Title

A population-based study of the epidemiology and influence of community violence on self-harm in California, 2005-2013

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By

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Committee in charge:

Professor Jennifer Ahern, Chair
Professor Maya Petersen
Professor Jennifer Skeem

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Abstract

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Professor Jennifer Ahern, Chair

Self-harm is a leading cause of morbidity and premature mortality in the United States and rates are increasing for reasons that are not well-understood. There is an urgent need to better understand the distribution and determinants of these worrisome trends and to identify effective interventions to mitigate rising rates of self-harm. A better understanding of the contribution of community-level contextual factors to self-harm incidence may help inform the design of effective prevention efforts. Community violence is an important social contextual factor that may affect self-harm, but studies to date are generally limited to small samples of adolescents and nonfatal, self-reported exposures and outcomes. Existing studies also suffer methodological limitations due to the strong correlation between community violence and other social contextual determinants of health such as income inequality.

The main objective of this dissertation was to characterize the epidemiology of self-harm in California, a large and diverse state with self-harm trends similar to those nationwide, and to systematically assess the relationship between exposure to community violence and risk of self-harm in statewide data. My first aim was to characterize trends in the epidemiology of total self-harm (completed suicide, attempted suicide and non-suicidal self-harm) and fatal self-harm (completed suicide) throughout California between 2005 and 2013, with particular focus on changes in rates and means of self-harm by demographic subgroup. My second aim was to quantify the association of exposure to overall levels of community violence with risk of self-harm and to estimate the impacts of specific changes in the distribution of community violence on self-harm corresponding to hypothetical interventions. My third aim was to quantify the association of acute increases from expected levels of community violence with risk of self-harm and to estimate the impacts of eliminating acute increases in community violence on self-harm.

To address these aims, I conducted three large, population-based studies: a descriptive study (Aim 1), a density-sampled case-control study (Aim 2), and combined case-control and case-crossover study (Aim 3). I used comprehensive statewide data on self-harm and community violence (homicide and assault) from death files from the California Department of Public Health Office of Vital Records and emergency department and inpatient hospital discharge records from the Office of Statewide Health Planning and Development (OSHPD) for the period 2005 to 2013. Cases included all deaths and hospital visits due to deliberate self-harm. Census-

based denominators were used to estimate age-adjusted rates of total and fatal self-harm overall and by age, sex, race/ethnicity, county, and method of self-harm ("means"). Controls were the cases themselves (case-crossover), or California resident participants of the American Community survey matched to cases on key confounders (case-control). Community violence was measured as the rate of deaths due to homicide and injuries due to assault in the Consistent Public Use Microdata Area of residence. I estimated parameters that avoid extrapolation and capture associations of specific changes in the distribution of overall levels of community violence and acute, within-community variation in violence with risk of self-harm.

Findings suggest that total and fatal self-harm increased substantially between 2005 and 2013 in California, rising 7% and 13%, respectively. Means of self-harm changed, trending away from firearms towards suffocation and drug poisoning. Overall trends mask substantial heterogeneity across subgroups, with particularly rapid increases observed for black, multiracial, and white Californians and some rural counties. After adjustment for confounders, reducing past-year community violence to the lowest monthly levels observed within each community over the study period was 30.1 (95% CI: 29.7 to 30.6) per 100,000 lower risk of nonfatal self-harm (approximately a 13% reduction in self-harm relative to the observed risk), but no difference in the risk of fatal self-harm. Associations for a parameter corresponding to a hypothetical violence prevention intervention targeting high-violence communities indicated a 5% decrease in self-harm at the population level. In the case-crossover study, 30-day periods with higher-than-expected levels of community violence were associated with a 1.2% increased risk of fatal self-harm (95% CI: 0.3, 2.1) and a 0.7% increased risk of nonfatal self-harm (95% CI: 0.4, 0.9).

To my knowledge, this is the first study to examine trends in rates and means of fatal and nonfatal self-harm by detailed demographic subgroups in California, and the first to study the association of exposure to community violence with self-harm in a population-wide dataset. Reasons for large increases or declines in self-harm in subgroups need to be understood. Appropriate public health programming should address high-risk subgroups. Changes in means of self-harm away from those that theoretically can be restricted towards those that are not feasible to restrict highlight the need to address fundamental causes of self-harm. This study strengthens evidence on the relationship between community violence and self-harm and on the health consequences of community violence. Future research should investigate reasons for differential associations by type of community violence, type of self-harm, age, and gender, assess critical time periods of increased risk of self-harm, and determine whether violence prevention efforts have meaningful impacts on self-harm.

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DEDICATION

To my mother, whose devotion to the underserved led me to find a career in public health, and to my father, whose scientific approach to all things gave me the mind to pursue epidemiologic methods, and to my husband, whose endless encouragement, companionship, and sense of humor have sustained me and helped me to persist. Without you three, nothing I ever do is possible.

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A version of Chapter 2 (Aim 1) of this dissertation was published in the *American Journal of Public Health*, volume 107, under the title, "Changing Patterns in Rates and Means of Suicide in California, 2005 to 2013". A version Chapter 3 (Aim 2) of this dissertation had been accepted to *Epidemiology*, under the title "Exposure to Community Violence and Self-ham in California: A Multi-level Population-Based Case-Control Study". I am grateful to my coauthors for facilitating this work and allowing its inclusion here.

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DISCLAIMER

The analyses, interpretations, and conclusions of this paper are attributable to the authors, and not to the California Department of Public Health or the National Institutes of Health.

CHAPTER 1: INTRODUCTION

Problem statement

Self-harm (attempted suicide, completed suicide, and non-suicidal self-harm) is a leading cause of injury and premature mortality in the United States and in California. In 2015, there were more than 44,000 deaths and 505,000 injuries nationally and more than 4,000 deaths and 49,000 injuries in California attributable to self-harm.[1,2] Rates of self-harm also vary substantially by population subgroup, with the highest risk among those with serious mental illness, early traumatic life events, and chronic physical conditions, as well as middle-aged white men, transgender individuals and sexual minorities, the unemployed, and residents of rural areas.[1,3–5] Beyond the immediate deaths and injuries, self-harm takes a toll on the families and friends of victims, and costs associated with fatal self-harm, including medical care and lost productivity, are estimated to be more than $400,000 per suicide.[6] Alarmingly, a 2012 study found that nearly 2% of all adult Californians—more than half a million—seriously thought about suicide in the previous year.[7]

Rates of self-harm are also increasing. In California, between 2005 and 2013, rates of fatal self-harm (suicide) and nonfatal self-harm increased 13% and 6%, respectively.[2] This overall increase parallels national trends, and while the reasons for these increases are not well-understood,[5] research suggests that several factors are possible contributors, including the increase in long-term morbidity, physical disability, and pain; rising rates of psychological disorder and substance abuse; declining job prospects; and growing social conflict, income inequality, and racial inequality.[5,8] Rising rates along with intensified focus on the role of firearms in society have increased attention on self-harm as an important population health issue.[5,9] However, research relevant to prevention continues to be under-prioritized,[10] and better understanding of the factors driving self-harm is needed.

Recent increases in self-harm suggest that the epidemiology of self-harm is changing, and that interventions to address self-harm may need to change as well. Public health interventions to prevent self-harm come in two main forms: those that target high-risk individuals and those that act at the population-level. Targeted interventions include adequate screening and treatment of mental and substance use disorders and intensive case management of those with previous self-harm, suicide attempts, or suicidal ideation.[3] Population-level strategies include restricting access to certain methods of self-harm such as firearms (“means restriction”), population-based screening and management of suicidal ideation and behavior, gatekeeper training, more generous unemployment benefits, and guidelines for media reporting of suicides.[3] Population-level prevention strategies hold promise because of their potential to prevent a large number of cases and to reach individuals who are not identified as high-risk and are therefore not affected by programs targeting high-risk groups. Recommended population-level strategies for prevention of self-harm often focus on means restriction. Although important, this approach has limitations because many suicides are completed with means that cannot be restricted, and increases in suicides in California have been driven primary by means which are less amenable to restriction.[1] The alteration of social environments provides one promising alternative avenue for intervention. A better understanding of community-level contextual factors that affect self-harm would be valuable for the development of alternative population-based prevention strategies.

