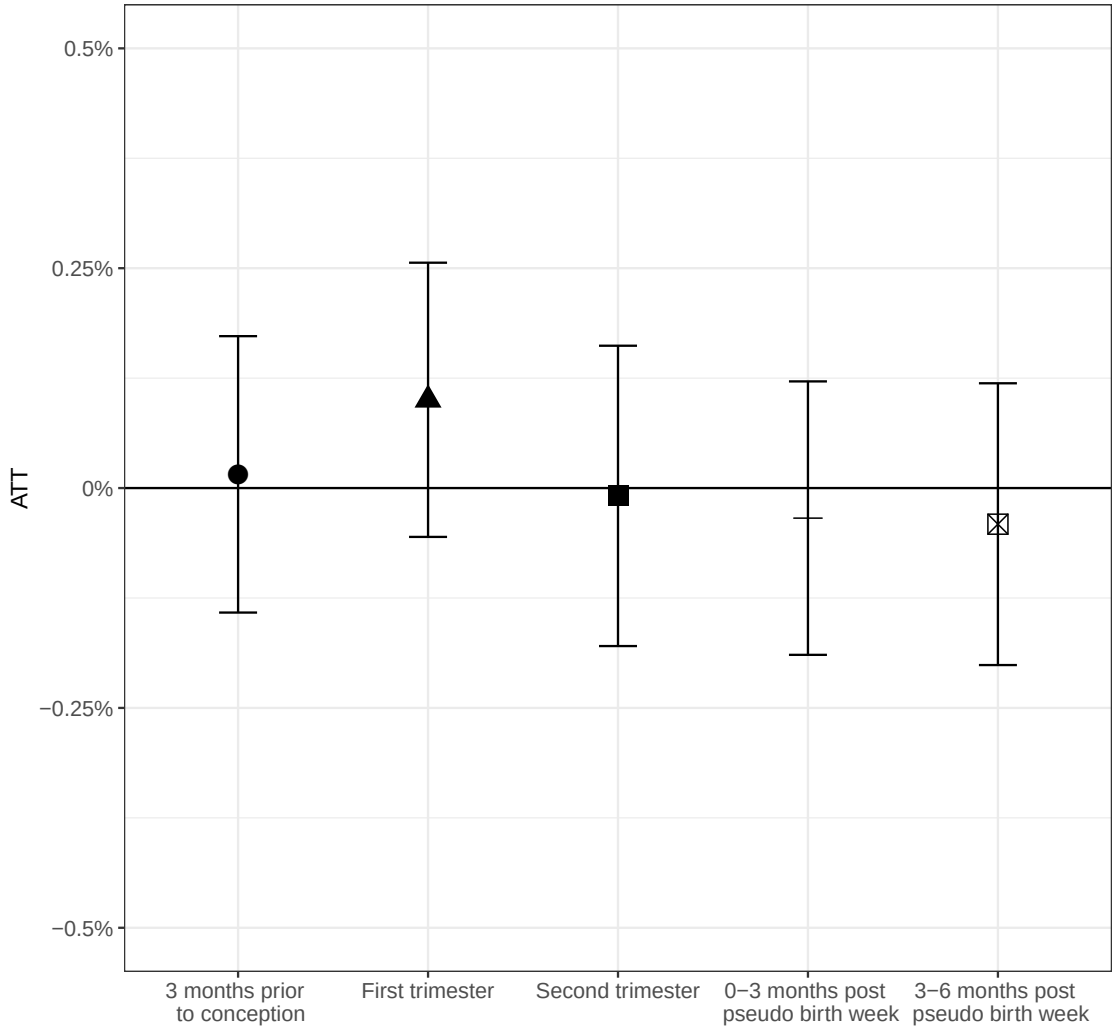


Figure 2.17: Estimated average treatment effect on the treated (ATT) of homicide exposure (adjusting for educational attainment with a missing category) on risk of SGA during the pre-conception period (1-12 weeks before conception), first trimester (weeks 0-12), second trimester (weeks 13-22), and the negative controls (0-12 weeks and 13-24 weeks after the pseudo-birth week) in California, 2007-2011.

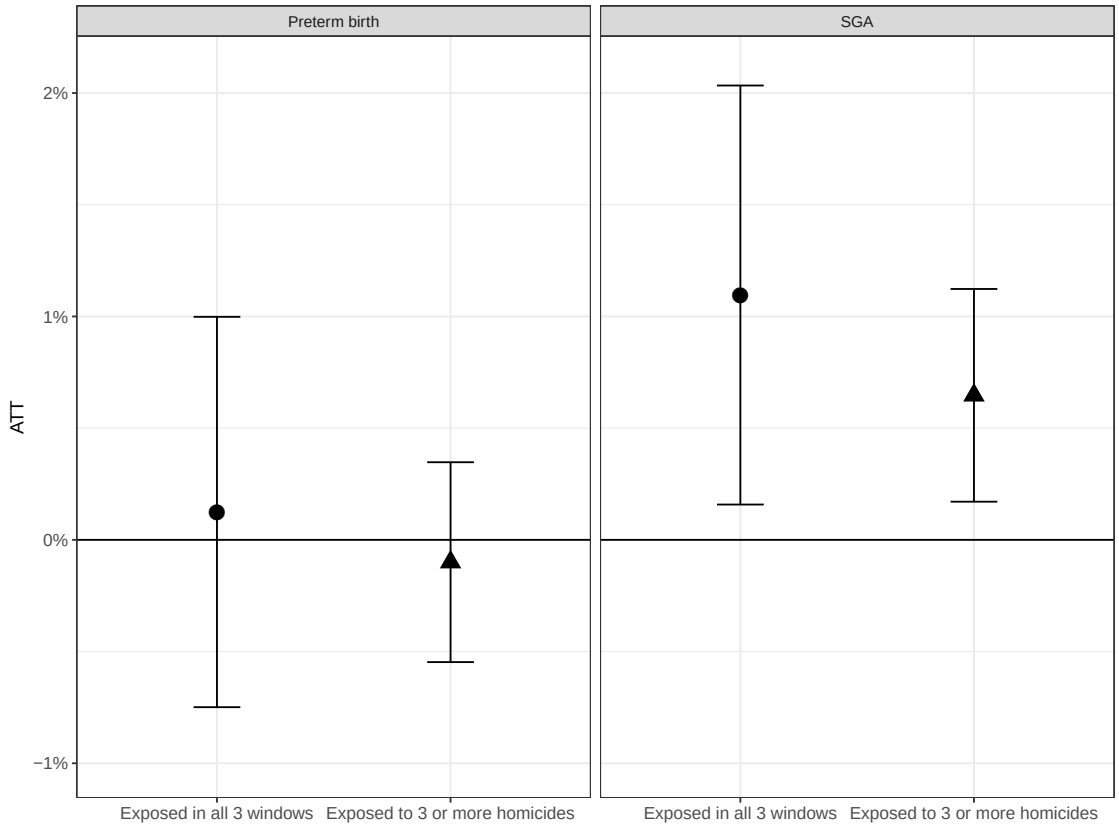


Figure 2.18: Estimated average treatment effect on the treated (ATT) of homicide exposure during all three exposure windows and exposed to at least three homicides (adjusting for educational attainment with a missing category) on risk of preterm birth and SGA in California, 2007-2011.

Chapter 3

Firearm Violence and Preterm Birth

3.1 Introduction

Firearm violence is a persistent problem in neighborhoods across the United States. In addition to causing premature death and significant costs from immediate and long-term health care needs, exposure to firearm violence can also affect health in the community overall [116–118]. Experiencing firearm violence in ones community can increase risks of stress-responsive diseases [116] and may influence birth outcomes through similar stress mechanisms [119]. In particular, exposure to high levels of firearm violence before and during pregnancy may increase detrimental health behaviors or have direct physiological effects on maternal health, which can both increase risk of preterm birth [120–125]. Accordingly, previous research has found evidence that exposure to community violence is associated with increased risk of adverse pregnancy outcomes [119, 126].

However, structural confounding is a substantial challenge when studying firearm violence exposure and pregnancy outcomes. Due to historical policies of residential segregation and economic exclusion [127], communities that have high levels of violence also tend to have other features of social and economic inequality, such as elevated poverty, fewer economic opportunities, and reduced educational attainment. Disentangling these factors is difficult to accomplish using statistical adjustment because they are so correlated [5]. Some previous studies have attempted to circumvent this issue by leveraging within-place variation in violence over time [101], rather than comparing across communities. However, this approach typically focuses on acute violence changes, and chronic violence is expected to have a much greater impact on health [45].

Chronic exposure to stressors like firearm violence may have physiologic consequences that manifest over time as pregnancy complications [128]. For example, studies have shown that chronic exposure to stressors like violence can dysregulate immune function [54], making pregnant women more susceptible to infection [19, 56]. Evidence also suggests that altered immune function may be linked to preeclampsia [63, 64] and asthma [65, 66]. In addition, chronic firearm violence exposure could increase the likelihood that women adopt coping

mechanisms for their own emotional well-being that may have negative health effects on pregnancy outcomes. For example, women may alter diet, exercise, substance use, or sexual risk-taking in response to stress from exposure to violence. These behavior modifications may have implications for maternal and fetal health, and understanding their role in pregnancy can help researchers better understand firearm violence as a potential driver of disparities in preterm birth. For example, risk factors for gestational diabetes include high maternal weight, poor diet, low exercise, and stress [129]. Violence exposure has previously been tied to altered health behaviors including increased sexual risk-taking [34, 130, 131]and substance use [33, 132–135].

In this study, we examined the association between community firearm violence exposure and risk of preterm birth. To address issues of structural confounding, we used machine learning to predict the propensity of neighborhoods to have high firearm violence and restricted our analysis to neighborhoods with similar propensities in the two exposure groups. We also investigated possible mediators of the relationship between firearm violence and preterm delivery, to identify potential mechanisms and improve our understanding of how features of the social environment, such as violence, can influence preterm birth.

3.2 Data and Methods

We used birth records linked to emergency department, hospitalization, and ambulatory surgery records during pregnancy; emergency department, hospitalization, and death records; and Census data from 2007 to 2011 to estimate the association of living in a neighborhood with high firearm violence with preterm birth in California. In order to avoid fixed cohort bias, we removed any births whose conception week was before January 1, 2007 or after March 1, 2011 [104]. We defined preterm birth as delivery of a live singleton birth before 37 weeks gestation based on the clinical estimate of gestational age in the birth record. We separated the analyses into spontaneous versus indicated preterm delivery based on ICD-9 codes for premature rupture of membranes or premature labor in the linked hospitalization and birth files (Supplemental Table 1). Individual-level covariates obtained from the birth record included maternal age, maternal age-squared to account for non-linearities in the effects of age, educational attainment, race/ethnicity, health insurance type, and the month-year of conception.

Neighborhood-level interpersonal firearm violence rates (hereafter referred to as firearm violence) were calculated at the Zip Code Tabulation Area (ZCTA) level using emergency department and hospitalization records from Californias Office of Statewide Health Planning and Development (OSHPD) and death records from Vital Statistics. Incidents of firearm violence were defined as any firearm related assault or fatality that was recorded in the emergency department, hospitalization, or mortality data. Each incident was geocoded based on the victims home address at the time of emergency department visit, hospitalization, or death. The nonfatal injury records from OSHPD were classified using ICD-9 codes while the fatal records used ICD-10 (the list of codes is available in Supplemental Table 1). Any

injury that presented to the emergency department and then transferred to a hospital will only be recorded in the hospitalization files, and any injury that ultimately proved to be fatal was only captured in the mortality data, so there is no double-counting of incidents across the data sources. The firearm violence rate was calculated by dividing the total number of firearm violence deaths and injuries by the estimated population over the 5-year period. Neighborhoods were classified as high firearm violence if the average firearm violence rate was greater than 50 per 100,000 people. This cutoff was created by visual inspection of the distribution of firearm violence rates. As a sensitivity analysis, we replicated the analysis using the 90th percentile of violence rates as the cutoff for defining high violence neighborhoods.

Neighborhoods with high levels of firearm violence also tend to suffer from other forms of social inequity, such as high poverty, low economic investment, and histories of racial and economic segregation [127], thus it was important to rigorously control for neighborhood-level factors that were associated with firearm violence and could influence risk of preterm birth. Therefore, we used a set of community covariates that have been shown to optimally predict firearm violence levels in California [136]. The variables used in the predictor set included measures related to residential segregation, poverty, educational attainment, social factors, and environmental characteristics; the full set is described in Supplemental Table 2. The measured were obtained from the 2007-2011 ZCTA-level measures from the American Community Survey, Census TIGER files, and PRISM. Exhaustive ZCTA-level covariates are only available in the 5-year ACS files, and therefore we used the same years to characterize firearm violence levels.

Pregnancy complications and health behaviors were examined as potential mediators. We identified pregnancy complications as adverse events or comorbidities that occurred during pregnancy, which may plausibly be affected by firearm violence and have been associated with increased risk of preterm birth in previous studies [137–140]. The pregnancy complications and health behaviors we examined were early onset preeclampsia, gestational diabetes, substance use, asthma, and infection, and were identified by ICD-9 codes available in the linked hospitalization records (see Supplemental Table 1). We focused on early-onset preeclampsia (diagnosis before 34 weeks gestation) because previous literature suggests late-onset preeclampsia may have distinct etiology [141]. We determined the timing of preeclampsia based on the date of the first record that indicated a preeclampsia diagnosis during pregnancy. The ICD-9 codes used to identify each complication were selected based on prior research that used linked birth and hospitalization records to identify complications [140, 142–149]. We coded each live birth as being associated with a pregnancy complication if at least one emergency department, hospitalization, or ambulatory surgery record for the mother existed during the pregnancy and had a diagnosis code indicating the presence of the complication.

Mediation analyses that estimate indirect effects present difficulties in questions like this because one key assumption of most methods is no mediator-outcome confounder affected by prior exposure (MOCAPE). However, chronic exposure to firearm violence may influence health behaviors and/or chronic conditions that both increase the likelihood of pregnancy

complications and may affect preterm delivery. This violation of the no MOCAPE assumption has required previous mediation analyses of social exposures and pregnancy outcomes to focus on controlled direct effects, in which the mediator values are set to the same level for everyone, blocking the mediated pathway [17, 139]. These effects are often difficult to interpret given that our understanding of why women develop complications is incomplete, and therefore setting the complication status to a certain value for all women is infeasible in reality. Furthermore, if the goal is to understand the indirect pathway, that is, the extent to which the exposure influences the outcome through the mediator, then estimating controlled direct effects are insufficient. To address this issue, we estimated stochastic direct and indirect effects, which allow us to estimate indirect effects, rely on fewer assumptions, and have the option of including post-exposure mediator-outcome confounders.

Previous literature has suggested preexisting chronic conditions are important to consider when evaluating the relationship between pregnancy complications and risk of preterm birth [150]. Living in a neighborhood with high firearm violence could affect diet and exercise habits or induce stress-related coping behaviors that affect weight gain or increase the prevalence of certain chronic conditions [78], so we identified pre-pregnancy BMI, pre-existing hypertension, and pre-existing diabetes as potential MOCAPE variables. Measures of pre-pregnancy BMI, hypertension, and diabetes that pre-existed pregnancy came from the same linked hospitalization and birth record files used to identify pregnancy complications and preterm delivery. Hypertension and diabetes were identified using ICD-9 codes (Supplemental Table 1). More discussion of these variables and their role in the analysis is in the statistical analysis section.

This study was reviewed and approved by the California Health and Human Services Agency Committee for the Protection of Human Subjects, and the University of California, Berkeley Committee for the Protection of Human Subjects.

Statistical analysis

Propensity score estimation for neighborhood-level firearm violence

We estimated a propensity score to identify neighborhoods with similar characteristics but differing levels of firearm violence. We used SuperLearner, an ensemble machine learning algorithm [151], along with the set of neighborhood covariates mentioned previously, to estimate the propensity for a neighborhood to be classified as high violence (see the supplemental material for a list of all algorithms included in the SuperLearner). Neighborhoods were included in the analysis if the propensity score fell between the 2.5th percentile of the values for neighborhoods that were observed to have high violence, and the 97.5th percentile of values for neighborhoods that were not observed to have high violence (the area of support), in accordance with prior recommendations [152]. We tested whether covariate balance was further improved by weighting by the odds of the propensity score among unexposed neighborhoods.

Estimation of total effects

We used targeted maximum likelihood estimation (TMLE) to estimate absolute differences in risk of preterm delivery comparing women living in high firearm violence and comparison communities [153]. We estimated the outcome and the exposure model using logistic regression. TMLE is a double-robust substitution estimator, meaning that if the exposure or the outcome model is correct, then the estimate will be consistent. In this application, since we have already restricted to neighborhoods with similar propensities for high firearm violence, the exposure model captures the individual-level characteristics associated with living in a high firearm violence neighborhood. To account for the clustering of women within ZCTAs, we used a non-parametric clustered bootstrap with Wald-style confidence intervals for inference.

Estimation of stochastic direct and indirect effects

To assess mediation of firearm violence and preterm birth by pregnancy complications and health behaviors, our goal was to estimate direct and indirect effects. Because we believed the assumption of no MOCAPE was violated in our understanding of the data-generating mechanism (Figure 3.1), we opted to estimate stochastic direct and indirect effects [154, 155], which only relies on the following independence assumptions: no unmeasured confounding of the exposure and outcome, and no unmeasured confounding of the mediator and outcome.

We define our variables as follows: Y is preterm birth; A is exposure to high firearm violence; M is a pregnancy complication or health behavior during pregnancy; Z is pre-pregnancy BMI, hypertension, and diabetes; and W is the set of individual covariates described above. Y_a is the counterfactual outcome if $A = a$, and Y_{a^*} is the counterfactual outcome if $A = a^*$. $Y_{a,m}$ is the counterfactual outcome if $A = a$ and $M = m$. The required confounding assumptions to identify stochastic direct and indirect can be written as:

$$A \perp Y_{a,m}|W \qquad\qquad A \perp M_a|W$$

$$M \perp Y_{a,m}|W, A = a, Z$$

The following positivity assumptions are also required: a positive probability of attaining each value of the exposure and mediators within covariate strata.

$$0 < P(M = m|A = a, W) < 1 \qquad\qquad 0 < P(A = a|W) < 1$$

The stochastic direct effect of a change in exposure from a to a^* is defined as

$$SDE = E(Y_{a,g_{M|a^*,W}} - Y_{a^*,g_{M|a^*,W}})$$

And the stochastic indirect effect of a change in the mediator distribution from $g_{M|a,W}$ to $g_{M|a^*,W}$ is defined as

$$SIE = E(Y_{a,g_{M|a,W}} - Y_{a,g_{M|a^*,W}})$$

Where $g_{M|A,W}$ is the observed distribution of the mediator conditional on covariates W and exposure A. By drawing from the distribution of mediator values rather than assigning a mediator value based on what we would expect it to be under different exposure values, we are able to identify direct and indirect effects (albeit with different interpretations) without needing to invoke the controversial cross-world assumption and can allow for post-exposure mediator-outcome confounders [156].

The stochastic direct effect compares the expected proportion of preterm births in high firearm violence neighborhoods compared to low violence neighborhoods, if the distribution of pregnancy complications and health behaviors in both types of neighborhoods were drawn from the distribution of pregnancy complications and health behaviors in the low violence neighborhoods. The stochastic indirect effect compares the expected proportion of preterm births in high violence neighborhoods, contrasting the distribution of pregnancy complications and health behaviors if they were drawn from the observed distribution in high violence neighborhoods to the distribution of pregnancy complications and health behaviors if they were drawn from that observed in low violence neighborhoods.

Statistical analyses were completed using R version 3.5 [157], and code is provided in the supplemental material.

3.3 Results

There were 2,084,417 mothers eligible to be included in the study who conceived between January 1, 2007 and March 1, 2011, delivered a single live birth, lived in California at the time of delivery, and had information on gestational age included in their birth or hospitalization record. We excluded women missing information on maternal race (0.1%), education (3.4%), health insurance type (2.6%), age (0.00005%), and pre-pregnancy BMI (7.5%). Due to computational limitations, we were unable to perform multiple imputation or other missing data procedures. Therefore, our analysis included 1,815,245 (87.1% of all eligible) mothers (Table 1).

The majority of ZCTAs had firearm violence rates below 50 per 100,000 (Figures 3.2 and 3.3). The propensity scores for high firearm violence exposure show that 194 (12.6%) ZCTAs had propensity scores within the range of support (Figures 3.4 and 3.5, and Supplemental Figure 1), reflecting the structural confounding. Limiting to this area of support corresponded to 314,986 women (Table 2 and Supplemental Figure 2). The location of high firearm violence ZCTAs and which ZCTAs are included in the analysis are shown in Figures 3.6, 3.7, and 3.8. Both the prevalence of preterm birth and the firearm violence rate were higher in the propensity-score restricted data compared to the full data (Table 2). Restricting the analyses to ZCTAs within the area of common support without weights resulted in

better covariate balance than weighting by the odds of the propensity score (Supplemental Table 2), and therefore we did not use weights in the subsequent analyses.

In the propensity-score restricted data, the firearm violence rate was approximately two times higher in the high violence neighborhoods compared to the lower violence neighborhoods, and the prevalence of preterm birth was about 0.6% higher among women who lived in a high violence neighborhood (7.1% versus 7.7%) (Table 2). Prevalence of early preeclampsia and gestational diabetes were almost 0.1% higher in high violence neighborhoods, and asthma and substance use were both 0.8% and 0.9% higher, respectively. Prevalence of infection was 2.1% higher among women living in high violence neighborhoods. The adjusted first stage effects show that the largest associations of pregnancy complications or health behaviors with high neighborhood violence were observed for infection, asthma, and substance use (Table 3). Furthermore, living in a neighborhood with high firearm violence was associated with increased risk of diabetes and higher pre-pregnancy BMI. The adjusted total effects show that women living in high violence neighborhoods had 0.49% (95% CI: 0.27%, 0.71%) higher prevalence of preterm birth, and the majority of this association was driven by higher prevalence of spontaneous preterm birth (Table 3).

The mediation results showed a direct effect of high firearm violence on risk of preterm birth, with stronger associations for spontaneous compared to indicated preterm deliveries (Figure 3.9). The stochastic indirect effects had consistent patterns across spontaneous and indicated preterm birth (Figure 3.10), although the results were strongest among spontaneous births. The largest mediation effects were observed for infection (SIE = 0.04% [95% CI 0.03%, 0.04%]), early preeclampsia (SIE = 0.03% [95% CI 0.03%, 0.03%]), and substance use (SIE = 0.06% [95% CI 0.04%, 0.07%]). While the magnitudes of the indirect effects are small, the proportion of the effect mediated is not trivial. For example, approximately 11% of the association between high firearm violence and preterm birth was mediated by substance use. About 6% of the association was mediated by early preeclampsia, and 8% by infection.

The results of the sensitivity analysis using the 90th percentile of firearm violence rates as the cutoff are very similar to those presented here (results are available in the supplemental material).

3.4 Discussion

This study estimated the relationship between living in a neighborhood with high firearm violence and risk of preterm birth, and assessed whether pregnancy complications or health behaviors during pregnancy mediated this relationship. Our findings suggest firearm violence increases risk of preterm birth, especially spontaneous preterm birth, with the strongest mediation effects for infection and substance use. We also found associations of firearm violence with pre-existing diabetes, pre-pregnancy BMI, asthma, infection, and substance use during pregnancy. These results suggest living in a neighborhood with high firearm violence affects maternal health before and during pregnancy, with implications for infant health.

Our results are consistent with most previous research that analyzed the relationship between living in a violent neighborhood and gestational outcomes [5, 14, 18, 30, 99, 117, 126, 158, 159]. The findings are also consistent with previous studies linking community violence exposure with asthma, substance use, and increased risky sexual behaviors, although prior work has predominantly been among children and adolescents [32–34, 131–135, 160–163]. For example, a study of children in San Juan, Puerto Rico found increased odds of asthma among children exposed to firearm violence in their communities [163], and a study in Boston, Massachusetts found increased risk of asthma among children jointly exposed to traffic-related air pollution and violence [161]. A study of sexually active adolescent girls recruited from a clinic in Texas found that witnessing violence was associated with increased likelihood of having sex with strangers or engaging in sexual activity while under the influence of drugs or alcohol [131], and a study of college students found lifetime community violence exposure was associated with sexual risk taking and substance use [32].

Some previous evidence has also linked community violence with preeclampsia and other hypertensive disorders of pregnancy. A study of mothers in Chicago found increased odds of both preterm birth and hypertensive disorders of pregnancy with increases in the neighborhood crime rate [164]. Another study found reduced risk of preeclampsia among mothers who engaged in recreational physical activity before and during pregnancy [165], which may be relevant to women living in high violence neighborhoods as they may avoid outdoor physical activity if they feel unsafe. A study in North Carolina found aspects of neighborhood deprivation were associated with increased risk of preeclampsia including physical incivilities and poor walkability, although only among white women [30]. We found increased risk of early preeclampsia among women living in high violence neighborhoods, and early preeclampsia mediated a small portion of the association with preterm birth.

A few prior studies have investigated mediation effects related to violence and gestational outcomes, although most studies assessed violence as the mediator rather than the exposure [17, 166]. In addition, as discussed previously, the methods utilized in prior studies relied on several strong assumptions. One previous study investigated the pathways between the neighborhood riska measure of deprivationand birthweight in Baltimore, Maryland. The authors found increased neighborhood risk was associated with low birthweight, and determined that substance use was the strongest mediator of the association [167]. The Baron and Kenny method [168] was used to assess mediation, which assumes there is no interaction between the exposure and the mediator and does not allow for MOCAPE variables. Given our attention to the issue of structural confounding and our use of a mediation method that addresses limitations of past approaches, our findings strengthen the evidence relating violence exposure and maternal and infant health.

Our analysis improved on previous studies but nevertheless had several limitations. We dichotomized neighborhood-level firearm violence in order to facilitate propensity score matching, but a choice of cutoff had to be made amongst many options. In order to assess whether this choice influenced our results, we replicated the analysis using the 90th percentile of firearm violence as the cutoff, and found no meaningful differences in the findings. Furthermore, as this study used administrative data there may be several potential sources

of measurement error. In particular, firearm violence injuries and deaths were geocoded to the ZCTA in which the victim lived at the time of hospitalization or death, rather than where the shooting occurred. This may therefore mischaracterize the extent of exposure if people tend to be victims of gun violence in neighborhoods other than those in which they reside. Womens residences during pregnancy were assumed to be those listed on the birth certificate at delivery, which may have resulted in some exposure misclassification if women moved between high and low violence neighborhoods during pregnancy. Due to the nature of the linked birth and hospitalization records used in this analysis, we only had maternal address information at the ZCTA-level. Therefore, as any analysis that characterizes a neighborhood unit, our findings may be sensitive to the choice of the geographic unit, known as the modifiable area unit problem [112]. However, the ZCTA was the smallest unit we had access to and larger units would not have captured local dynamics in firearm violence. Pregnancy complications and health behaviors were identified based on the maternal hospitalization, emergency department, and ambulatory surgery records linked to the birth record. Complications identified only from routine prenatal appointments that were not recorded at the time of delivery were not captured, and therefore we expect this analysis captured the most severe instances of complications and behaviors. In addition, we excluded some neighborhoods with high levels of firearm violence because their propensity score did not fall within the area of support, thus there were no appropriate control units for these neighborhoods.

Firearm violence may be one mechanism by which histories of economic and racial segregation affect maternal health in the current day [127]. The unique history of these neighborhoods makes studying their characteristics a challenge for researchers. We attempted to improve upon prior literature by using machine learning and restricting to neighborhoods with similar characteristics and propensity to experience high firearm violence. Our results suggest firearm violence exposure is associated with preterm birth, and that this association is partially mediated by increased risk of infection, early preeclampsia, and substance use. Violence exposure was also related to diabetes, maternal pre-pregnancy BMI, and asthma, all of which have implications for maternal health during pregnancy and over the life course.

While reducing firearm violence may not be a feasible goal for reducing maternal and infant health disparities, at least in the short-term, it may be possible to develop interventions that provide social support and coping resources for women in communities with high violence. There is evidence to suggest that strong social support may buffer the impact of community violence exposure [135, 169]. Interventions to foster social groups and support in the prenatal care setting have had success in improving birth outcomes [170–172] and maternal health behaviors during pregnancy [173]. Doula care during pregnancy has also been shown to improve both maternal and infant health outcomes [174, 175]. To the extent that the association between firearm violence and preterm birth in our study is due to internalized stress or maladaptive coping behaviors, interventions to support women exposed to violence may be especially helpful in improving birth outcomes and maternal health in this population.

	Full data	Propensity score restricted data
N	1,815,245	314,986
Preterm birth	6.80%	7.30%
Firearm violence rate per 100,000	22.9	36.8
High firearm violence	11.10%	23.00%
Pregnancy complications		
Early preeclampsia	0.80%	0.90%
Asthma	3.20%	3.20%
Gestational diabetes	7.40%	7.40%
Substance use	1.50%	1.90%
Any infection	23.00%	25.70%
Race/ethnicity		
Non-Hispanic White	27.00%	11.90%
Non-Hispanic Black	5.20%	6.70%
Non-Hispanic Alaska Native or American Indian	0.30%	0.40%
Non-Hispanic Asian	12.30%	7.40%
Non-Hispanic Hawaiian or Pacific Islander	0.40%	0.40%
Other or two or more races	2.00%	1.40%
Hispanic	52.70%	71.80%
Educational attainment		
Less than high school	24.80%	38.50%
Less than college	50.20%	52.20%
College graduate or more	25.00%	9.30%
Age (in years)	28.2	26.7
Insurance type		
Private coverage	47.90%	29.00%
Public coverage	50.00%	69.00%
Indigent coverage	0.10%	0.10%
Self pay or other	2.10%	1.90%

Table 3.1: Comparison of preterm birth, violence rate, pregnancy complications, and demographics across full and propensity-score matched data

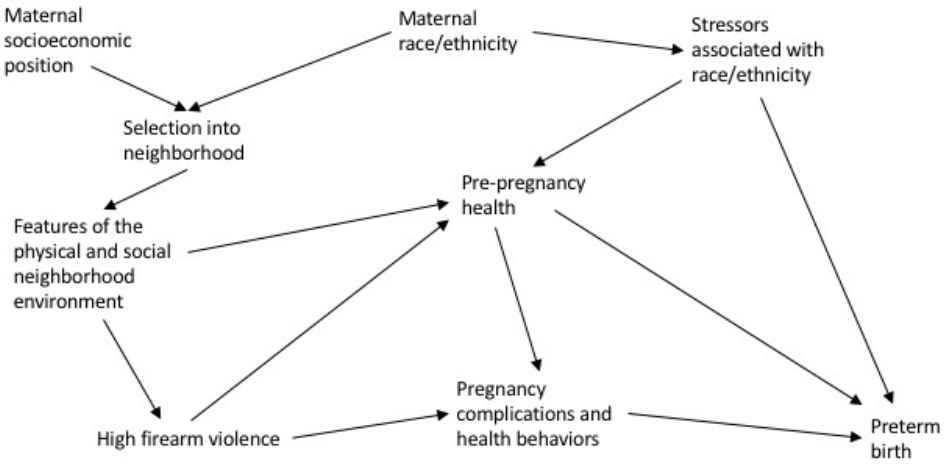


Figure 3.1: Conceptual diagram illustrating the relationships between firearm violence exposure, pregnancy complications and health behaviors, and preterm birth.