Epidemiologic evidence suggests that exposure to community violence—an under-studied feature of the social environment—may place individuals at higher risk of self-harm.[11–17] However, existing studies are restricted in scope and have important limitations (see Chapters 3 and 4). To inform priority-setting for future targeted and population-level prevention programs, there is a need to understand how the epidemiology of self-harm is changing (Aim 1) and to identify factors that may be driving these changes (Aims 2 and 3). The main objective of this dissertation was to characterize the epidemiology of self-harm in California and to systematically assess the relationship between exposure to community violence and risk of self-harm.

Specific aims and organization of the dissertation

Detailed rationale for studying the epidemiology of self-harm in California and community violence as a social contextual risk factor for self-harm are provided in the chapters that follow.

<u>My first aim</u> was to characterize trends in the epidemiology of total self-harm (completed suicide, attempted suicide and non-suicidal self-harm) and fatal self-harm (completed suicide) throughout California between 2005 and 2013, with particular focus on changes in rates and means of self-harm by demographic subgroup. This Aim is addressed with a descriptive study in Chapter 2.

<u>My second aim</u> was to quantify the association of exposure to overall levels of community violence with risk of self-harm and to estimate the impacts of specific changes in the distribution of community violence on self-harm corresponding to hypothetical interventions. This Aim is addressed with a large, population-based case-control study in Chapter 3.

<u>My third aim</u> was to quantify the association of acute increases from expected levels of community violence with risk of self-harm and to estimate the impacts of eliminating acute increases in community violence on self-harm. This aim is addressed with a combined case-control and case-crossover study in Chapter 4.

Chapter 5 discusses overarching themes of the three studies and presents concluding remarks.

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CHAPTER 2: CHANGING PATTERNS IN RATES AND MEANS OF SELF-HARM IN CALIFORNIA, 2005-2013

Introduction

Suicide is a leading cause of premature mortality in the United States (US), accounting for 42,773 deaths in 2014.[1] For reasons that are not well-understood, suicide rates have increased 23% from 10.5 per 100,000 in 1999 to 12.9 per 100,000 in 2014,[1] with rates nearly three times higher in certain groups such as sexual minorities and older white non-Hispanic men.[1,2] Previous studies suggest that suicide rates are also unacceptably high among those with mental illness, early traumatic life events, and chronic physical conditions, as well as transgender individuals, the unemployed, and residents of rural areas.[3,4] In addition to the toll that suicide takes on the friends and family of suicidal individuals, costs associated with suicide including medical care and lost productivity are estimated to be more than $400,000 per suicide.[5] Rising rates along with intensified scientific and media focus on gun control have increased attention on suicide as an important population health issue.[6,7] Despite the increasing burden of total self-harm (attempted suicide, completed suicide, and non-suicidal self-harm together), it continues to be under-prioritized in research,[8] especially investigations necessary for the design and implementation of population-level prevention initiatives.

Recommended population-level strategies for prevention of self-harm often focus on restricting access to certain methods of self-harm ("means") such as firearms. These interventions can be extremely effective, reducing fatal self-harm rates by as much as 33% in some settings.[9] To implement effective means restriction strategies, timely information on means of self-harm is needed. Approximately half of fatal cases of self-harm in the United States are completed with firearms,[1] but a growing proportion of fatal and the vast majority of non-fatal self-harm involve other means, such as drug poisoning and suffocation.[1] In fact, the proportion of suicides completed with firearms has steadily declined, and as of 2014, non-firearm means now account for the majority of completed suicides.[1] These trends, along with increasing overall rates, highlight that the epidemiology of self-harm is changing, and prevention programs and policies may need to adjust as well. Given these changes, it is important to examine patterns in means of self-harm in more detail. Additional information on who is at risk and via what means is a critical input for public health policy decision-making.

Recent developments in self-harm in California are of interest for public health nationally for several reasons. First, demographic factors such as race/ethnicity and immigration status are strongly associated with self-harm.[3,4,10,11] While currently less diverse, the US population is expected to comprise greater proportions of racial/ethnic minorities and foreign-born individuals in the future and to appear more similar in composition to California.[12] Thus, California's experience may help guide planning for future trends in self-harm nationally. Second, in 2014, just under 50% of suicides in the United States were attributable to firearms, while in California, only 38% of suicides were by firearms.[1] California has some of the most restrictive firearm policies in the country, and thus patterns may be informative about developments that might be expected nationwide if such policies were more broadly adopted. Finally, 12% of Americans (39.1 million) live in California and the state's size and diversity, including substantial multiracial and American Indian populations, allows for stable estimation of rates of self-harm in important, high-risk subgroups. Thus, findings for the state may reveal important information relevant to high-priority groups and the country as whole.[13]

To our knowledge, only two publications systematically addressed the epidemiology of self-harm in the California context. The first examined firearm suicides between 1997 and 1999 among young adults below age 21.[14] The second is a RAND Corporation report published in 2014 that described trends in suicide fatalities by age, sex, and region, with a brief discussion of implications for state prevention and early intervention programs.[15] There is a need for more detailed information on the epidemiology of self-harm, particularly among minority groups, at the local level, and for non-fatal outcomes. The California Mental Health Services Authority and county health departments, increasingly funded by the Mental Health Services Act of 2004, are actively developing and implementing new self-harm prevention initiatives.[16] Thus, a more detailed, timely, and rigorous assessment that examines different means, includes non-fatal outcomes, and investigates more specific subgroups provides valuable input for the design of such programs which may be helpful both in California and nationally.

This analysis systematically examines how rates and means of self-harm in California vary across people and place from 2005 to 2013. The overall aim of this description of patterns and trends in self-harm is to inform priority-setting for targeted and population-level prevention programs.

Methods

We identified all cases of fatal and non-fatal self-harm in California between January 1, 2005 and December 31, 2013 using CDC-recommended classifications[17,18] and data from two sources: death files from the California Department of Public Health, Office of Vital Statistics and hospitalization (emergency department and inpatient hospital discharge) records from the Office of Statewide Health Planning and Development (OSHPD). This time period was selected because emergency department discharge records are not available prior to 2005, and 2013 is the most recent year of disaggregated data available from both sources. To avoid unstable rates and protect against identifiability, we restricted the study population to Californians aged 15 to 84 at the time of death or injury.

Records included information on the means of self-harm, age, race/ethnicity, sex, and zip code of residence. Means in death records were classified with the International Classification of Diseases (ICD), Revision 10 for the primary cause of death, and hospital utilization records were classified with ICD-9 for up to five causes of hospital visit. We grouped means of self-harm into nine categories (see appendix for ICD codes): poisoning by medicinal substances or drugs; poisoning by non-medicinal substances or drugs; hanging, strangulation, or suffocation; drowning; handguns; other firearms or explosives; sharp objects, cutting, or piercing; falls; and “other” means including “intentional self-harm by other specified means” such as electrocution, or exposure to extreme cold, “late effects of self-inflicted injury”, or “lying or jumping in front of a moving object” such as a motor vehicle or train. Non-suicidal self-harm could not be distinguished from suicide attempts in hospital utilization records. Thus, analyses including non-fatal outcomes incorporate all forms of self-harm sufficiently serious to result in a hospital visit.

We estimated rates of total and fatal self-harm overall and by age, sex, race/ethnicity, year of death or injury, county, and urbanicity of residence by dividing the number of cases in death and hospitalization records by Census and American Community Survey-based interpolated population estimates. We grouped race/ethnicity into six categories: Hispanics of all races and non-Hispanic American Indian, Asian or Pacific Islander, black, multiracial, and white. Rates were age-standardized to the state age distribution in 2010 using the direct method and 5-year age groups. We characterized patterns of total and fatal self-harm by tabulating, plotting, and mapping estimated rates to identify important trends and patterns. We also examined temporal trends for evidence of age-, period-, or cohort-effects (presented in appendix).

Data analysis was conducted using SAS 9.3 and R 3.2.1 (R Foundation for Statistical Computing, Vienna, Austria). We used the “epitools” package[19] for age standardization and the “ggplot2” package[20] for making graphics. This study was approved by the State of California and University of California, Berkeley Committees for the Protection of Human Subjects.

Results

Between 2005 and 2013, there were 374,404 cases of non-fatal self-harm resulting in hospital visits and 30,029 cases of fatal self-harm in California. Fatal and non-fatal self-harm occurred in all ages, sexes, and racial/ethnic groups, but the distribution was extremely heterogeneous. In 2013, 7.8% of all recorded self-harm was fatal, but this proportion varied substantially by age and sex, ranging from 0.6% in women aged 15 to 19 to 50.2% among men aged 75 to 79.

Both the rate and composition of means of total and fatal self-harm changed over the study period. The rate of total self-harm increased 6.5% from 150.4 to 160.2 per 100,000, and the rate of fatal self-harm increased 12.6% from 11.2 to 12.6 per 100,000. In terms of total self-harm, the proportion by firearms remained steady around 2.7%, the proportion by drug poisoning declined from 60.1% to 54.5%, and the proportion by cutting/piercing and “Other” means increased from 20.8% to 22.8% and 8.0% to 11.0%, respectively. In terms of fatal self-harm, the proportion by firearms decreased from 29.0% to 26.9% while the proportion by drug poisoning and suffocation increased 15.9% to 17.1% and 25.8% to 29.0%, respectively.

Figure 1 presents levels and trends in age-adjusted rates of total self-harm and fatal self-harm by means and sex. Total self-harm was more common among women, but fatal self-harm was substantially more common among men. Rates of total self-harm increased for both men and women between 2005 and 2013, but the composition of means varied. Among women, the vast majority of total self-harm involved drug poisoning or cutting/piercing, and fatal self-harm was predominantly completed with drug poisoning or suffocation. Among men, drug poisoning and cutting/piercing were also the most common means of total self-harm, but “other” means and firearms played a larger role than for women for both total and fatal self-harm.

Figure 2 depicts levels and trends in age-adjusted rates of total and fatal self-harm by means and race/ethnicity. The composition of means of total and fatal self-harm was similar for all racial/ethnic groups, with the exception of American Indians for whom use of firearms declined substantially and use of suffocation and “other” means increased markedly. Of note, multiracial individuals (comprising 2% of the California population) experienced a 137% increase in fatal self-harm, beginning with the lowest rate of all racial/ethnic groups in 2005 (5.3 per 100,000, or 25 cases) and reaching the second highest rate by 2013 (12.6 per 100,000, or 81 cases). Total self-harm among the same multiracial group was consistently high throughout the study period.

Also of note, fatal self-harm among black individuals remained relatively constant over the study period while total self-harm increased rapidly from 173.8 to 226.4 per 100,000 between 2005 and 2011, a 30% increase in just six years.

Figure 3 presents levels and trends in rates of total and fatal self-harm by means and age group. Among younger populations, there is far more total self-harm but relatively little fatal self-harm, while in older populations, there is far less total self-harm but a much higher proportion is fatal. Directions in trends of self-harm also varied markedly by age group. For example, the fatal self-harm rate increased dramatically between 2005 and 2009 and then declined between 2009 and 2013 in ages 45-59, while it increased slightly throughout the study period for ages 15-29 and 30-44, increased consistently and substantially for ages 60-74, and generally decreased in ages 75-84. Drug poisoning was the most common means for total self-harm in all age groups; the second most common means was cutting/piercing at younger ages and firearms at older ages. In terms of means of fatal self-harm, suffocation was the most common means in younger age groups, while firearms and “other” means were more common at older ages. In the appendix, we present figures investigating potential age, period and cohort effects, and find that for total self-harm, within each of the youngest age groups, cohorts born in more recent years experienced notably higher rates of total self-harm, while other age groups showed no cohort effects. In contrast, for fatal self-harm, cohort effects were observed in almost all age groups, but the direction of effects varied substantially depending on the age group.

Figure 4 presents maps of age-adjusted rates of total and fatal self-harm in 2013, and change in rates between 2005 and 2013, by county. Massive disparities in total and fatal self-harm rates are immediately apparent, with rural counties generally experiencing much higher rates than urban counties (generally the northern and eastern areas of the state are less urban). Rapid increases and rapid decreases in both large and small counties are also apparent during the eight-year period (see appendix Figure 5). Some counties had high levels of total self-harm but low levels of fatal self-harm and vice versa. In the appendix, we present maps displaying the number of cases of self-harm by county, which have inverse spatial patterning to that of rates, highlighting that most cases occur in urban areas where rates are generally lower, while rural regions have smaller caseloads but manifold greater rates. The appendix also presents plots of variation in the dominant means of self-harm by county and the relationship between baseline rates and change in rates during the study period.

Discussion

In this study, we systematically assessed the epidemiology of total and fatal self-harm by means and demographics for the period from 2005 to 2013 in California. We found heterogeneous and alarming trends across geographic and demographic groups that indicate the need for increased efforts to mitigate rising rates of total and fatal self-harm among all groups but particularly among those with high or rapidly increasing rates: the youngest, the oldest, black, American Indian, white, and multiracial individuals, and residents of rural counties.

Findings in context

Consistent with past literature on national patterns, our results indicate higher rates of total self-harm among women and younger groups, with more fatal self-harm among men, older groups, whites, and American Indians.[1,3,4] However, other results were distinct from past studies. We documented dramatically increasing rates of total self-harm among blacks, suggesting that the "black-white suicide paradox"[21] may be disappearing. This paradox observes that blacks experience more risk factors for but lower rates of suicide than whites. As of 2013, the gap between black and white rates of total self-harm was eliminated due to increasing rates in blacks. The disparity between the black and white fatal self-harm rates persists in California, though it is closing nationally due to increases in young black people,[22] and thus should continue to be monitored. We also identified substantial but previously unrecognized increases in fatal self-harm among multiracial individuals. These developments warrant an immediate public health investigation and response.

Also undocumented in past studies, blacks and multiracial individuals exhibited opposing trends in total and fatal self-harm; among blacks, total self-harm increased but fatal self-harm was steady, while among multiracial individuals, total self-harm was steady, but fatal self-harm increased. These developments are plausible, given that total and fatal self-harm exhibited such disparate trends throughout the study. However, different reporting of race/ethnicity in mortality files compared with hospitalization files may explain this pattern.[23] The same groups may be at increased risk for both total and fatal self-harm, but may be classified differently in different records.

Among age-specific patterns, increasing rates of self-harm among the youngest age group (Figure 3) and among younger cohorts within the youngest age groups (appendix Figure 1) are of substantial concern. Similar trends have been documented nationally for earlier time periods,[24] but more recent patterns and cohort effects are under-recognized. Changes in the prevalence of substance abuse, untreated depression, young adult veterans with mental illness, and use of internet and social media in ways that can promote suicidal behavior may contribute to these patterns.[25–28] In addition, the dramatic peak and decline in suicide which is only present for ages 45-59 may be related to the Great Recession;[29] future research should assess this hypothesis.

Implications for intervention and policy

Consistent with national trends,[1] we found that means of fatal self-harm are shifting away from firearms, which are theoretically easier to restrict, towards means such as suffocation, which would not be feasible to restrict. This pattern is particularly prominent among American Indians, and merits further investigation. The movement towards less restrict-able means highlights the need to better understand and address the fundamental causes of suicide and self-harm. For example, the development and implementation of interventions that address key risk factors such as early life trauma,[3,22] poor quality of life due to chronic physical conditions,[30] and lack of support for those experiencing acute psychosocial crises due to life events such as job loss or loss of a loved one[31] could have important long-term impacts on self-harm. Adequate screening and treatment of psychiatric disorders and substance abuse can also reduce long-term suicide risk,[32–34] as opposed to addressing acute suicidal episodes. Such efforts may achieve more broad-reaching and long-lasting results.

Prevention strategies for suicide and self-harm fall into two major categories: those targeting high-risk groups and population-level strategies. This study may help inform targeted approaches by identifying high-risk groups and the types of risk (fatal or non-fatal, by which means) in need of further intervention. However, this study also shows that many groups are high-risk and which groups are high-risk is changing, in some cases very rapidly, which may make targeting high-risk groups challenging. Thus, population-level strategies such as broad screening and education in primary care[35,36] and schools[37,38] as well as guidelines for media reporting of suicidal behavior[39,40] should also be prioritized. These approaches also have the benefit of reaching individuals who might not be identified as high-risk. Given the observed shift away from restrict-able means, population means restriction strategies may not be as effective as alternatives, except in particular subgroups.