	Full data		Propensity score restricted data	
	Comparison	High violence	Comparison	High violence
N	1,612,944	202,301	242,565	72,421
Preterm	6.60%	7.90%	7.10%	7.70%
Firearm violence rate per 100,000	14.9	87.2	29.3	62.2
Pregnancy complications				
Early preeclampsia	0.80%	1.00%	0.80%	0.90%
Asthma	3.10%	3.70%	3.00%	3.80%
Gestational diabetes	7.40%	7.30%	7.40%	7.50%
Substance use	1.40%	2.20%	1.70%	2.50%
Any infection	22.50%	27.20%	25.20%	27.30%
Race/ethnicity				
Non-Hispanic White	29.40%	7.60%	12.20%	11.10%
Non-Hispanic Black	4.00%	15.00%	6.40%	7.70%
Non-Hispanic Alaska Native or American Indian	0.30%	0.30%	0.40%	0.40%
Non-Hispanic Asian	13.10%	5.90%	7.50%	6.80%
Non-Hispanic Hawaiian or Pacific Islander	0.40%	0.50%	0.40%	0.40%
Other or two or more races	2.10%	1.50%	1.40%	1.50%
Hispanic	50.60%	69.20%	71.70%	72.20%
Educational attainment				
Less than high school	22.60%	42.20%	38.00%	40.40%
Less than college	50.20%	50.60%	52.20%	52.00%
College graduate or more	27.20%	7.20%	9.80%	7.60%
Age (in years)	28.4	26.5	26.8	26.4
Insurance type				
Private coverage	51.00%	23.60%	30.20%	24.90%
Public coverage	46.90%	74.40%	67.70%	73.40%
Indigent coverage	0.10%	0.10%	0.10%	0.10%
Self pay or other	2.10%	1.90%	2.00%	1.60%

Table 3.2: Unadjusted prevalences of preterm birth, pregnancy complications and health behaviors, firearm violence rate, and demographics across control and high violence communities in full and propensity score restricted data

	Risk difference between high violence and comparison neighborhoods (95% CI)
Intermediate variables	
Hypertension	0.10% (-0.11%, 0.31%)
Diabetes	0.24% (0.07%, 0.40%)
BMI <=20	-0.22% (-0.87%, 0.43%)
BMI >20 and <30	-1.27% (-2.49%, -0.05%)
BMI>=30	1.48% (0.10%, 2.86%)
Pregnancy complications and health behaviors	
Any infection	1.33% (-0.23%, 2.89%)
Early preeclampsia	0.05% (-0.04%, 0.15%)
Gestational diabetes	0.30% (-0.65%, 1.25%)
Asthma	0.69% (0.04%, 1.35%)
Substance use	0.70% (0.13%, 1.26%)
Preterm birth	
All	0.49% (0.07%, 0.91%)
Spontaneous	0.40% (-0.01%, 0.81%)
Indicated	0.09% (-0.23%, 0.42%)

Table 3.3: Association between high firearm violence exposure and pregnancy complications, health behaviors, intermediate variables, and preterm birth

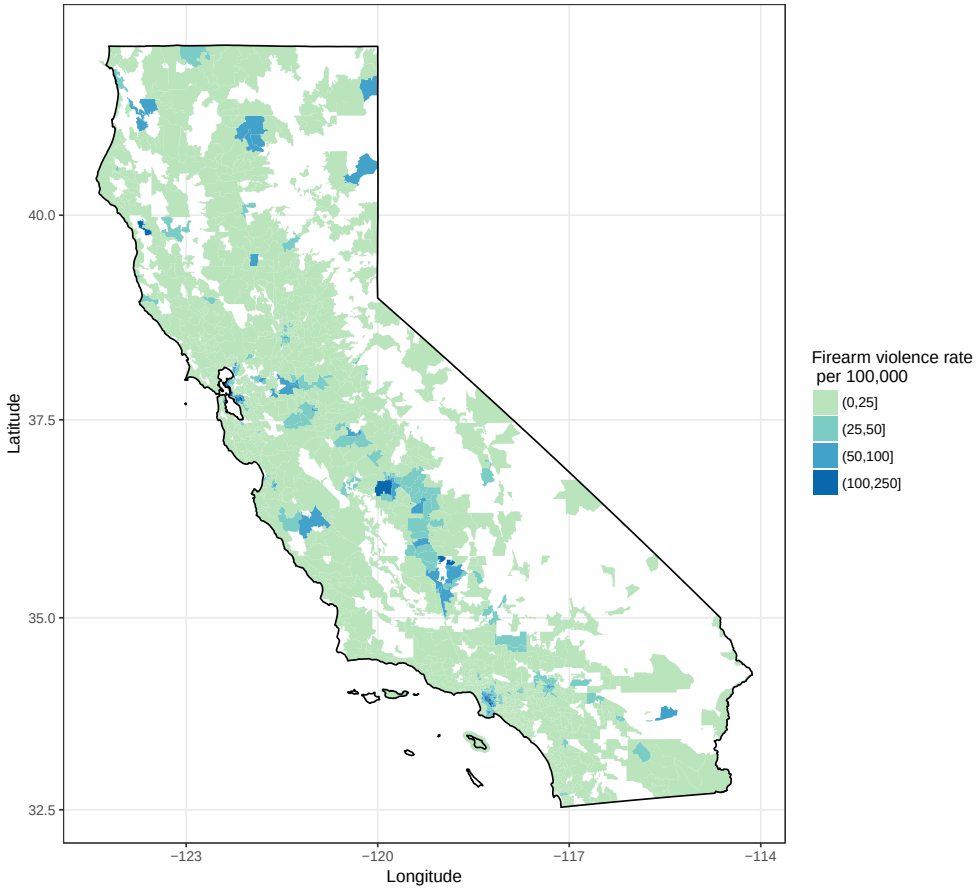


Figure 3.2: Map of firearm violence rates from 2007-2011 across ZCTAs in California.

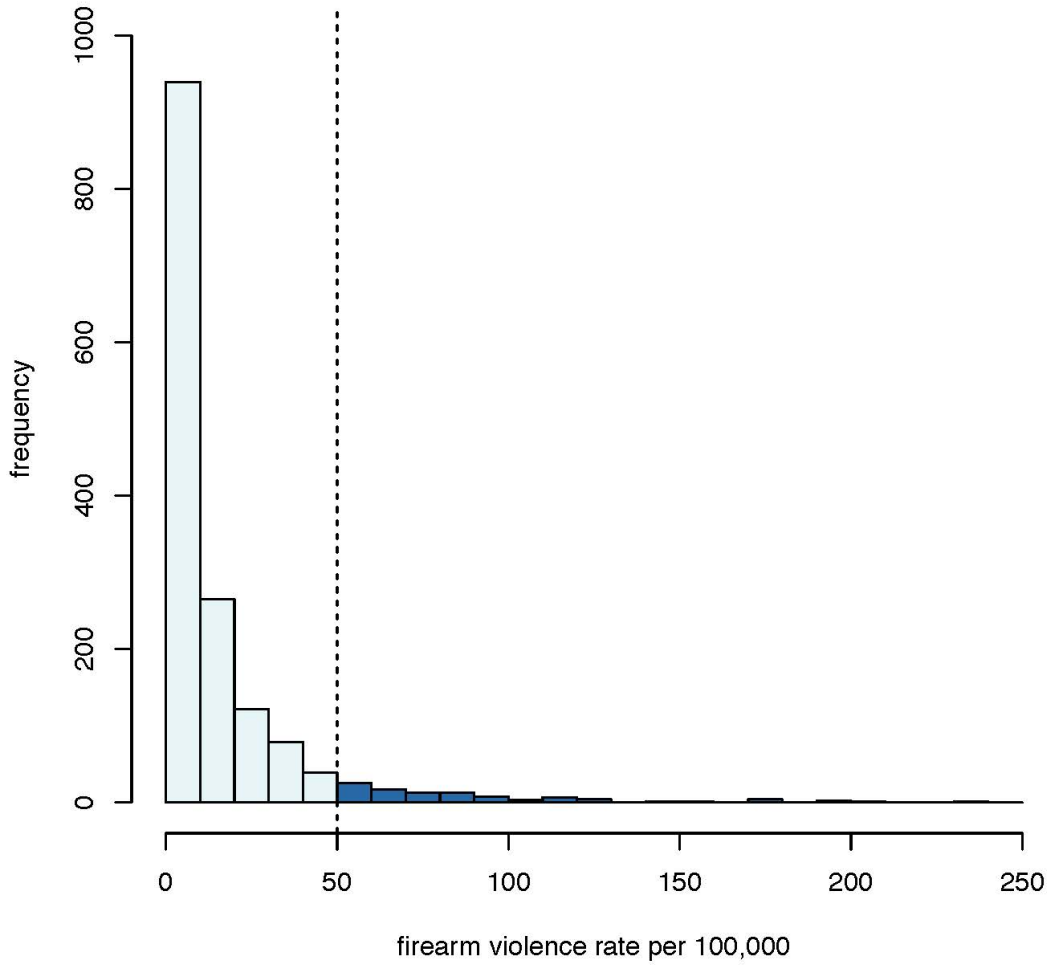


Figure 3.3: Distribution of firearm violence rates from 2007-2011 among ZCTAs in California.

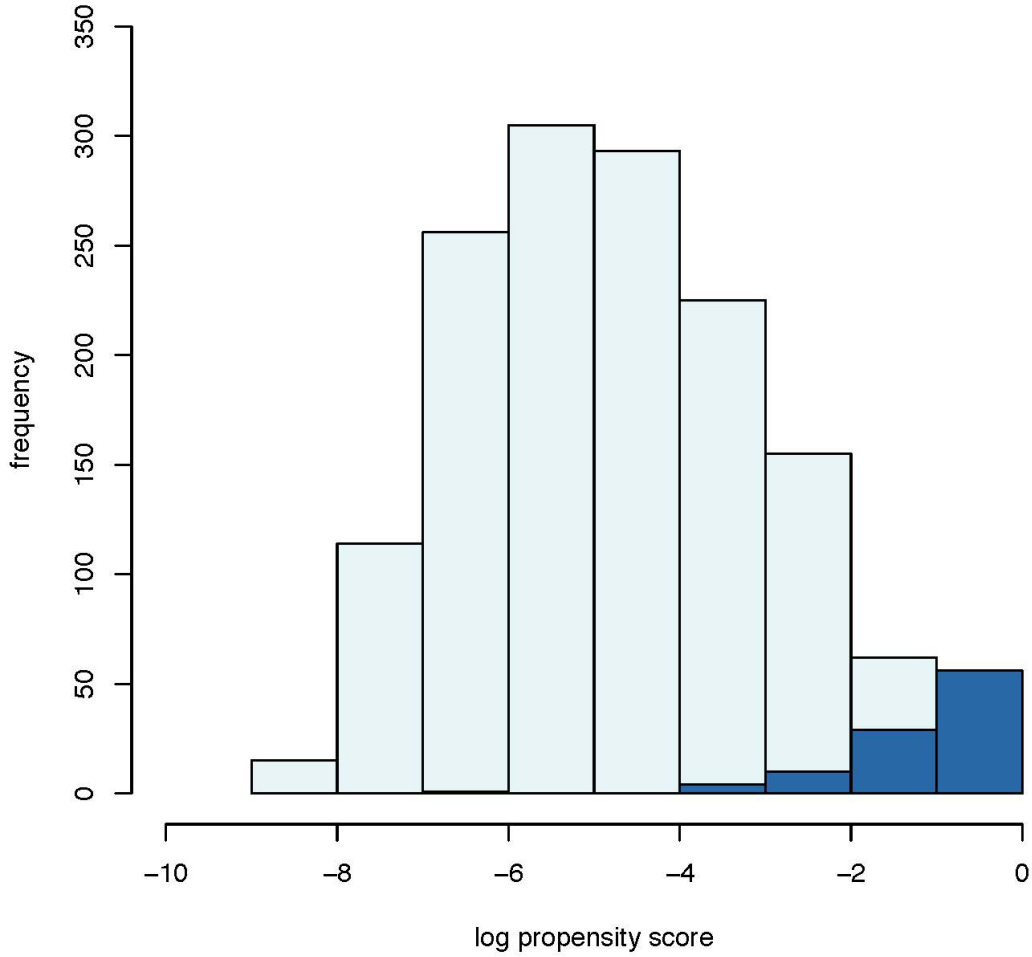


Figure 3.4: Log propensity scores for having a firearm violence rate above 50 per 100,000 (high violence) neighborhood.

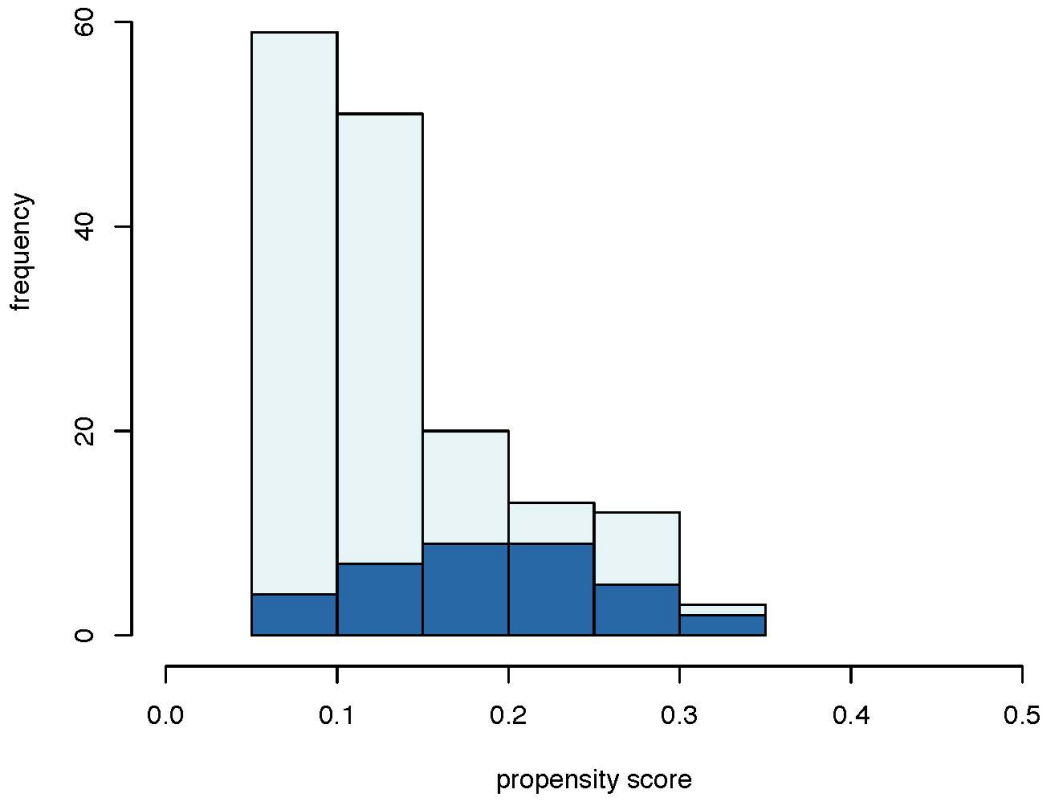


Figure 3.5: Area of common support of ZCTAs with and without high firearm violence.

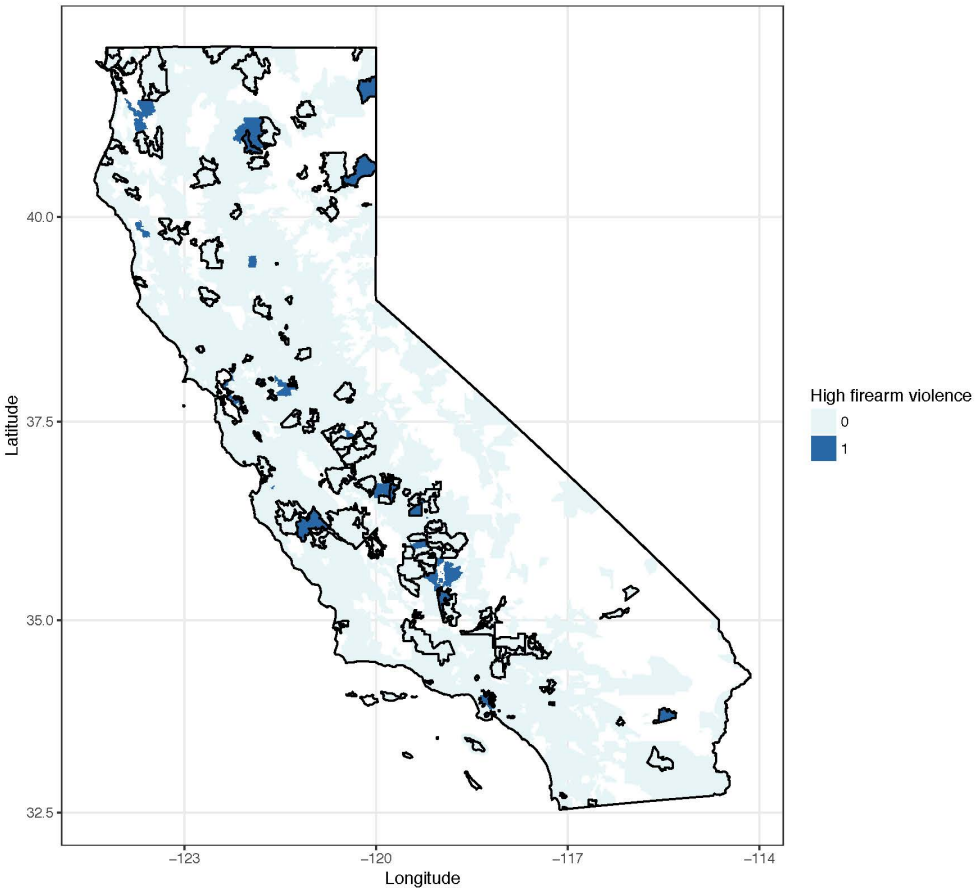


Figure 3.6: Map of the locations of high violence and control neighborhoods in California from 2007-2011.