Areas for future research

This study indicates the need for research on the causes of recent increases in self-harm. Possible explanations that merit further investigation include the rising proportion of people living with long-term morbidity, physical disability, and pain; rising rates of psychological disorder and substance abuse; declining job prospects; and growing social conflict, income inequality, and racial inequality.[6,29,41,42] An important question that could not be accurately addressed with the existing data is whether the changing composition of means is driven by means substitution. Further investigation of this question is vital to identify the most appropriate future prevention efforts—namely, whether additional means restriction efforts are expected to be effective. Future research should also identify the determinants of geographic variation in suicide rates. There is potentially much to be learned about successful and unsuccessful prevention approaches by comparing circumstances and programs across counties with rapidly increasing or rapidly decreasing rates of self-harm. Of particular interest are the effects of variation in county mental health services programming and investments, such as changes following the implementation of the California Mental Health Services Act of 2004. Finally, future research should assess whether the declining role of firearms in self-harm in California is attributable to California's firearm laws.

Strengths and limitations

Naturally, this study was subject to several limitations. First, this study did not capture self-harm that did not result in death or medical attention at a hospital and therefore underestimates the true rate of self-harm. At the national level, the self-reported prevalence of suicide attempts in national surveys is notably higher than that based on hospital records, and the same is likely true in California.[11,43] Thus, reported rates are likely underestimates and should be interpreted as lower bounds. Second, we were unable to distinguish between non-fatal suicide attempts and non-suicidal self-harm. These behaviors may have different characteristics, and our combination of the two may explain some of the differences we observed between total and fatal self-harm. Third, cause of death classification on death certificates is known to be imperfect. Suicides may have been miscoded as homicides or unintentional deaths, and vice versa. However, studies that have examined this issue in greater detail generally conclude that that the degree of misclassification is not substantial enough to alter major trends and patterns.[44,45] Fourth, we were only able to report trends through 2013, the most recent year of data available for research.

Additional investments in health information systems for states that allow for more timely preparation of data for research, evaluation, and planning on important health topics would be a valuable investment in future public health.

These weaknesses are balanced by several important strengths. We were able to examine both fatal and non-fatal self-harm. The limited existing evidence contrasting population-level patterns in fatal and non-fatal self-harm suggests that the nature of fatal and non-fatal self-harm are very different, and both need to be examined and understood for appropriate public health response.[3,4,46] In addition, the data were population-wide over the period of almost a decade, and the large population size allowed us to compare relatively rare outcomes among important subgroups groups for whom previous assessments have been limited.

Conclusions

In sum, results from this study show a clear need for additional efforts to address self-harm. This study highlights groups in need of prioritization and should aid in raising awareness and helping health professionals, funders, and decision-makers set priorities. Evaluations of self-harm prevention efforts will need to appropriately account for important variation in risk across groups and secular trends in rates and means underscored in this study. A key tension will be to balance prioritizing intervention in urban areas where the most cases occur with prioritizing rural areas where rates are highest.[15] Arguments for efficiency would point to the former while arguments for social justice would point towards the latter. We identify the who, where, when, and how of self-harm. Now, a better understanding of the factors driving self-harm, and the identification and implementation of effective treatment and prevention strategies is needed to put this information to good use to mitigate rising rates of self-harm.

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Tables and figures

Figure 1: Trends in age-adjusted rates total self-harm and fatal self-harm, by means and sex, California, 2005-2013

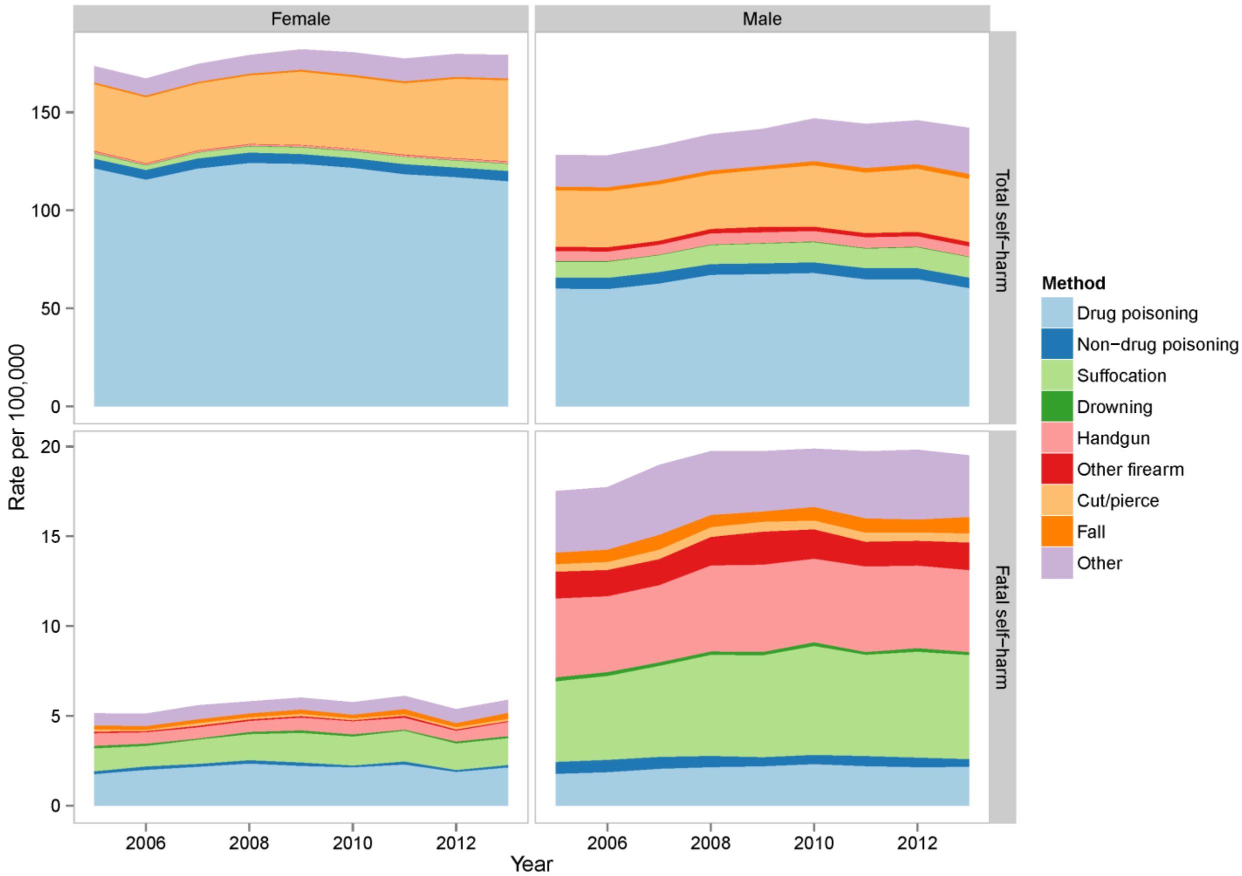


Figure 2: Trends in age-adjusted rates of total self-harm and fatal self-harm, by means and race/ethnicity, California, 2005-2013

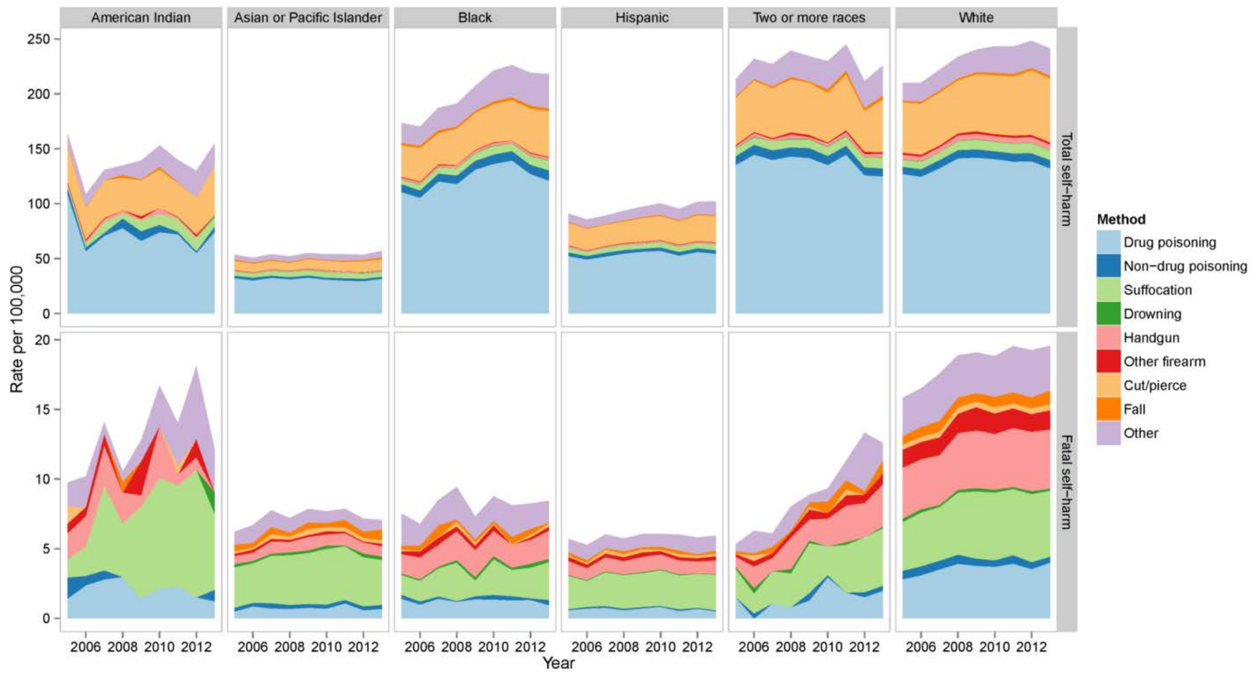


Figure 3: Trends in rates of total self-harm and fatal self-harm, by means and age group, California, 2005-2013

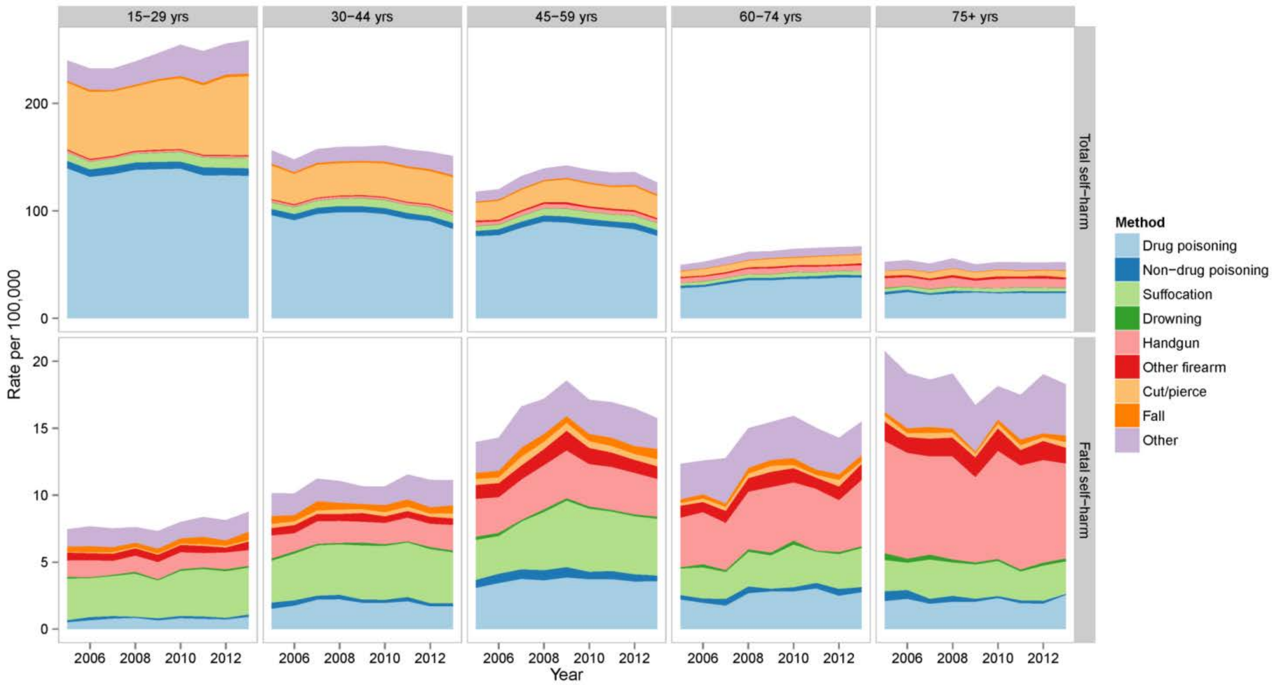
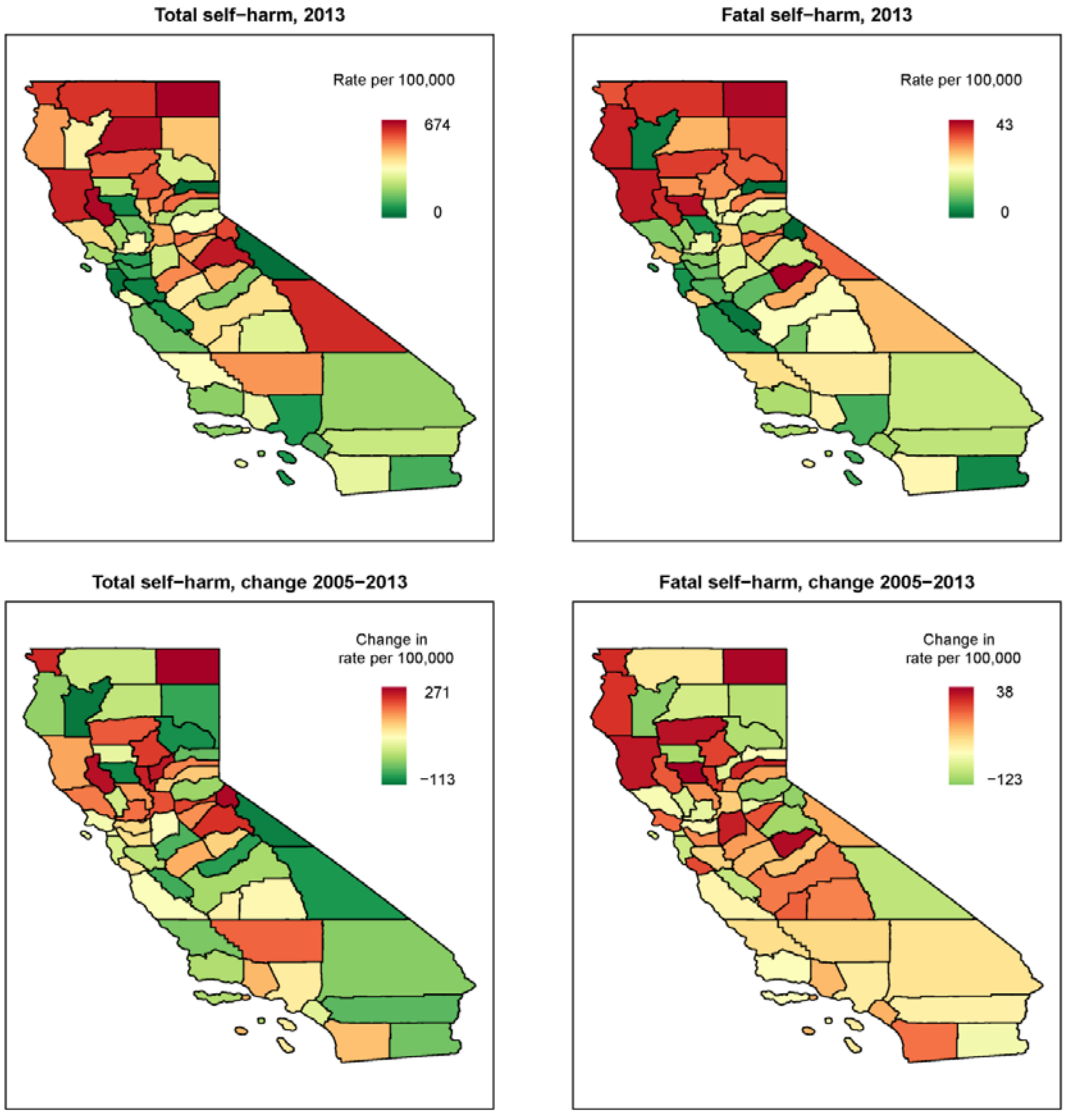