Note: The ZCTAs outlined in black are those included in the analysis, meaning their propensity score for high violence fell within the area of support.

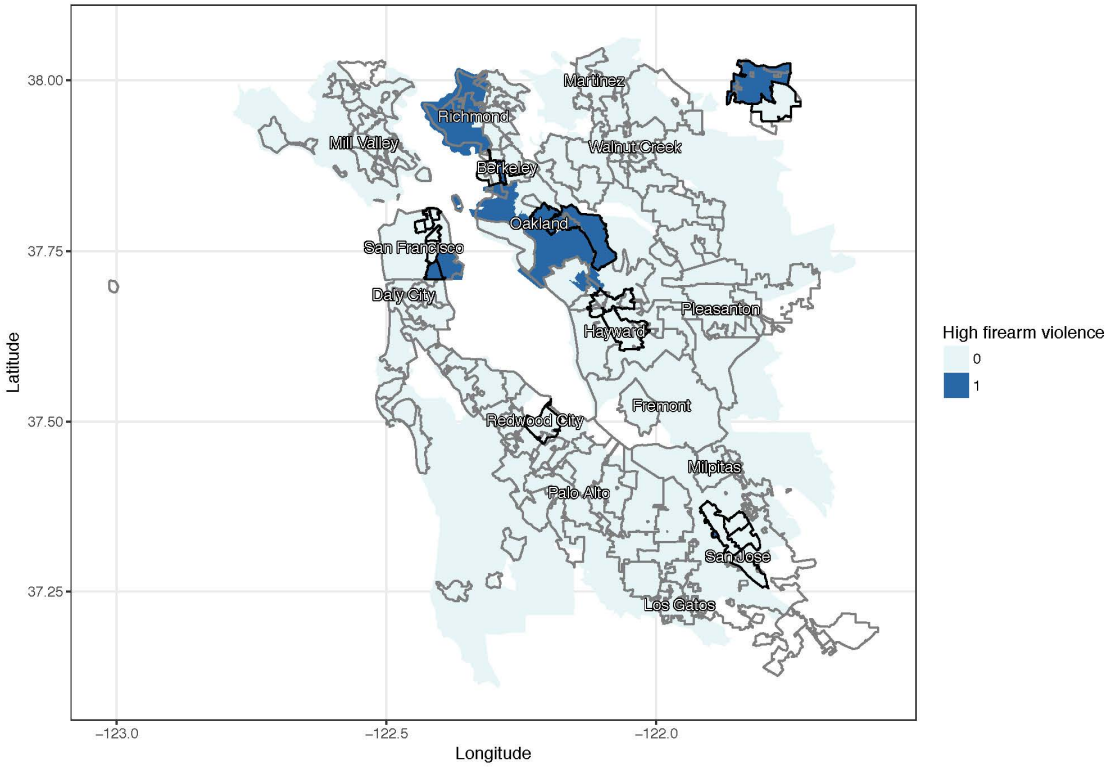


Figure 3.7: Map of the locations of high violence and control neighborhoods in the Bay Area from 2007-2011.

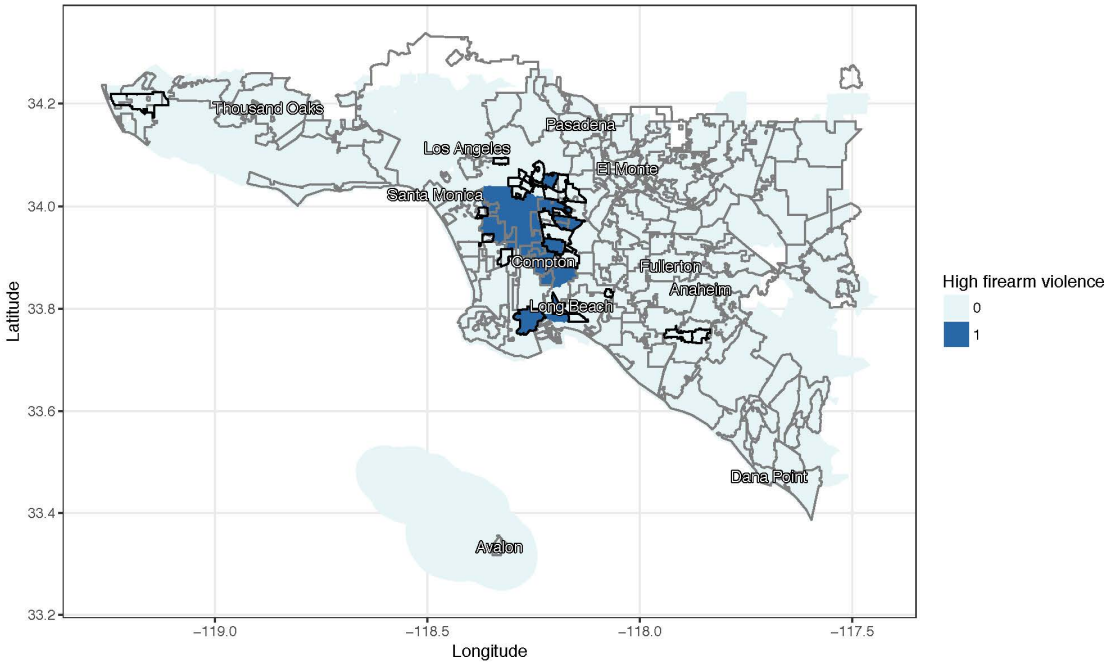


Figure 3.8: Map of the locations of high violence and control neighborhoods in the Los Angeles area from 2007-2011.

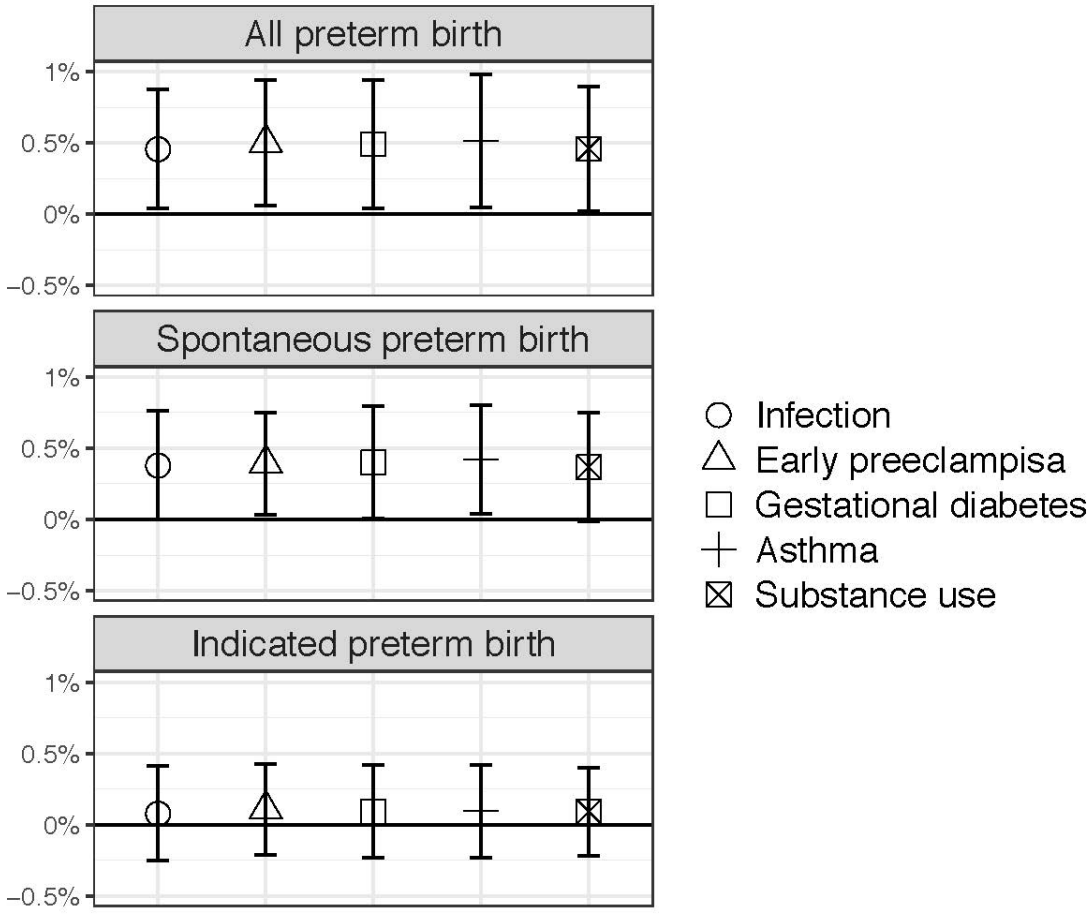


Figure 3.9: Stochastic direct effects of high firearm violence on preterm birth by pregnancy complications and health behaviors.

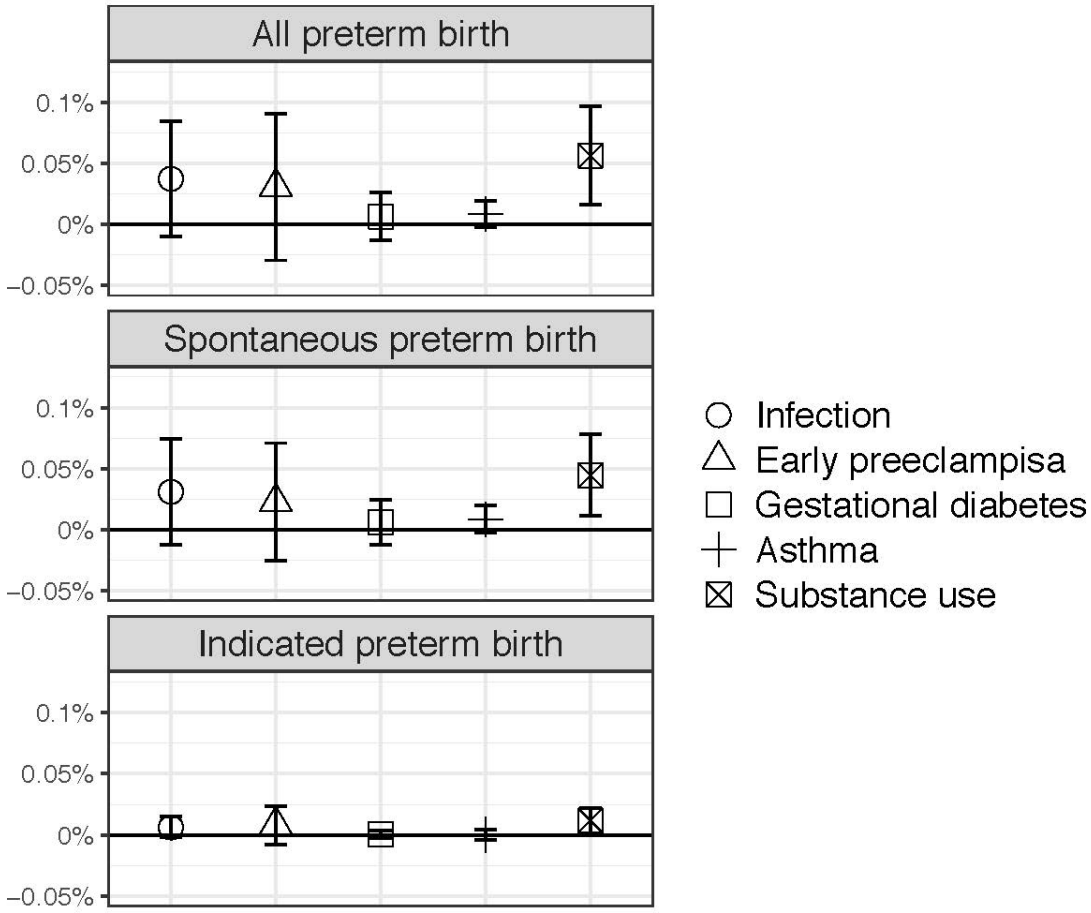


Figure 3.10: Stochastic indirect effects of high firearm violence on preterm birth by pregnancy complications and health behaviors.

3.5 Supplemental Material

SuperLearner Algorithms

The algorithms included in the SuperLearner included Bayesian generalized linear models, generalized linear models, generalized linear models with interactions, regularized generalized linear models, mean, multivariate adaptive polynomial spline regression, stepwise model selection, forward stepwise model selection, stepwise model selection with interactions, and stepwise model selection using AIC.

The number of folds used in the cross-validation was 10, and the least squares loss function was used to assess performance.

Firearm violence	
Injuries due to firearm	E9550 E9554, E9650 E9654, E970, E9850 E9854 with E960 - E969 and E970 E977 to identify assaults
Deaths due to firearm	X72 X73, X93 X95, Y350, Y22 Y24, with U01*, U02*, X85 Y09, Y871, Y35* to identify homicide or manner of death classified as homicide
Complications and behaviors	
Hypertension	64206422, 7962, 99791, 4010, 4011, 4019, 4020, 4021, 4029
Diabetes	25002508, 6480
Preeclampsia	64236427, 6429
Gestational diabetes	6488064883
Asthma	493
Substance use	6483, 3030, 3039, 30403059
Infections	
Organism-specific infection categories	
Bacterial infections	001005, 008.0008.5, 010018, 020027, 030041, 079.9, 079.98, 080083,088.0, 090098, 320322, 373.11, 424, 461, 481, 482, 483, 537.9, 540, 542, 567, 574.00, 575.1, 590, 595, 597, 599.0, 614, 616, 646.5, 647.0, 647.1, 647.3, 658.4, 659.31, 670.02, 681, 682, 684, 686, 795.5, 919.5
Mycoses	110118
Parasitic infections	006, 007, 084087, 088.8, 120129, 130136, 647.4, 647.8
Viral infections	008.6, 008.8, 042, 045049, 050057, 060066, 070077, 079.0079.89, 079.99, 460, 466, 480, 487, 571.4, 573.1573.3, 647.5, 647.6
Unknown organism	099.9, 350.2, 351.0, 370.20, 371.00, 372.00, 372.3, 372.30, 373.00, 380.10, 380.23, 381.3, 381.4, 382.9, 462, 463, 464.0, 465.9, 466.0, 472.0, 473.9, 486, 490, 491.9, 528.0, 530.1, 530.10, 533.90, 535.0, 535.00, 535.50, 575.8, 575.9, 597.80, 599.7, 616.0, 616.10, 646.61646.63, 647.23, 647.81, 647.83, 647.91, 686.9, 730.90
Organ-specific infection categories	
Cardiovascular	421, 422.0, 424
Skin	035, 053, 078.0, 078.1, 110, 111, 117.1, 117.2, 133, 680682, 684686
Ear	380382
Eye	054.4, 076, 370373
Gastrointestinal	001009, 040.2, 041.4, 070, 072 112.0, 123.0, 123.2123.9, 528.0, 528.1, 528.3, 530.1, 530.2, 531.9, 531533, 535, 537.9, 540, 542, 570573, 574.00, 575.0, 575.1, 575.8, 575.9, 577.0, 577.1
Genitourinary	041.6, 054, 078.1, 079.9, 079.98, 098.0098.3, 112.1, 131.00131.02, 132.2, 590, 599, 614, 616, 646
Lower respiratory	010012, 115, 466,480487, 490, 491, 795.5
Upper respiratory	032, 033, 040.1, 041.5, 460465, 472, 473, 476
Unknown organ	038.0, 038.11, 047.9, 041.04, 041.10, 041.11, 041.19, 041.85, 041.89, 041.9, 052.9, 054.8, 054.9, 079.89, 079.99, 097.1, 099.9, 112.9, 134.9, 350.2, 351.0, 567.2, 647.01, 647.23, 647.61, 647.63, 647.51, 647.81, 647.83, 647.91, 658.41, 659.31, 670.02, 730.90, 919.5, 998.5
Spontaneous labor	65810, 65811, 65813, 64400, 64403, 64410, 64413, 64420, 64421

Table 3.4: List of ICD-9-CM codes used to identify firearm injuries, pregnancy complications, and health behaviors, and ICD-10-CM codes used to classify firearm deaths.