Figure 4: Age-adjusted rates of total self-harm and fatal self-harm in 2013 and change in rates 2005 to 2013 in California counties



CHAPTER 3: EXPOSURE TO COMMUNITY VIOLENCE AND SELF-HARM IN CALIFORNIA: A MULTI-LEVEL POPULATION-BASED CASE-CONTROL STUDY

Introduction

Self-harm is a leading cause of morbidity and mortality in the United States (US), accounting for more than 44,000 deaths and 505,000 injuries in 2015.[1] Between 2001 and 2015, rates of fatal self-harm (suicide) increased 24% and rates of nonfatal self-harm increased 39%.[1] The reasons for these increases are not well-understood. Rates of self-harm also vary substantially by population subgroup. For example, compared to the general population, fatal self-harm rates are nearly three times higher among older men and nonfatal self-harm rates are four times higher among young women.[1] Rising rates have drawn attention to self-harm as an important population health issue.[2,3] Additional research is needed to understand the drivers of self-harm and to identify effective interventions.[4]

Aspects of the social environment such as social fragmentation and inequality are key risk factors for self-harm.[5] Community violence is an important and modifiable feature of the social environment[6] that may contribute to the burden of self-harm, particularly in the US where levels of community violence are high and rising.[7] Exposure to community violence, meaning witnessing, hearing about, or directly experiencing violence in one's community,[8] may increase the risk of self-harm in several ways. Increased stress, depressed mood, anxiety, symptoms of post-traumatic stress, and mental disorders can result from exposure to community violence[9–14] and are strong risk factors for self-harm.[15–20] Similarly, exposure to community violence can lead to substance use[21,22] or social isolation (e.g. staying inside),[23] thereby increasing risk for self-harm.[24–27] Moreover, exposure to community violence can normalize violence and aggression, another important risk factor for self-harm.[28,29]

Epidemiologic research on the relationship between community violence and self-harm is limited.[30–36] Although positive associations between community violence and suicidal ideation or nonfatal suicidal behavior have been observed, existing studies are generally limited to small samples of urban adolescents. To our knowledge, no previous studies have quantified the association of community violence with self-harm in a general population. Moreover, no studies have examined both fatal and nonfatal self-harm in the same population, which is critical because these forms of self-harm appear to differ in their distribution and determinants.[1,37] Finally, no studies have estimated parameters corresponding to the potential impacts of specific reductions in community violence, which are particularly informative for public health decision-making.

Existing studies also suffer methodological limitations, making it difficult to draw meaningful conclusions. In particular, community violence is strongly associated with other features of communities that are also associated with self-harm (e.g. economic opportunity). This makes it difficult to disentangle the effects of community violence from such factors.[38] When these factors are controlled using standard regression methods, the analysis often relies on extrapolation beyond the observed data, which can bias the results.[39] Previous studies have also relied on self-reported measures of community violence exposure and suicide-related outcomes. This approach can introduce same-source bias, where self-report of both the exposure and outcome leads to spurious associations due to correlated measurement error (for example, due to pessimistic outlook).

In this study, we assessed the association of exposure to community violence with fatal and nonfatal self-harm, overall and by age and gender. We applied a population-based case-control design to a large dataset including all deaths and hospital visits in California, a state with self-harm trends similar to those nationwide. We estimated risk difference parameters that avoid extrapolation and are relevant to potential public health interventions.

Methods

Data and study design

We compiled data on self-harm and community violence for the period 2005 to 2013 from two sources: deaths records from the California Department of Public Health Vital Records and emergency department and inpatient hospitalization discharge records from California's Office of Statewide Health Planning and Development. Records included all deaths and hospital visits statewide, except active duty military hospitals, and captured the external cause of death or injury (i.e. coding as due to accidents or violence including environmental events, circumstances and conditions as the cause of injury, poisoning, and other adverse effects), demographic characteristics, and residence of the patient or decedent. External cause of injury coding in California's hospital discharge records is mandatory, subject to ongoing quality assurance measures, and considered 100% complete.[40] Studies also indicate completeness and validity of external cause of mortality codes for homicide and self-harm in mortality data.[41] Emergency department records are not available prior to 2005.

We treated the residents of California as a cohort and conducted a population-based, nested case-control study.[42,43] Cases were all deaths and hospital visits due to deliberate self-harm in California, 2006-2013 (ICD-9-CM hospital visit code: E95; ICD-10 death codes: X6-X8). Self-harm outcomes were included starting in 2006 so that data on community violence were available for the relevant pre-injury exposure period (see Exposure assessment). We made efficient use of an existing population-representative sampling frame by sampling population-based controls[42] from California resident participants in the American Community Survey (ACS). The ACS is an ongoing, nationwide survey conducted by the US Census Bureau. It is designed to generate population-representative small-area estimates of demographic, economic, and social indicators over time. ACS interviews were conducted with between 170,000 and 220,000 Californians annually between 2006 and 2013.

We created a state-representative pseudo-population of control units by duplicating each ACS record by the corresponding ACS person weight[44] and drew controls from this expanded dataset. For statistical efficiency,[43] we matched 4 controls to each case on confounders strongly associated with self-harm: gender, race/ethnicity, 5-year age group, and year of survey/injury. We used population-based controls to avoid the possibility of Berkson's bias that could result from hospital- or death record-based controls.[42] We assumed that selected controls were not also cases at the time they were selected as controls; this is reasonable because self-harm risk was low (<0.5% in all matching strata). We restricted to individuals residing in California at the time of survey/injury and to those aged 15 to 84 years due to small numbers outside that age range.

Exposure assessment

Exposure to community violence was defined as the average of the monthly rate of deaths due to homicide (ICD-10 death codes X85-X99, Y00-Y09, Y35, U01, U02, Y871) and injuries due to assault (ICD-9-CM hospital visit codes E960-E969, E970-E977) in the Consistent Public Use Microdata Area (CPUMA) of residence for the 12 months prior to survey/injury. CPUMAs are mutually exclusive and collectively exhaustive geographic units designated by the US Census Bureau. CPUMAs include at least 100,000 residents and are consistently defined over the study period. There are 110 CPUMAs in California. In urban areas (95% of the California population), CPUMAs correspond to known neighborhoods (e.g. Chinatown in San Francisco). In rural areas, CPUMAs are counties or aggregations of small counties.

Decedent addresses from vital records were geocoded to the CPUMA of residence. Patient zip codes from hospital records were assigned to the corresponding CPUMA of residence using a geographic crosswalk.[45] We selected CPUMAs, instead of census tracts or zip codes, to define neighborhoods because they are locally-recognized places of residence but are large enough for stable estimation of monthly community violence rates. CPUMAs are also the smallest geographic identifier available in the ACS. We used objectively measured rates of community violence because they are strongly correlated with frequency of experiences of direct injury and witnessing violence reported by residents[46] while avoiding same-source bias. We used Census-based population estimates equivalent to the ACS pseudo-population as denominators to calculate rates.

We used the average monthly violence rate over the 12-month period immediately prior to occurrence of self-harm for each case and selection of each corresponding control, because we conceptualize community violence as a chronic predisposing factor that theoretically can interact with acute stressors (e.g., psychosocial crisis) to cause self-harm.[47] The 12-month time frame is a proxy for longer-term exposure, given its strong association with multiyear measures (e.g. $R>0.95$ with 36-month measure). A 12-month exposure ensured that seasonality did not impact the results, without extending so far back in time that residential mobility introduced excessive measurement error (within a year, 14% of people move and only 5% change counties of residence).[48] Crime data may also be used to measure community violence, but differences in reporting practices between jurisdictions and over time may introduce bias.[49,50] Victimization surveys are also available but rely on self-report and cannot be conducted among individuals who have committed suicide.