Residential segregation measures
Black isolation index
Black segregation index
Poverty measures
% of households using food stamps
% with income below the poverty line
% of individuals with income from rental properties or investments
% workers who commute by car
% families with children in which no adults are working
Educational attainment measures
% men 65 and older with a high school degree
% men 25 and older with less than a 9th grade education
% men age 25-34 with at least a high school education
% veterans with less than high school education
% men age 65 and older with a college degree
% men 25 and older with a graduate degree
Social measures
% percent never married
% separated
Environmental measures
Maximum average monthly temperature
Latitude
Longitude

Table 3.5: Covariates included in predictor set used to estimate the propensity score.

	Comparison	High firearm violence	Absolute difference	Standardized bias
Firearm violence rate per 100,000 residents	9.9	87.6	77.6	49.5
Covariates				
Percent with income from interest or rental properties	25.3	11.1	14.2	10.5
Percent men with less than 9th grade education	8.5	20.8	12.3	11.4
Percent of men age 25 and older with graduate school education	12.1	5	7	6
Percent of men age 25-34 who graduated high school	83.9	66.2	17.7	9.7
Percent of men 65 and older who graduated high school	79.4	57.5	21.9	11.2
Percent of men 65 and older who graduated college	31.4	14	17.4	8
Percent with income below the poverty line	13.4	26.6	13.2	13.5
Percent of workers who travel by car to work	84.3	79.9	4.4	3.6
Percent of families with children with no adults working	5.3	12.8	7.5	9.7
Percent of people who are separated	2.1	3.4	1.3	8.6
Percent of people who have never been married	31.4	41.7	10.3	9.7
Percent of veterans with less than high school education	7.3	12.3	5	5.4
Black segregation index	0	0.1	0.1	17.8
Black isolation index	0	0.1	0.1	17.4
Percent of households receiving foodstamps	5.9	15	9.1	13.9
Maximum average temperature	71.9	70.9	0.9	1.7
Longitude	-119.9	-120.1	0.3	1.2
Latitude	36.3	36.5	0.2	0.7
Propensity score for high violence	0	0.5	0.47	39.4
Sum of absolute covariate differences			142.9	160

Table 3.6: Averages of zip-code level predictors of firearm violence in all communities

Note: The sum of absolute covariate differences only includes the covariate differences, not the firearm violence or propensity score differences, in the total.

	Comparison	High firearm violence	Absolute difference	Standardized bias
Firearm violence rate per 100,000 residents	22	63.5	41.5	14.6
Covariates				
Percent with income from interest or rental properties	12.5	14.1	1.5	0.9
Percent men with less than 9th grade education	19.4	20.2	0.8	0.3
Percent of men age 25 and older with graduate school education	5.2	5.7	0.5	0.3
Percent of men age 25-34 who graduated high school	66.1	66.8	0.6	0.2
Percent of men 65 and older who graduated high school	57.6	59.5	1.9	0.4
Percent of men 65 and older who graduated college	15.8	17.7	1.9	0.5
Percent with income below the poverty line	23.7	23.1	0.6	0.3
Percent of workers who travel by car to work	80.5	84.4	3.8	1.2
Percent of families with children with no adults working	10.8	9.8	1	0.5
Percent of people who are separated	3	3.1	0.1	0.3
Percent of people who have never been married	39.5	38.1	1.4	0.7
Percent of veterans with less than high school education	13.9	11.8	2.1	0.8
Black segregation index	0	0	0	0.5
Black isolation index	0	0	0	0.5
Percent of households receiving foodstamps	12.3	14	1.7	1.3
Maximum average temperature	70.7	71	0.3	0.3
Longitude	-120.3	-120	0.3	0.7
Latitude	36.9	36.7	0.2	0.4
Propensity score for high violence	0.1	0.2	0.05	4.1
Sum of absolute covariate differences			18.9	10.2

Table 3.7: Averages of zip-code level predictors of firearm violence in propensity-score restricted communities

Note: The sum of absolute covariate differences only includes the covariate differences, not the firearm violence or propensity score differences, in the total.

	Comparison	High firearm violence	Absolute difference	Standardized bias
Firearm violence rate per 100,000 residents	23.5	63.5	39.9	17.3
Covariates				
Percent with income from interest or rental properties	11.6	14.1	2.5	1.4
Percent men with less than 9th grade education	20.1	20.2	0.1	0.1
Percent of men age 25 and older with graduate school education	4.6	5.7	1.1	0.7
Percent of men age 25-34 who graduated high school	65.3	66.8	1.5	0.5
Percent of men 65 and older who graduated high school	55.8	59.5	3.7	0.9
Percent of men 65 and older who graduated college	14	17.7	3.6	1.2
Percent with income below the poverty line	24.8	23.1	1.7	1
Percent of workers who travel by car to work	81.1	84.4	3.2	1.3
Percent of families with children with no adults working	11.8	9.8	2	1.3
Percent of people who are separated	3.1	3.1	0	0
Percent of people who have never been married	39.6	38.1	1.5	0.9
Percent of veterans with less than high school education	13	11.8	1.2	0.7
Black segregation index	0	0	0	0.1
Black isolation index	0	0	0	0
Percent of households receiving foodstamps	13	14	1.1	0.8
Maximum average temperature	70.8	71	0.2	0.2
Longitude	-120.2	-120	0.2	0.7
Latitude	36.8	36.7	0.1	0.3
Propensity score for high violence	0.2	0.2	0.02	1.34
Sum of absolute covariate differences			23.8	12.0

Table 3.8: Averages of zip-code level predictors of firearm violence in propensity-score restricted communities that were also weighted by the odds of the propensity score

Note: The sum of absolute covariate differences only includes the covariate differences, not the firearm violence or propensity score differences, in the total.

Results of analysis with alternate cutoff for high violence

We replicated our analyses categorizing high violence as above the 90th percentile of the firearm violence rates (which corresponded to a rate of 37.97 per 100,000 persons) across ZCTAs. This resulted in the classification of 155 ZCTAs as high violence. After restricting based on the propensity score overlap, 246 ZCTAs were included in the analysis, of which 52 were high violence and 194 were the comparison neighborhoods.

The first stage effects were very similar to those in the main analysis (Supplemental Table 3). The only substantive differences between the main analysis and the sensitivity analysis were the association between living in a high violence neighborhood and gestational diabetes, which was a positive association in the main analysis but negative in the sensitivity analysis. Furthermore, there was a positive relationship with indicated preterm birth in the main analysis but negative in the sensitivity analysis. In the sensitivity analysis, as with the primary analysis, the relationship between firearm violence and preterm birth was driven by spontaneous rather than indicated preterm birth.

The mediation results were also very similar to those in the main analysis, with the main mediation effects observed for infection, preeclampsia, and substance use (Supplemental Figures 3 and 4).

	Risk difference between high violence and comparison neighborhoods (95% CI)
Intermediate variables	
Hypertension	0.07% (-0.11%, 0.25%)
Diabetes	0.23% (0.11%, 0.36%)
BMI <=20	-0.43% (-1.01%, 0.14%)
BMI >20 and <30	-0.71% (-1.81%, 0.40%)
BMI>=30	1.11% (-0.25%, 2.47%)
Pregnancy complications and health behaviors	
Any infection	1.52% (0.12%, 2.93%)
Early preeclampsia	0.05% (-0.03%, 0.13%)
Gestational diabetes	-0.10% (-0.86%, 0.65%)
Asthma	0.80% (0.14%, 1.47%)
Substance use	0.61% (0.12%, 1.10%)
Preterm birth	
All	0.47% (0.14%, 0.80%)
Spontaneous	0.49% (0.17%, 0.81%)
Indicated	-0.02% (-0.19%, 0.15%)

Table 3.9: Association between high firearm violence exposure (defined as the 90th percentile of firearm violence levels) and pregnancy complications, health behaviors, intermediate variables, and preterm birth

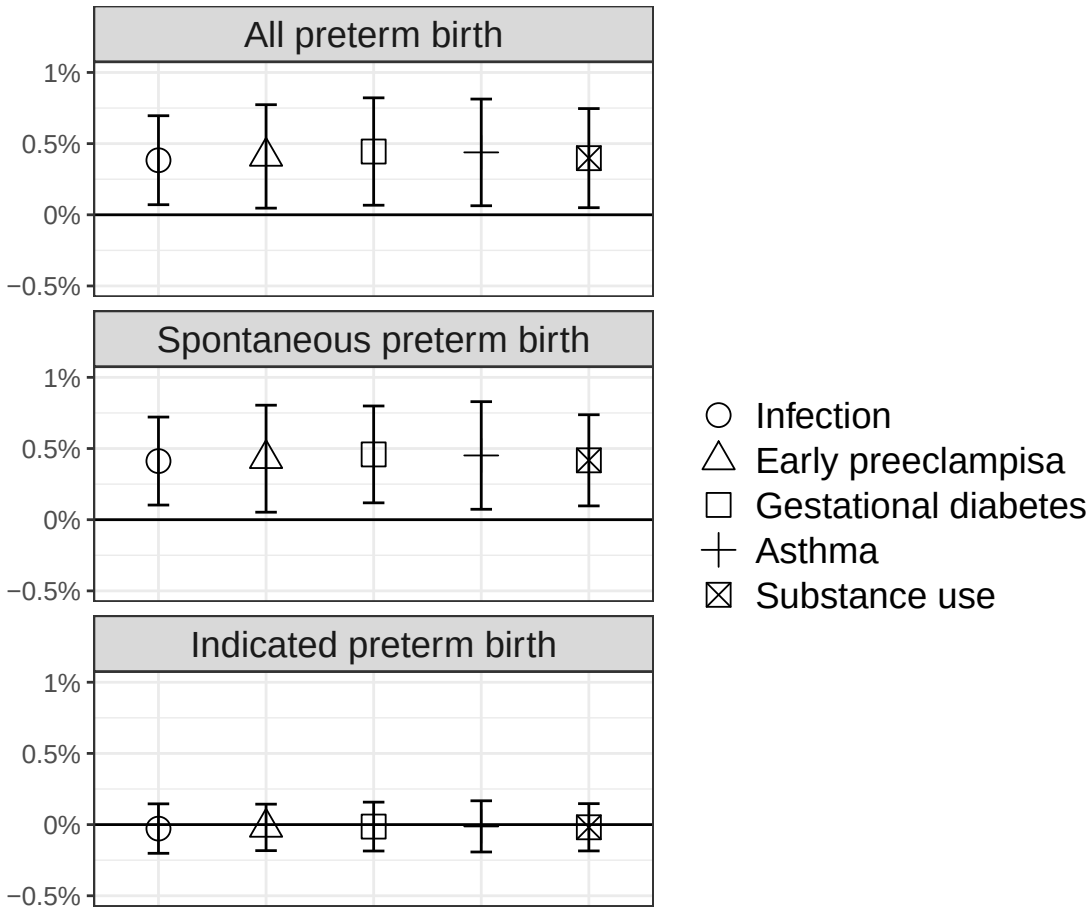


Figure 3.11: Stochastic direct effects of high firearm violence (defined as the 90th percentile of firearm violence levels) on preterm birth by pregnancy complications and health behaviors.

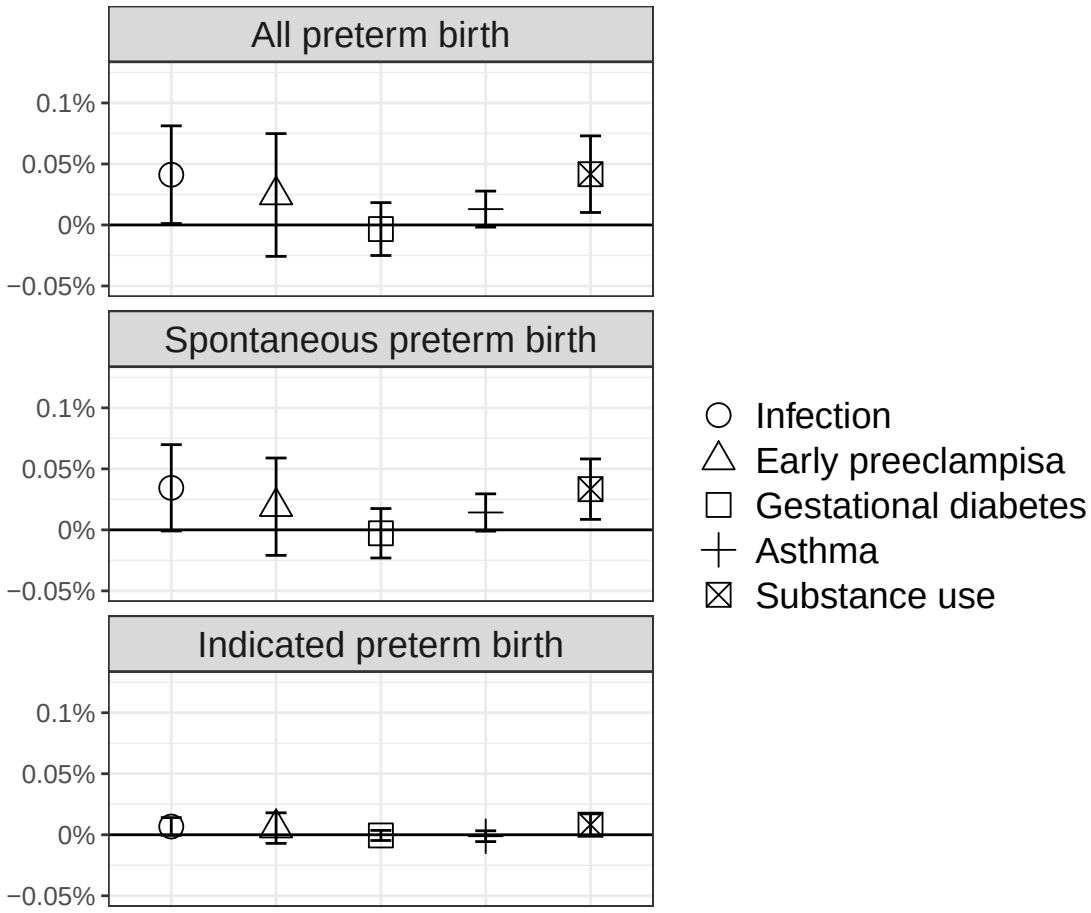


Figure 3.12: Stochastic indirect effects of high firearm violence (defined as the 90th percentile of firearm violence levels) on preterm birth by pregnancy complications and health behaviors.

Chapter 4

Fatal Police Violence and Preterm Birth

4.1 Introduction

Police violence has been recognized as a critical problem in public health and a barrier to health equity [70, 176]. In the United States, Black men and women are more likely to be victims of fatal police violence compared to people of other races and ethnicities [71, 177]. Communities of color are more likely to experience elevated police surveillance and to have interactions between residents and police become violent [72, 178–180]. Witnessing or hearing about police violence in one's neighborhood can also have detrimental health effects that are likely to be disproportionately felt by people of color [71, 181].

In particular, women who are exposed to fatal police violence in their neighborhood during pregnancy may experience stress and worry about the safety of their neighborhood for themselves and their families. Such stress may have deleterious consequences for the health of the mother and infant, especially if women perceive such violence as a potential threat to their children. For example, exposure to fatal police violence in one's neighborhood may induce an acute stress response among pregnant women that could have negative physiologic implications for the pregnancy or instigate maladaptive coping behaviors with adverse effects on maternal and infant health. Recent evidence suggests unarmed killings of black men in America increased reports of poor mental health among black adults [182]. Additionally, some studies indicate that people exposed to violence in their community can suffer from increased anxiety, depression, or post-traumatic stress disorder [183–186]. Exposure to these stressors during pregnancy has previously been tied to adverse pregnancy outcomes, including spontaneous abortion, preterm birth, and small-for-gestational age births [187–190]. Furthermore, previous evidence indicates that exposure to violence in one's community is associated with adverse pregnancy outcomes [22, 30, 101, 119, 164, 191].

Research on the relationship between exposure to police violence and reproductive health outcomes is limited, however, and a recent call for literature on this topic has thus far not

been answered [192]. To address this gap, this study estimates the relationship between exposure to fatal police violence over the course of pregnancy on hazard of preterm birth among mothers in California. Women of color are more likely to reside in communities experiencing police violence and therefore may be more likely to internalize fatal police violence as a threat to themselves and their families; given this, coupled with experiences of chronic stress, including interpersonal and structural racism, poverty, and interpersonal and/or sexual violence [193], we expect that an acute stressor like exposure to fatal police violence in one's neighborhood during pregnancy may uniquely affect women of color.

In addition, Black women in California have the highest levels of preterm birth [194]. Differences in risk for adverse outcomes have not been adequately explained by individual-level risk factors [2], and examination of structural disadvantages (including exposure to fatal police violence during pregnancy) hold promise to explain persistent inequities in reproductive health [15]. Therefore, we evaluated whether race/ethnicity modified the relationship between fatal police violence and preterm birth among women in California. Investigating the relationship between exposure to fatal police violence during pregnancy and adverse pregnancy outcomes could improve our understanding of how and why persistent disparities exist in maternal and infant health by race/ethnicity.

4.2 Data and Methods

This study utilized birth records from Californias Vital Statistics from 2007-2015, death records from Vital Statistics from 2006-2015, and the Fatal Encounters database, which is described in more detail below. We included all births whose conception occurred after January 1, 2007 and before March 1, 2015 to avoid fixed cohort bias [104]. This study was reviewed and approved by the California Health and Human Services Agency Committee for the Protection of Human Subjects, and the University of California, Berkeley Committee for the Protection of Human Subjects.

Exposure to Fatal Police Violence

Exposure to fatal police violence in one's Census tract during pregnancy was assessed using two sources of data. We used death records from California Vital Statistics, which identified cases of fatal police violence via the ICD-10 legal intervention cause of death codes. The relevant codes were Y35.0-Y35.7, capturing death due to legal intervention involving firearm discharge, explosives, gas, blunt objects, sharp objects, or unspecified means. We excluded code Y35.5, which is used to identify death due to legal execution. Code Y89.0 was also included in order to identify deaths due to late effects of legal intervention injury. These records likely undercounted deaths due to legal intervention [195]; therefore, we also used the Fatal Encounters database of incidents of fatal police violence [196]. These data are publicly available and compiled by paid researchers and crowdsourced contributions using media coverage and public records to identify instances of police action that results in a fatality.

Each entry is validated against other existing media databases and data from government agencies.

The death records were geocoded to the decedent's address at the time of death, and the Fatal Encounters records were geocoded to the location of the fatal interaction between the decedent and the police. Therefore, our analyses using the death records will evaluate whether having a neighbor killed by police during pregnancy is associated with the hazard of preterm birth, whereas the analyses using the Fatal Encounters database will assess whether having a fatal incident involving police in a womans neighborhood occurring during her pregnancy is associated with hazard of preterm birth. Evaluating both of these forms of the exposure will provide information as to which forms of exposure to fatal police violence may be more relevant for pregnancy outcomes. Furthermore, both sources likely have different biases due to the collection methods; comparing them will mitigate the potential for systemic bias in one source to exert undue influence on our findings.

The neighborhoods where police violence occurs may have unique characteristics that make them difficult to compare to other places. For example, they tend to have suffered from decades of over-policing and underinvestment in the built environment, transportation infrastructure, education, health care services, and sources of economic mobility [177, 197, 198]. Exposure to fatal police violence itself may be conceptualized as a form of structural racism [72]. However, because neighborhood characteristics associated with deprivation tend to co-occur, communities with high levels of police violence likely have many other social and environmental characteristics that could be detrimental for reproductive health, including high poverty, elevated levels of non-police violence, poor walkability, or increased levels of pollutants [199, 200]. Because these characteristics are so highly correlated, it is difficult to disentangle them by statistical adjustment alone [5]. Therefore, in order to isolate the association between incidents of fatal police violence and preterm birth, we compare women within neighborhoods who conceived at different times and therefore had different exposures to fatal police violence during pregnancy. Accordingly, to ensure there was variation in the exposure within neighborhoods, we restricted the analysis to Census tracts in which at least one incident of fatal police violence occurred over the study period.

Covariates

Time to birth was measured using the obstetric estimate of gestational age in weeks [201]. We hypothesized that women of color may have a heightened response to witnessing or hearing about fatal police violence during pregnancy compared to white women. Therefore, we assessed whether there was effect modification by maternal race/ethnicity. In California birth records, race and Hispanic origin are recorded separately. Potential racial categories included white, Black or African American, Asian, Pacific Islander, Native American, and Alaska Native. Any women of Hispanic origin, including Mexican, Mexican-American, Puerto Rican, Cuban, Spanish, or other Hispanic origin were categorized as Latina regardless of their recorded race. Due to small numbers, we combined the Native American and Alaska Native groups, and the Asian and Pacific Islander groups. Non-Latina women with other or two or

more listed races were also combined, although multiracial non-Latina Black women were analyzed separately.

Models were adjusted for maternal age, age-squared to account for possible non-linearities in the effect of age, educational attainment, health insurance type, parity, and the year and season of conception. Educational attainment categories included less than high school, high school graduate, some college or associates degree, bachelors degree or higher. Insurance types were Medi-Cal or other public insurance, private insurance, and self-pay or other. Parity was categorized as 1, 2, or 3 or more. Due to the size of the data set and the computational limitations involved in analyzing these confidential data on a secure server, we performed a complete case analysis, as it was not feasible to perform multiple imputation or other procedures to account for missing data.

Statistical Analysis

Evaluating the relationship between exposures that occur during pregnancy and preterm birth can be problematic because infants born preterm by definition have less time in utero to accrue exposure. To address this, we analyzed time to birth as a survival outcome. Exposure to incidents of fatal police violence within a womans Census tract was a binary variable indicating whether an incident had occurred from the estimated conception week up through the gestational week of interest. We estimated Cox proportional hazard models stratified by Census tract [202], with separate models for early versus late preterm birth. Women were considered to be at risk for early preterm birth during gestational age 22-31 weeks and at risk for late preterm birth during gestational age 32-36 weeks. Stillbirths were included as an "event", therefore functioning as a competing risk for preterm birth. The proportional hazards assumption was evaluated and covariates that violated the assumption were interacted with time. Inference was clustered at the Census-tract level. These analyses were performed using Stata version 13.1.

Simulation study

Our conceptual framework suggests that stress in early pregnancy, and the behavioral and physiological responses to it, underlie the proposed relationship between exposure to fatal police violence in one's neighborhood and increased hazard for preterm birth. However, given that stressful experiences have also been tied to increased risk of miscarriage [183] and may inform reproductive decision-making, it is possible that exposure to fatal police violence may alter risk for spontaneous or medically induced abortion, which can be considered either a competing risk or censoring event, respectively, for preterm birth. Selection bias therefore may be created by only including gestations that persisted until at least 22 weeks in our analysis. Previous studies in perinatal epidemiology have utilized simulation to illustrate how methodological choices and/or data availability can influence results [203–206]. However, no previous studies have investigated how left truncation due to pregnancy loss may bias results of stressful exposures and birth outcomes in a survival context. Therefore, we conducted a

simulation study to address this question in the context of the relationship between fatal police violence and preterm birth.

Evidence suggests that 15-30% of recognized pregnancies end in spontaneous abortions, and 30-40% of unintended pregnancies (about 20% of recognized pregnancies) end in medically induced abortion [207, 208]. Therefore, up to 50% of the total number of recognized pregnancies may be excluded from an analysis that uses birth records. Given the magnitude of this left-truncation, selection bias due to exclusion of these conceptions could be large, even if the association between fatal police violence exposure and fetal loss is relatively weak. To test this, we conducted a record-level simulation study that examined a range of possible relationships between exposure to fatal police violence during early pregnancy and risk of spontaneous or induced abortion, and re-estimated the relationship between fatal police violence and time to birth including fetal losses. We conducted a simulation with a sample size of 500,000 to approximate one year of births in California. We simulated an extra 250,000 records (an increase of 50%) to include potential spontaneous and induced abortions. Exposure was assumed to be binary and was randomly assigned across all records with a probability of 0.025, corresponding to the prevalence of exposure to one or more fatal police violence incidents in the observed linked birth and Fatal Encounters data.

Spontaneous abortions were randomly assigned with baseline probability of 0.18, with potential increases in the probability associated with exposure. We varied the probability of spontaneous abortion associated with exposure from 0 to 0.01, by increments of 0.00025. For those who were assigned spontaneous abortion, the time until miscarriage was simulated from a Weibull model with shape parameter equal to 1.5 and the scale parameter equal to 0.5, with an intercept of 5 and multiplied by 10.

$$Binomial(1, p = 0.18 + \beta_1 A) * (5 + Weibull(shape = 1.5, scale = 0.5) * 10) \tag{4.1}$$

This parameterization was chosen as it yielded a minimum, mean, first quartile, median, third quartile, and maximum that approximated the average rate of spontaneous abortion by gestational age observed in California women [209]. Induced abortions were randomly assigned with baseline probability of 0.20, and potential increases due to exposure of 0 to 0.005 by increments of 0.00025. Among those who were assigned to induced abortion, the time until abortion was simulated from a Weibull model with shape parameter equal to 2 and scale parameter equal to 1 multiplied by 8.

$$Binomial(1, p = 0.2 + \beta_2 A) * Weibull(\lambda = 2, \gamma = 1) * 8 \tag{4.2}$$

This parameterization approximated the average rate of medically induced abortion by gestational age in the US [210]. The time to birth was simulated using a Weibull model with a shape parameter of 1.75 and a scale parameter of 1.5 plus potential effects of exposure.

$$43 - Weibull(\lambda = 1.75, \gamma = 1.5 + \beta_3 A) * 3.5 \tag{4.3}$$

The times simulated from the Weibull model were multiplied by 3.5 and subtracted from 43, to approximate the distribution of delivery by gestational age observed among actual

births in California. The potential influence of the exposure on the scale parameter ranged from -0.05 to 0.05 by increments of 0.05. This corresponds to shifting the distribution of gestational age at birth slightly earlier (for effect equal to 0.05 with an increased hazard of birth) or later (for effect equal to -0.05 with a decreased hazard of birth) (Figure 4.1).

Each record had an indicator for spontaneous and induced abortions assigned from a Bernoulli trial. If observations were assigned an indicator for both spontaneous and induced abortion, the times of each event were compared, and women were assigned to the event which occurred first. Women who experienced induced abortions in our simulations were classified as having their event times censored, whereas women who experienced spontaneous abortions were considered as having an event.

The strength of association between exposure to fatal police violence and spontaneous or induced abortion is not known, and so we tested a range of possible associations. For each parameter combination, we estimated a Cox proportional hazards model that first included and then excluded the simulated fetal losses, and compared the estimated associations for the hazard of preterm birth. We refer to the cohort that included all spontaneous abortions as the "full" cohort, and the model that included only observations whose simulated gestational age was greater than 22 weeks as the "truncated" cohort.

We compared the difference in estimated hazard ratios for preterm birth for the full and truncated cohorts, and examined how varying the strength of the relationship between exposure and spontaneous or induced abortion could affect the results. We also explored how truncation of the cohort due to exclusion of spontaneous and induced abortions affects the power to detect an effect. R version 3.5.0 was used for this simulation [157].

4.3 Results

There were 4,172,150 women who gave birth in California with estimated conception dates between January 1, 2007 and March 1, 2015. Of these, 0.4% were missing information on gestational age, 2.0% were missing maternal race/ethnicity, 3.8% were missing information on maternal educational attainment, 0.2% were missing information on health insurance type, and 0.1% were missing information on parity, and 0.01% were missing age or had implausible values. By restricting to complete cases, we excluded 4.6% of women, leaving a sample size of 3,979,340.

We then restricted our analyses to neighborhoods in which there was one or more incidents of fatal police violence at some point over the study period. For the analysis using death records, restriction to neighborhoods with variation in exposure resulted in a sample size of 387,618 (Table 4.1). Of these, 34,974 women (9.02%) were exposed to fatal police violence during their pregnancy. Using the Fatal Encounters data, there were 968,012 women who lived in a neighborhood in which there was a police-related fatality at some point during the study period, and 100,125 women (10.34%) were exposed during pregnancy. There were no neighborhoods in which all women were exposed, and therefore no exposed women were excluded from the analysis.

Women exposed to fatal police violence were somewhat more likely to be black or African American and Latina, and less likely to be white or Asian or Pacific Islander (Table 4.1). Exposed women were also more likely to have lower educational attainment, more likely to have public insurance, and more likely to have 3 or more children.

Using the death records measure of fatal police violence, we found reduced hazard of early preterm birth (HR = 0.85, 95% CI = 0.75, 0.97) and increased hazard of late preterm birth (HR = 1.05, 95% CI = 1.00, 1.09) associated with exposure to fatal police violence during pregnancy (Table 4.2). Using the Fatal Encounters records, we found similar magnitudes of association with early preterm birth (HR = 1.02, 95% CI = 0.95, 1.10) and late preterm birth (HR = 1.03, 95% CI 1.00, 1.06), but the variance was larger for the estimate of effect for early preterm birth. We did not observe any effect modification by race/ethnicity in the analyses using the death records or the Fatal Encounters records.

Simulation results

In the simulation, we examined how estimates of the hazard ratio for preterm birth differed in a truncated cohort (the births whose gestations exceeded 22 weeks that would be observed in birth records) from those in a full cohort (all conceptions, which includes spontaneous and induced abortions plus births). We varied three sets of parameters: the relationship of exposure to fatal police violence on risk of spontaneous abortion, relationship of exposure on likelihood of induced abortion, and relationship of exposure with time to delivery among live births. We observed that the relationship of exposure with induced abortion did not affect the pattern of bias between the full and truncated cohort estimates. Therefore, results that illustrate the change in the estimated hazard ratio between the full and truncated cohorts by strength of relationship between exposure and induced and spontaneous abortion are presented in the supplemental material.

The patterns in the estimated hazard ratios in the full cohort of all conceptions versus the truncated cohort of just births differ based on the relationship between exposure and time to live birth. We examined three scenarios in the simulation: exposure to fatal police violence has no effect on time to live birth, shortens time to live birth, or lengthens time to live birth. These effects are independent of the relationship between exposure to fatal police violence and spontaneous abortion.

If there was no effect of the exposure on time to birth except through the exposure's effect on spontaneous abortion, then the truncated cohort was not able to detect any effect irrespective of the strength of the relationship with spontaneous abortion. Figure 4.2 demonstrates that as the strength of the relationship between the exposure and spontaneous abortion increases, the hazard ratio in the full cohort of all conceptions increased, as did the power to be able to detect an effect. However, the truncated cohort of just births did not show any relationship between the exposure to fatal police violence and hazard of preterm birth. The points in the truncated cohort in which an effect was detected are reflective of the Type I error rate of 0.05 (accordingly, in the truncated cohort 4.6% of points indicate an effect was detected).

When exposure to fatal police violence shortened time to live birth (by increasing the hazard), the truncated cohort of just births overestimated the hazard ratio compared to the full cohort of all conceptions. For example, Figure 4.3 demonstrates that while both the full and truncated cohorts showed an increased hazard of preterm birth associated with exposure, the association was stronger in magnitude in the truncated cohort. This is because there were more conceptions included in the full cohort that were not affected by exposure. The truncated cohort restricted to live births, which are the only conceptions affected by the exposure, and thus, the hazard ratio was larger compared to the full cohort. As the relationship between exposure and spontaneous abortion strengthened, however, the hazard ratio in the full cohort increased in magnitude.

When exposure tended to lengthen time to live birth (by reducing the hazard), we observed a protective effect of the exposure on the hazard of preterm birth in the truncated cohort. Figure 4.4 showed how the association changed with increasing effect of the exposure on risk for spontaneous abortion. Similarly to Figure 4.3, the effect in the full cohort was attenuated compared to the truncated cohort, due to the additional conceptions that were unaffected by the exposure. As the relationship between the exposure and risk for spontaneous abortion increased, however, the tendency for the exposure to reduce gestational time by increasing risk of spontaneous abortion and lengthening gestational time among live births started to cancel one another out.

In the real data, we observed a positive hazard ratio associated with exposure to fatal police violence and this was observed in a truncated cohort of only births. The simulation results suggest this would be observed if exposure tended to increase the hazard of late preterm birth independent of its effect on spontaneous abortion.

4.4 Discussion

We found that exposure to fatal police violence during pregnancy was associated with a small increased hazard of late, but not early, preterm birth. Our results were similar in models utilizing two different measures of exposure to fatal police violence, suggesting that both living in a neighborhood in which fatal police violence occurs (Fatal Encounters database measure) and having a neighbor who was killed by the police (death records measure) may be detrimental for women during pregnancy. We hypothesized there would be effect modification by race/ethnicity, but we did not observe any. This may be related to a lack of statistical power. Given the rare exposure and small effect size, there simply may not be enough women in each race/ethnic group within Census tracts to detect meaningful differences in associations across groups.