Confounder assessment

Individual- and community-level confounders were identified *a priori* based on the scientific literature and development of a directed acyclic graph (see appendix). We considered established risk factors for self-harm and factors that affect community violence or share common causes with community violence. Variables controlled in the final analysis depended on availability in death, discharge, and ACS records. Individual-level confounders included in analyses of fatal self-harm were marital status, education, foreign born, history of military service, and recent immigration to the United States. Analyses of nonfatal self-harm controlled for individual-level primary language spoken. Sensitivity analyses for nonfatal self-harm also controlled for health insurance type, a proxy for socioeconomic status, which was available in the ACS after 2007.

Community-level confounders in all analyses were annual or monthly community measures of sociodemographic composition, economic factors, social cohesion, firearm access, population mental health status, primary care provider density, alcohol outlet density, and weather (see appendix for details).

Parameters

We estimated three risk difference parameters that capture how the population risk of self-harm is associated with specific changes in the distribution of community violence. Accurate estimation of these parameters relies on positivity, meaning that individuals in all confounder subgroups have to be observed under the different exposure conditions for which estimates are made. Positivity is a particular concern in studies of community violence, because individuals with certain covariate combinations may only be present in either high-violence or low-violence communities.

To ensure that the risk difference parameters did not rely on extrapolation, we identified the highest and lowest monthly violence rates within each community between 2005 and 2013, and, for each individual, we only estimated the risk difference for reductions/increases in community violence to the minimum/maximum observed in their community. By restricting the predictions to violence levels actually observed within communities, we minimized bias from extrapolating predictions beyond what is supported by the data. Specifically, we estimated:

(1) $RD_{overall}$: the overall population risk difference comparing the estimated risk of self-harm if all individuals were exposed to 12-month average violence rates equal to the *highest* versus the *lowest* monthly violence rate observed within their communities[51]

(2) RD_{PA}: the population attributable risk difference comparing the *observed* risk of self-harm to the risk of self-harm if all individuals were exposed to 12-month average violence rates equal to the *lowest* monthly violence rate observed within their communities

(3) $RD_{targeted}$: the population risk difference comparing the *observed* risk of self-harm to the risk of self-harm if individuals in the *top quartile* of community violence (i.e. individuals living in high-violence communities) were exposed to 12-month average violence rates equal to the *lowest* monthly violence rate observed within their communities and exposure for all other individuals were left unchanged[52]

The last parameter corresponds to the expected change in the population-level risk of self-harm under a hypothetical violence-prevention intervention that targets the most violent communities and reduces violence substantially but within the range previously experienced. This parameter is also an example of a dynamic treatment regime[53,54] in that the change in exposure is based on its observed level at baseline, so the hypothetical intervention is tailored to those we expect to benefit most.

Statistical analysis

To estimate these marginal parameters, we used g-computation,[51] which allows estimation of additive scale parameters and summarizes the association between community violence and self-harm for the population overall, rather than within covariate sub-groups, as in typical regression. We used generalized additive models with a logit link to model the risk of self-harm as a function of community violence, frequency matching factors (year, 5-year age group, race/ethnicity, and gender), and the confounders.[55] We used cubic smoothing splines[55] for all continuous independent variables, including community violence, to capture potential non-linear relationships with self-harm risk. We then used the fitted model to predict the risk of self-harm for each individual under the different exposure scenarios and took the difference of the average estimated risks for the relevant contrasts to estimate the three RD parameters. All analyses were weighted to be population-representative by assigning weights equal to the risk of self-harm within each matching strata (q_0) for cases and weights equal to $(1- q_0)/J$ to controls, where J is the ratio of controls to cases.[56] We estimated 95% confidence intervals (CIs) using the nonparametric bootstrap.[51] We also confirmed that the observed risks aligned with the modeled estimated risks in the absence of modifications to the exposure distribution.

All analyses were stratified by self-harm type (fatal versus nonfatal) because the distribution and relative impacts of different determinants of self-harm vary by type.[1,37] We report results for overall associations and for analyses stratified by 5-year age group and gender, because age and gender define the groups most commonly described as high-risk,[1,37] and we hypothesized that these groups would respond differently to community violence.

Case records with incomplete covariate data (2.8%) were excluded from analyses, resulting in a final sample of 27,027 self-harm fatalities, and 331,203 nonfatal self-harm injuries. Data analysis was conducted using R 3.2.1 (R Foundation for Statistical Computing, Vienna, Austria), and model fitting and prediction were done using the gam package. This study was approved by the State of California and University of California, Berkeley Committees for the Protection of Human Subjects.

Nonfatal cases include only suicide attempts and self-harm injuries that were sufficiently serious to result in an emergency department visit or hospitalization. To assess the sensitivity of results to the inclusion of less severe cases for whom care-seeking may be optional and dependent on factors potentially associated with community violence (e.g. health insurance), we tested analyses restricted only to those nonfatal cases requiring inpatient hospitalization.

To assess the potential role of confounding due to unmeasured factors, we conducted a quantitative bias analysis. Using the bias equations presented by VanderWeele and Arah,[57] we estimated the characteristics of an unmeasured confounder that would yield the observed association between community violence and nonfatal self-harm, if the true effect were null.

Results

Table 1 presents the risk of fatal and nonfatal self-harm overall and by age group, gender, and quartile of past-year violence in community of residence. The risk of self-harm varied substantially by age group, gender, and type of self-harm and was positively correlated with community violence. Observed 12-month average levels of community violence ranged between

6.9 and 126.6 per 100,000. The lowest within-community monthly violence rates ranged from 2.4 to 64.7 per 100,000; the highest ranged from 14.5 to 154.6 per 100,000 (see appendix Figure 2 for geographic distribution). The appendix presents the number of cases and controls by age and gender.

Table 2 presents the overall associations between community violence and self-harm, adjusted for observed confounders. There were no associations of community violence with fatal self-harm ($RD_{overall}$: 0.0 per 100,000 [CI: -2.0, 1.9]; RD_{PA}: 0.0 per 100,000 [CI: -1.1, 1.0]; $RD_{targeted}$: 0.2 per 100,000 [CI: -0.2, 0.6]). For nonfatal self-harm, the $RD_{overall}$ was 62.9 per 100,000 (CI: 61.9, 63.7), or approximately a 27% reduction in self-harm relative to the observed risk. The RD_{PA} was 30.1 per 100,000 (CI: 29.7, 30.6), or a 13% reduction. The $RD_{targeted}$ was 10.8 per 100,000 (CI: 10.6, 11.0), or a 5% reduction. The median difference in community violence for affected communities for the $RD_{overall}$, RD_{PA}, and $RD_{targeted}$ were 21.2, 9.9, and 14.8, per 100,000, respectively.

Overall associations masked substantial sub-group heterogeneity. Figure 1 presents the RD_{PA} by age and gender and shows that community violence was associated with increased risk of nonfatal self-harm predominantly among the young and middle-aged groups (ages 15-59), with the strongest relationships for women ages 15-24 and men ages 40-49. Community violence was generally not meaningfully associated with fatal self-harm. $RD_{overall}$ (appendix Figure 3) and $RD_{targeted}$ (Figure 2) estimates showed similar age and gender patterns to the RD_{PA}, but $RD_{overall}$ were larger in magnitude and $RD_{targeted}$ were smaller.

In sensitivity analyses (Table 2), restricting nonfatal self-harm to 2008-2013 to additionally control for health insurance type slightly attenuated the association of community violence with self-harm. Restriction to only inpatient cases reduced the overall risk of self-harm and showed similarly patterned but attenuated risk differences compared to the main analysis. The $RD_{overall}$, RD_{PA}, and $RD_{targeted}$ for inpatient self-harm corresponded to 9%, 4%, and 2% less self-harm, respectively.

Results of the bias analysis are presented in the appendix. Briefly, for the association of community violence with nonfatal self-harm to be spurious, there would have to be an unmeasured confounder that is at least 50 percentage points more prevalent in high versus low violence communities and that causes a 100 per 100,000 increase in the risk of nonfatal self-harm (a very large association relative to the observed risk of 240 per 100,000).