We also observed a protective association of exposure on early preterm birth using the death records exposure measure, which was unexpected. Several potential explanations could be behind this result. Exposure could prolong gestation among early preterm births, so the babies that would have been born early preterm are born late preterm or at term instead. However, prior evidence suggests the opposite occurs more often [49, 187]. Measurement

error may exist only among exposed early preterm births, so that their gestational ages are systematically misclassified. This would not explain, however, why we do not observe the same pattern with the Fatal Encounters data. One might consider that there could be an increased risk of spontaneous abortion associated with exposure that selects out births that may have otherwise been born during the early preterm period. However, the results from our simulation suggest that if there is no effect of the exposure on time to birth but there is an effect on spontaneous abortion, restricting to live births would not result in a protective association. The counterintuitive findings could also be a result of random error.

To our knowledge, no previous study has examined the association between exposure to fatal police violence in one's community during pregnancy and preterm birth. However, several prior studies have looked at changes in homicide exposure within communities and risk of preterm birth. A previous study in California did not find a relationship between experiencing a homicide in one's Census tract in early pregnancy and risk of preterm birth [191], and a study in Mexico found no relationship between increases in municipal-level homicide rates and gestational age at delivery [101]. Conversely, a study of mothers in Brazil found an increased risk of preterm birth associated with one standard-deviation increase in homicide rates, with stronger associations observed among women with lower educational attainment [119]. Our findings suggest fatal police violence only increases risk of late preterm birth; therefore, studies that did not separate late and early preterm birth may have obscured potential effects in later gestational ages. Differences in context, or the specific nature of police violence compared to other types, may also explain the difference in results.

This study had several limitations. In particular, we used administrative data that has several potential sources of measurement error. We only had access to womens addresses at the time of delivery. Therefore, any women who moved during pregnancy are subject to exposure misclassification. Our outcome used the clinician estimate of gestational age at delivery, which is most accurate when an ultrasound in performed early in pregnancy [211]. Accuracy therefore may differ based on the timing of the first prenatal visit; it is possible that women exposed to fatal police violence in their neighborhoods experienced stress that influenced whether or not they received prenatal care early in pregnancy. In addition, data on fatal police violence have historically not been reliably collected in California or the United States generally. Because of this, we used two sources of data that capture different measures of fatal police violence. Each source, however, has its own potential sources of error. Deaths due to legal intervention are captured in the death records from vital statistics, but the process by which legal intervention is recorded as the cause of death is not systematic, and evidence suggests vital statistics records undercount legal intervention deaths [195]. The Fatal Encounters data rely primarily on media reports about deaths caused by police activity, although they also supplement these data with records obtained by public records requests. While the data are more complete than the hospitalization and death data, the majority of cases are identified using internet-published media reports, and therefore earlier years may have differentially fewer cases. Furthermore, increased media attention in recent years around deaths due to police activity may have resulted in more incidents being captured in later years. By including fixed effects for the year of conception in our analysis, we aimed

to reduce the possibility that these time trends in reporting biased our results.

Finally, we used birth records to estimate the effect of a stressful in utero exposure on hazard of preterm birth. While we were able to include all live births that occur among women living in California, these data exclude any conceptions ending in spontaneous or induced abortion, resulting in left-truncation of the cohort. Previous studies have illustrated that risk of spontaneous abortion that is differential by exposure can induce selection bias into the analysis [203]. However, this only introduces systematic error into our study if the goal is to estimate the non-conditional effect of exposure to fatal police violence on the hazard of preterm birth. By using a survival analysis framework, we can still achieve unbiased results as long as our results are interpreted as the association between exposure to fatal police violence and hazard of preterm birth, conditional on conceptions having survived until at least 22 weeks gestation.

However, in order to more completely explore how this left-truncation may be affecting our results, we conducted a simulation study. We found that if there is no effect of exposure to fatal police violence on the gestational age at birth, but there is an increased risk of spontaneous abortion due to exposure to fatal police violence, our findings (in a truncated cohort of births only) would underestimate the hazard ratio. We also found, however, that if exposure to fatal police violence reduced gestational time among live births, the hazard ratio in the truncated cohort would be larger in magnitude compared to the full cohort (of all conceptions including spontaneous abortions), unless the corresponding effect on spontaneous abortion were as strong or stronger. Finally, if exposure to fatal police violence lengthened gestational time, then the results from the truncated cohort would demonstrate this protective effect. The effect would be closer to the null in full cohort, and would approach the null as the effect on spontaneous abortion cancelled out the effect among live births.

This study examined exposure to acute incidents of fatal police violence during pregnancy. Chronic exposure is likely to have even greater effects. Studying exposure to chronic community violence is challenging because of structural confounding; nevertheless, future research should find creative ways to study long-term exposure to fatal police violence, as it is likely to be even more impactful for understanding disparities in reproductive health.

Given the long history of discriminatory police violence in the United States and its role in perpetuating the marginalization of certain communities in California, exposure to fatal police violence during pregnancy may be an important link in the pathway between structural racism and maternal and infant health. Demonstrating an intergenerational connection between violent deaths and adverse birth outcomes within communities has the potential to clarify the relationships between historical inequities and health in those communities today [192]. This work suggests that exposure to fatal police violence during pregnancy is a potentially important source of inequity in reproductive health outcomes. The fetal origins literature points to long-term consequences for infants associated with exposure to stress while in utero [89, 90], and evidence also suggests that maternal health during pregnancy is linked to later cardiovascular and metabolic outcomes [87, 88, 91]. Therefore, exposure to police violence during pregnancy may contribute to health disparities not only during pregnancy, but for maternal and infant health across the life course and intergenerationally.

In 2018, the American Public Health Association released a policy statement regarding law enforcement as a public health issue, including suggested steps that could reduce incidents of fatal police violence [70]. These include improving surveillance of fatal police violence by local public health departments, and shifting government priorities from addressing the symptoms of social inequity to engaging in primary prevention. For example, decriminalization of factors associated with social determinants of health, including homelessness, substance use, sex work, mental illness, and immigration, may reduce police interaction with marginalized groups who are at risk for becoming victims of police violence. Furthermore, redirecting funds from policing to investing in education, transportation, economic opportunities, treatment for mental illness and substance use, and access to affordable housing could help reduce health disparities and minimize the need for a persistent armed law enforcement presence in affected communities.

In addition, whether or not it is feasible to eliminate fatal police violence completely, clarifying how such incidents may or may not contribute to disparities in reproductive health outcomes is critical to understanding their etiology. Documenting the relationship between fatal police violence and reproductive health outcomes is necessary for the development of community-based and clinical interventions to improve birth outcomes. For example, evidence suggests that doula care and group prenatal care can be important sources of social support that may buffer the effects of stressful life experiences during pregnancy [170, 171, 173–175]. It is possible these types of interventions may also be beneficial for mitigating the effects of exposure to fatal police violence. Interventions that fail to acknowledge exposure to fatal police violence as a potential source of stress in womens lives may miss opportunities to improve birth outcomes.

	Death records			Fatal encounters		
	Total	Not exposed to fatal police violence	Exposed to fatal police violence	Total	Not exposed to fatal police violence	Exposed to fatal police violence
N	387,618	352,644	34,974	968,012	867,887	100,125
Race/ethnicity						
White	21.0%	21.1%	20.2%	22.2%	22.2%	21.6%
Black	5.8%	5.8%	5.9%	7.0%	7.0%	7.3%
Alaska Native or American Indian	0.4%	0.4%	0.5%	0.4%	0.4%	0.4%
Asian or Pacific Islander	9.5%	9.5%	8.6%	10.7%	10.8%	9.6%
Other or two or more races, non-Black	1.1%	1.1%	1.0%	1.2%	1.2%	1.2%
Multiracial Black	0.7%	0.8%	0.7%	0.8%	0.8%	0.9%
Hispanic	61.5%	61.3%	63.2%	57.7%	57.5%	59.0%
Educational attainment						
Less than high school	28.5%	28.3%	29.9%	26.8%	26.7%	27.7%
High school graduate	29.5%	29.5%	29.6%	29.0%	29.0%	29.6%
Some college	24.3%	24.3%	23.8%	25.1%	25.1%	25.0%
College graduate	17.8%	17.9%	16.7%	19.1%	19.3%	17.7%
Age (mean)	27.7	27.7	27.5	27.9	27.9	27.7
Insurance type						
Public	59.3%	59.2%	60.4%	58.0%	57.8%	59.7%
Private	37.6%	37.7%	36.7%	38.6%	38.8%	36.9%
Self-pay or other	3.1%	3.1%	3.0%	3.4%	3.4%	3.4%
Parity						
1	36.7%	36.7%	36.7%	37.6%	37.6%	37.5%
2	30.3%	30.3%	30.0%	30.5%	30.5%	30.1%
3 or more	33.1%	33.1%	33.3%	31.9%	31.9%	32.4%

Table 4.1: Sociodemographic characteristics of women living in a neighborhood with any incident of fatal police violence during the study period and by exposure status across data sources.

	Death Records		Fatal Encounters	
	Early preterm birth	Late preterm birth	Early preterm birth	Late preterm birth
	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
All	0.85 (0.75, 0.97)	1.05 (1.00, 1.09)	1.02 (0.95, 1.10)	1.03 (1.00, 1.06)
White	0.80 (0.57, 1.12)	0.97 (0.88, 1.08)	0.96 (0.80, 1.15)	1.07 (1.00, 1.14)
Black	1.22 (0.78, 1.91)	1.13 (0.94, 1.36)	1.08 (0.84, 1.40)	0.98 (0.89, 1.09)
American Indian or Alaska Native	-	0.85 (0.49, 1.46)	2.08 (0.92, 4.72)	0.77 (0.53, 1.13)
Asian or Pacific Islander	1.33 (0.79, 2.25)	1.13 (0.96, 1.34)	1.12 (0.85, 1.49)	0.92 (0.83, 1.03)
Other or two or more races, non-Black	2.23 (0.84, 5.91)	1.20 (0.82, 1.76)	1.36 (0.74, 2.50)	1.20 (0.95, 1.51)
Multiracial Black	1.04 (0.31, 3.50)	1.41 (0.83, 2.39)	1.10 (0.58, 2.07)	1.01 (0.77, 1.32)
Latina	1.01 (0.70, 1.46)	1.08 (0.96, 1.22)	1.06 (0.82, 1.29)	0.95 (0.89, 1.02)

Table 4.2: Hazards of early and late preterm birth associated with exposure to fatal police violence in one's Census tract during pregnancy, by exposure data source and race/ethnicity.

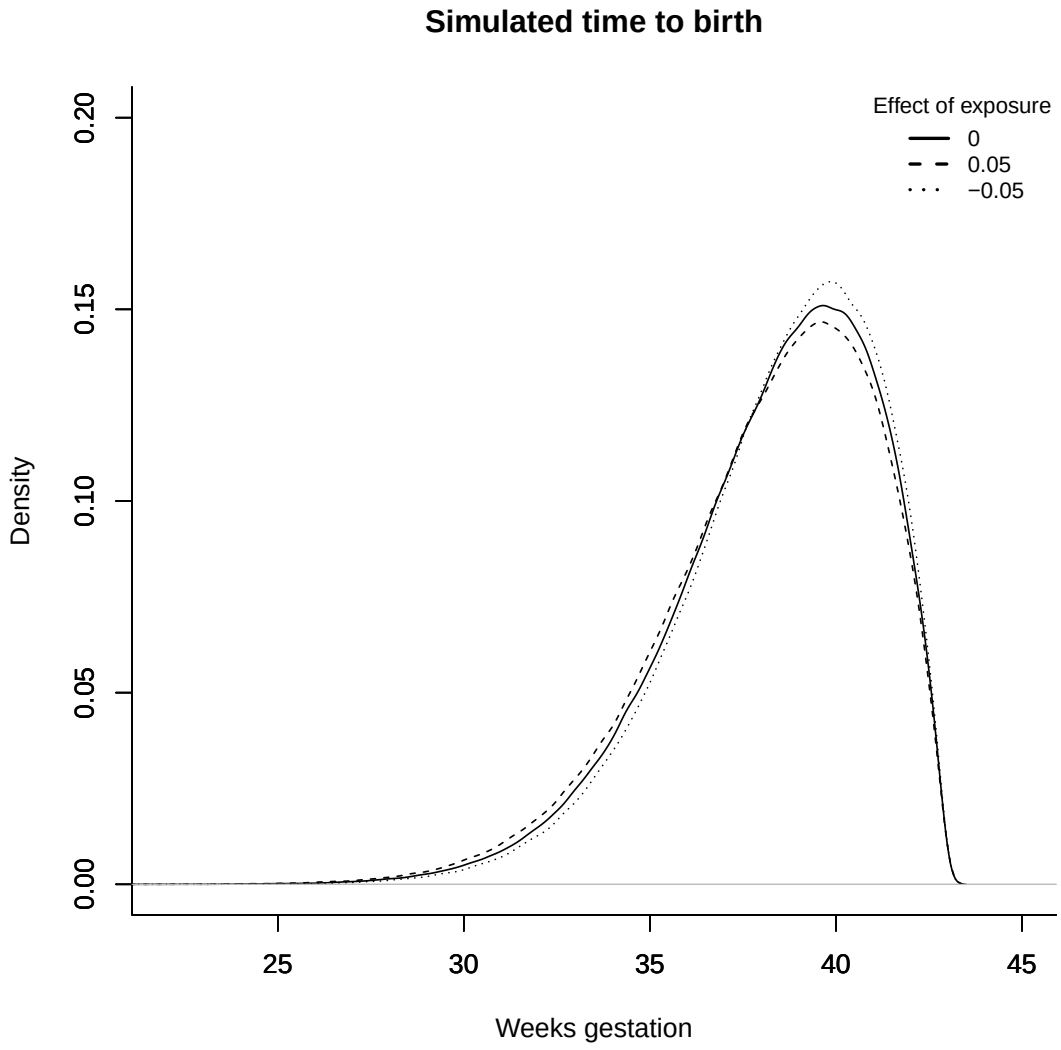


Figure 4.1: Changes in distribution of simulated time to birth associated with exposure.

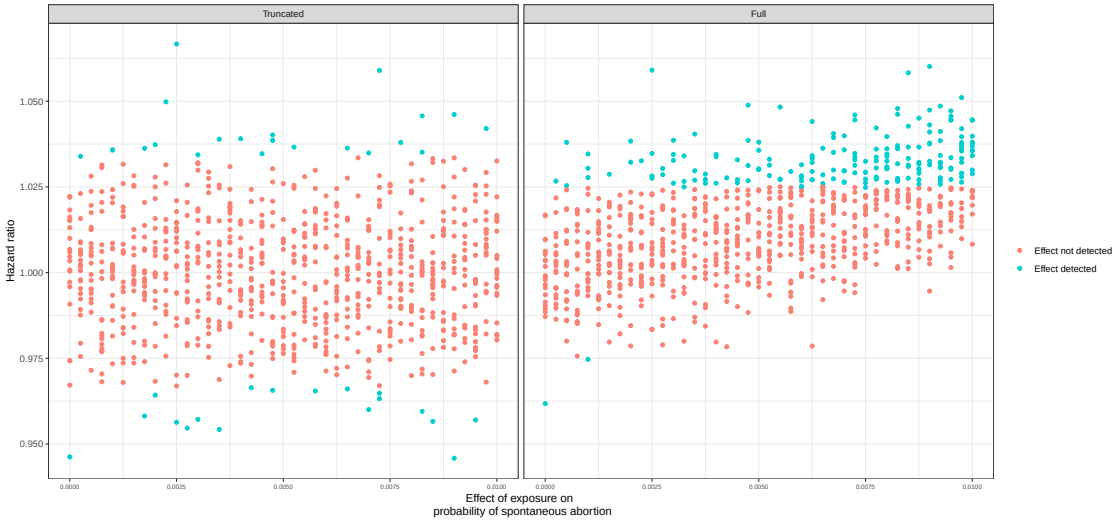


Figure 4.2: Hazard ratio and whether or not an effect was detected by full and left-truncated data when there is no effect of exposure on time to delivery by effect on risk of spontaneous abortion.

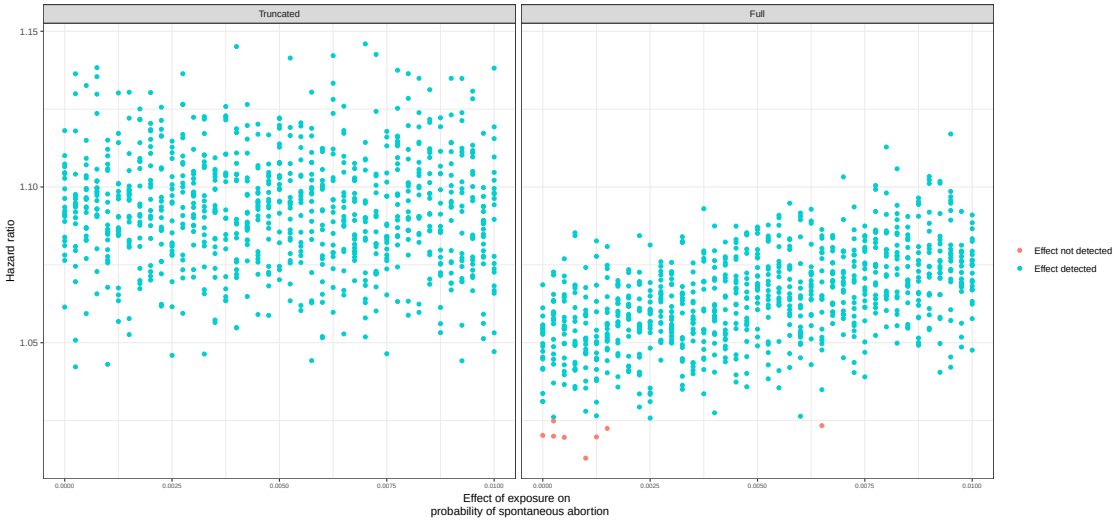


Figure 4.3: Hazard ratio and whether or not an effect was detected by full and left-truncated data when there is a negative effect of exposure on time to delivery by effect on risk of spontaneous abortion.

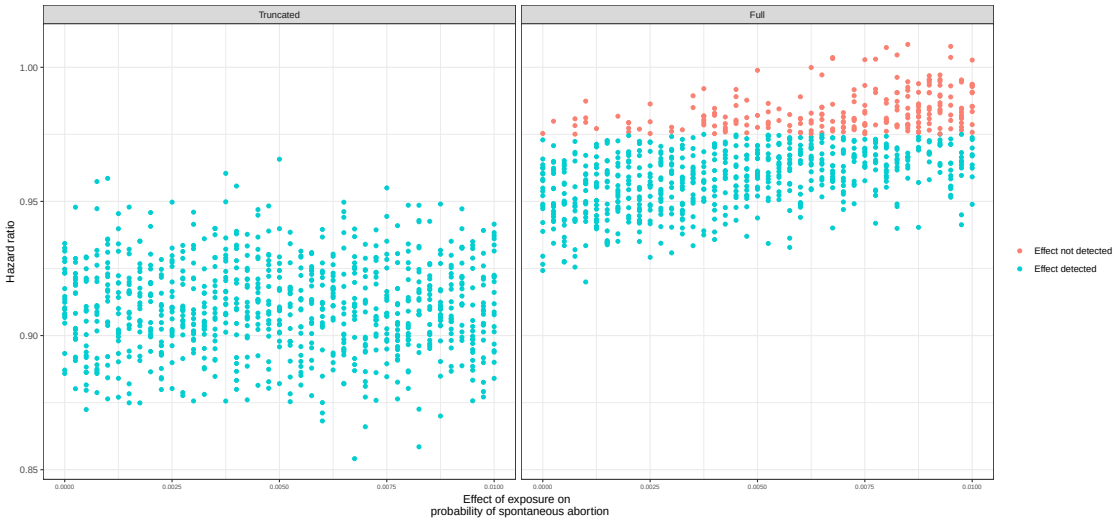


Figure 4.4: Hazard ratio and whether or not an effect was detected by full and left-truncated data when there is a positive effect of exposure on time to delivery by effect on risk of spontaneous abortion.

4.5 Supplemental Material

Results of the simulation by induced abortion

First, focusing on the simulation results under the scenario in which the exposure does not affect time to gestation among births that survive past 22 weeks (i.e., the effect of exposure on the time to birth = 0). We observed that the distance between the true hazard ratio for preterm birth and that estimated from the truncated data (in which the hazard ratio was only estimated among conceptions that survived past 22 weeks) increased as the effect of exposure on risk of spontaneous abortion increased (Supplemental Figure 4.5). As the true hazard ratio increased due to the effect of exposure on risk of miscarriage early in pregnancy, the truncated data did not reflect this and underestimated the hazard of exposure. This pattern was not appreciably changed by differences in the effect of exposure on the probability of censoring by induced abortion.

When the effect of the exposure on the outcome shortened time to birth (i.e., the effect on time to birth = 0.05), truncation due to excluding spontaneous abortions tended to overestimate the hazard ratio, especially when the effect of exposure on spontaneous abortion was small (Supplemental Figure 4.6). As the strength of the effect on spontaneous abortion increased, the truncated estimates of the hazard ratio approached the true estimate. The relationship of the exposure on induced abortion did not substantively change the pattern of results.

In the scenario in which the exposure lengthened the time to birth (i.e., the effect on time to birth = -0.05), the truncated estimate of the hazard ratio was consistently lower than the true hazard ratio (Supplemental Figure 4.7). The true estimate of the hazard ratio was below 1, and the truncated estimate was even lower; this distance widened as the effect of exposure on spontaneous abortion increased. There was no difference in this pattern according to the strength of association between the exposure and probability of induced abortion.

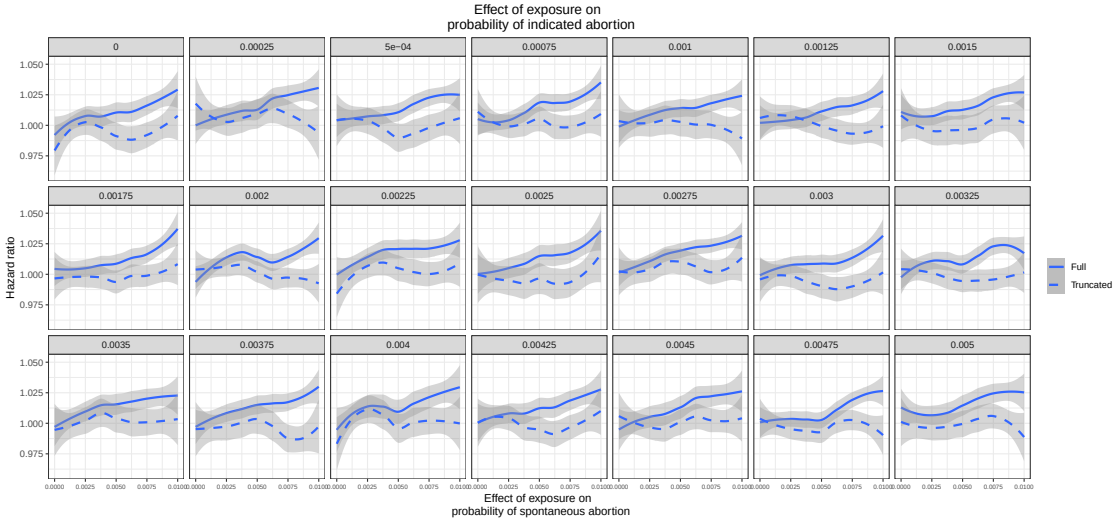


Figure 4.5: Bias in hazard ratio for preterm birth that occurs due to left-truncation and censoring, by strength of association between exposure and induced or spontaneous abortion, when there is no effect of exposure on time to birth.

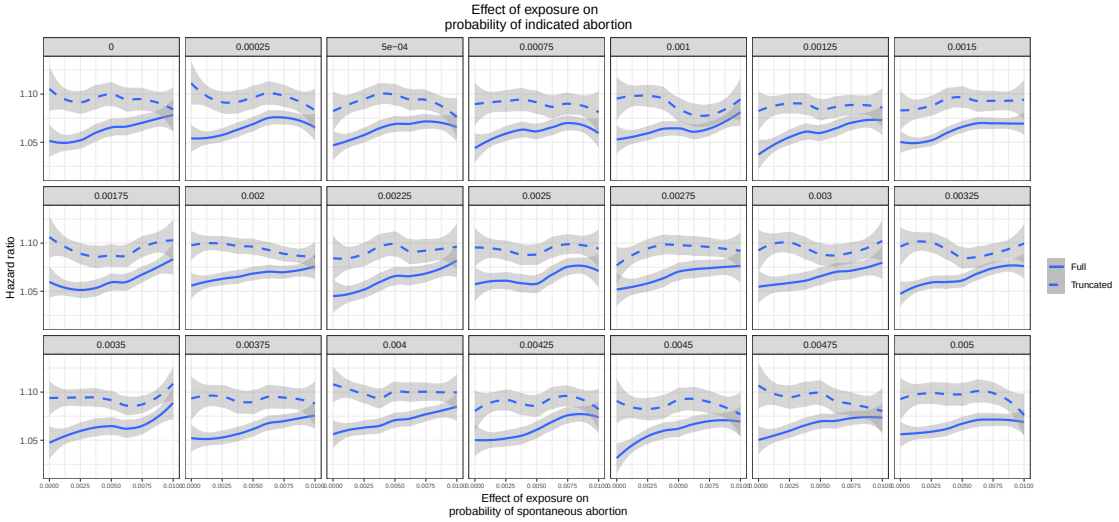


Figure 4.6: Bias in hazard ratio for preterm birth that occurs due to left-truncation and censoring, by strength of association between exposure and induced or spontaneous abortion when exposure shortens time to birth.

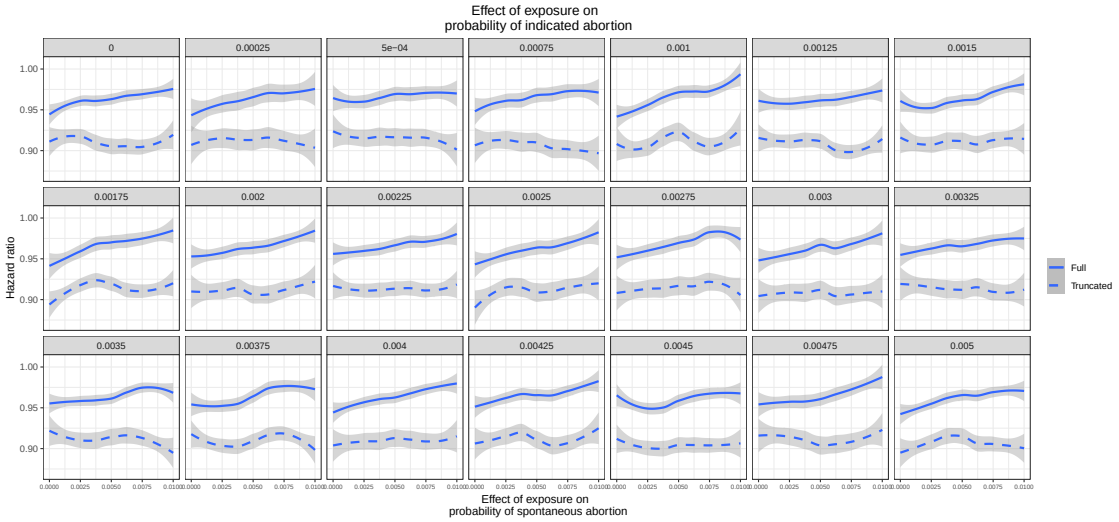


Figure 4.7: Bias in hazard ratio for preterm birth that occurs due to left-truncation and censoring, by strength of association between exposure and induced or spontaneous abortion when exposure lengthens time to birth.

Chapter 5

Conclusion

Community violence is a feature of the social environment driven by historical inequities and economic and social marginalization that have persisted to the current day. This dissertation demonstrates that both acute and chronic exposure to community violence can influence maternal and infant health, which has the potential to affect the intergenerational transmission of health disparities. Acknowledging community violence as a social stressor that affects pregnant women in addition to the victims themselves helps contextualize violence as a public health issue that deserves attention from policy-makers and clinicians. Even if it is not possible to eliminate exposure to community violence, identifying specific features of the social environment that are associated with health inequities is critical to understanding their etiology. Furthermore, awareness of community violence exposure as a risk factor for adverse pregnancy outcomes can assist public health practitioners who are engaged in developing and targeting interventions for both women and communities. The findings from this dissertation suggest that violence reduction efforts may have spillover effects on reproductive health.

Chapter 2 investigates acute exposure to homicide in a mother's Census tract during the pre-conception period and early pregnancy, and found that homicide exposure in the first trimester is associated with a small increased risk of having a baby that is born small for its gestational age. Stronger associations were observed among mothers who experienced three or more homicides during pregnancy, suggesting that repeated exposure to fatal violence in one's community can increase risk for intrauterine growth restriction, which has been tied to poorer health and achievement outcomes over the infant's life course. No associations were observed between homicide exposure and risk of preterm birth. These results were robust to negative controls, indicating that the findings were not a result of residual temporal patterning of violence and adverse birth outcomes.

Chapter 3 focuses on chronic exposure to firearm violence, and attempts to tackle the problem of structural confounding by using propensity score restriction to ensure exchangeability of the zip codes included in the study. The findings indicate there is an increased risk of preterm birth among mothers living in communities with high firearm violence. The mediation analysis utilizes a statistical method that estimates stochastic direct and indi-

rect effects, and permits inclusion of pre-pregnancy health indicators as exposure-induced mediator-outcome confounders. The results suggest that early preeclampsia, infection, and substance use during pregnancy partially mediated the association between exposure to high firearm violence and preterm delivery. Asthma during pregnancy and gestational diabetes were also elevated in neighborhoods with high firearm violence, as were pre-existing hypertension, diabetes, and maternal BMI greater than or equal to 30. These results suggest chronic exposure to firearm violence affects both maternal and infant health, which suggest potential long-term implications for the health and well-being of affected mothers and their children.

Chapter 4 examines the role of acute exposure to fatal police violence in a woman's Census tract during pregnancy on the hazard of preterm birth. Using a survival analysis framework addresses a challenge inherent in studies of exposures during pregnancy and preterm birth, namely that women who deliver preterm have less time during pregnancy to accrue exposure. The results show increased hazard of late preterm delivery associated with fatal police violence exposure. These results were consistent across two data sources that measured fatal police violence according to where the incident occurred and where the victim lived at the time of death. No effect modification by race/ethnicity was observed, which may have been due to a lack of statistical power.

The analyses in this dissertation contrast women exposed and unexposed to certain forms or levels of violence in their communities. The assumption made when articulating these contrasts is that if it were possible to remove "exposure", we could predict the change in risk for adverse birth outcomes at the population level. Reduction of community violence is a significant challenge, to say nothing of eliminating it altogether. Many governmental and public health organizations have identified violence reduction as a critical public health goal because of the substantial human and economic cost of deaths and injuries associated with it [114]. Therefore, implementing interventions that reduce violence are a public health victory in and of themselves. The fact that birth outcomes may also be improved would be an ancillary benefit of those interventions. However, the focus of this dissertation is not to study whether an intervention to reduce community violence could affect birth outcomes. Rather, this work poses a more etiological question by attempting to elucidate the ways in which historical policies and social institutions shape the social environment in ways that affect maternal and infant health.

Prior studies on this topic have primarily focused on cross-sectional studies of violence levels and birth outcomes across communities. These studies are limited by the co-occurrence of other neighborhood-level factors and neighborhood selection processes that also likely contribute to risk for adverse reproductive health outcomes. This dissertation attempts to isolate the effect of community violence of birth outcomes by leveraging temporal variation in violence exposure within communities, and by using propensity score restriction to ensure cross-neighborhood comparisons do not rely on extrapolation. Assessing the relationship between community violence, in which people are killed or injured within communities, and adverse birth outcomes, in which people are born with suboptimal health at the beginning of life, is critical to understanding how health inequities are reproduced over time and across

generations. This work contributes new evidence that helps clarify how features of the social environment affect health disparities at the population level.

This dissertation focuses on the role of violence in communities across California in explaining the geographic and social patterning of adverse pregnancy outcomes. The findings suggest that the effects of community violence are not singularly felt by victims and their families, but also impact the health of pregnant women living nearby. These results indicate that reducing levels of violence may contribute to improving birth outcomes in economically neglected neighborhoods. Furthermore, given the distribution of exposure to violence by race/ethnicity, the findings from this dissertation suggest that exposure to community violence contributes to disparities in maternal and infant health by race/ethnicity. To the extent that the effect of violence on birth outcomes is mediated by maternal stress, clinical interventions to provide support and resources to women early in pregnancy who may be exposed to community violence may also be effective in improving birth outcomes and reducing disparities.

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