Discussion

To our knowledge, this is the first study to examine the relationship of community violence with self-harm in a general population. We found that higher past-year community violence was associated with increased risk of nonfatal self-harm but not fatal self-harm, and that a parameter corresponding to setting community violence to lower levels for the highest-violence communities shows associations indicating meaningful reductions in nonfatal self-harm at the population level. Findings suggest that previously reported associations between community violence and nonfatal self-harm among adolescents[30,31,33,34] extend statewide to the entire

California population. Further, we identified important heterogeneity by age and gender, with the strongest associations for women ages 15-24 and men ages 40-49.

As in all observational studies, there may be residual confounding in the observed associations between community violence and self-harm. Confounding control was limited by the covariates available in death, discharge, and ACS records. The quantitative bias analysis indicates that for the observed association to be spurious, there would have to be an unmeasured factor that very strongly affects both community violence and self-harm. Identifying such a factor is possible. For example, mental disorder strongly increases self-harm risk, and also makes one more likely to live in a high-violence community. Confounders of particular concern include the type, extent, and history of mental and substance use disorders, personality traits, early life adversity, and precipitating life circumstances such as the loss of a loved one. However, exposure to community violence may causally precede these (e.g. incite substance use; contribute to the loss of a loved one). If these factors are on the causal pathway, adjusting for them would be inappropriate. We controlled for a large set of confounders including demographic, socioeconomic, contextual, and health indicators. However, additional research using longitudinal designs, more detailed covariate data on participants, and mediation analyses would help to separate these influences.

The community violence-self-harm association may also be driven by the co-occurrence of self-directed and outward-directed violence among the same individuals. Indeed, studies suggest that perpetration of violence against others (i.e., participating in community violence) is linked with psychiatric disorder, aggression, and other traits predisposing to self-harm, and that violence and suicidality mutually affect one another.[29,32,58] We did not capture whether cases or controls were also direct contributors to community violence and therefore could not assess the co-occurrence of internally- and externally-directed violence. Further investigation is needed to disentangle these factors, particularly for non-adolescents for whom existing research is limited.

Our finding that community violence is associated with nonfatal self-harm but not fatal self-harm may indicate that nonfatal self-harm is more responsive to community violence. Community violence may induce psychological distress or other psychological and behavioral correlates sufficient to provoke expressions of self-harm, but insufficient to induce serious intent to kill oneself. Nonfatal self-harm can be a means of coping with distress,[59] whereas fatal self-harm may be a means of escaping distressing environments.[5] These are fundamentally different responses, and community violence may be more likely to prompt one than the other. Differences in the covariates controlled in the analyses of fatal versus nonfatal self-harm or differential effects of residual confounding may also explain the different associations observed for nonfatal and fatal self-harm.

Our finding that the strongest associations were for young women and middle-aged men may be due to differences in vulnerability to stressors. Theory and evidence suggest that young women may be particularly vulnerable to life stressors and depression that can lead to suicidal behavior.[60,61] There is less research on psychological vulnerability to stressors among middle-aged men, but this group is less likely to seek or receive needed mental health care.[62,63] Thus, untreated mental or substance use disorders or psychological distress precipitated by community violence may be more likely to lead to self-harm in this group. Other work has documented recent increases in suicide among non-Hispanic White middle-aged men and suggested that rising rates of long-term physical disability and mental and substance use disorders in addition to declining job prospects may contribute.[2] Given rising rates of community violence,[7] our study suggests that community violence may contribute or exacerbate the risk of self-harm in this group. We also found minimal associations between community violence and self-harm among some high-risk groups (e.g. fatal self-harm for older men). This may indicate that community violence is not a key contributor to risk in these groups, and other social environment and individual factors would be worth examining.

Unlike previous studies that operationalize community violence as a binary "all or nothing" contrast, we used a continuous measure and estimated the impacts of plausible changes in exposure in an effort to more accurately estimate population-level impacts and better inform public health decision-making. [52] Differences in the magnitudes of the $RD_{overall}$, RD_{PA}, and $RD_{targeted}$ reflect differences in the levels of community violence contrasted and the proportion of people affected. The $RD_{overall}$ intervenes on everyone maximally, the RD_{PA} intervenes on individuals exposed to higher-than-minimum violence to varying degrees, and the $RD_{targeted}$ intervenes most restrictively on only individuals in high-violence communities. The $RD_{targeted}$ may be particularly informative because it corresponds to a hypothetical intervention to reduce violence in the highest-risk communities to achievable levels observed within those communities at some point over the study period. Focused deterrence strategies such as the Cure Violence model[64,65] and mentoring programs for delinquent youth,[66] are examples of scientifically supported, locally-targeted programs to reduce community violence that would fit this hypothetical scenario and have successfully reduced community violence by levels similar to those in parameters estimated in this study.

Our data do not include suicide attempts or other self-harm not resulting in hospital visits or deaths. Thus, we may be underestimating the burden of self-harm associated with community violence. In addition, if cases of self-harm of the same severity are more or less likely to receive care in a hospital depending on whether they live in a more or less violent community, selection bias may result. This pattern might result from less health insurance coverage, lower social support for care-seeking, or less access to emergency medical services in high-violence communities. However, results from sensitivity analyses restricted to the most severe cases for whom receipt of hospital services is unlikely to be optional were consistent, albeit attenuated, with those in the main analysis. Our control of proxy measures of healthcare access and other community-level determinants of care-seeking also help to address this concern.

Several other limitations of this study must be noted. First, records on the cause of death and injury classification are imperfect. However, studies suggest the degree of misclassification is not substantial enough to alter major trends and patterns.[40,41] Second, we lacked long-term exposure data for cases and controls, and exposure misclassification may occur if study participants did not actually reside at the reported location for the 12 months prior to injury/survey. Third, we used distinct data sources to draw cases and controls, which may generate differences in the measurement of covariates or result in residual confounding. Finally, we used CPUMAs as a proxy for communities. Although these units are locally-recognized places of residence, they may not fully capture the social environments of persons in this study.

Overall, this study strengthens the evidence on the relationship between community violence and self-harm. We used complete, population-wide data that included all deaths and hospital visits due to self-harm in California over an eight-year period, which allowed us to compare rare outcomes among important subgroups for whom previous assessments have been limited. We estimated easily-interpretable population-level parameters that avoided extrapolation and made novel and efficient use of an existing population-representative survey to draw controls. This approach could serve as a model for future investigations seeking to reconstruct population exposure and outcome experiences to answer important public health questions using existing big data. This study suggests that lower levels of community violence, even when limited to the highest-violence communities, are associated with lower risk of nonfatal self-harm, particularly among young and middle-aged persons. Future research should strive for greater confounding control through study design or by measuring and controlling for more detailed covariate data, investigate reasons for differential associations by age and gender, and assess whether community violence prevention programs have meaningful impacts on self-harm.

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Tables and figures

Table 1: Risk of fatal and nonfatal self-harm by participant characteristics, California, 2005-2013

Characteristic		Fatal		Nonfatal	
		Female	Male	Female	Male
All		8.8	24.8	279.0	173.4
Quartile of past-year violence in community of residence	Lowest	7.4	21.4	255.5	139.9
	2	9.0	24.7	263.3	161.0
	3	9.2	25.5	282.3	180.8
	Highest	9.5	27.5	315.0	211.7
Age group (years)	15-19	3.3	8.9	493.6	237.2
	20-24	4.6	15.4	304.8	223.8
	25-29	4.7	15.7	237.1	196.9
	30-34	5.4	16.9	213.5	170.2
	35-39	7.3	19.2	216.2	152.4
	40-44	9.3	21.8	219.8	151.3
	45-49	10.5	27.7	211.3	145.4
	50-54	13.0	30.9	172.6	123.1
	55-59	11.6	32.3	121.4	97.0
	60-64	9.7	27.8	74.2	66.0
	65-69	7.8	25.3	53.1	45.3
	70-74	6.4	27.7	38.5	38.6
	75-79	7.4	39.1	36.1	39.5
	80-84	7.2	43.2	37.7	44.3

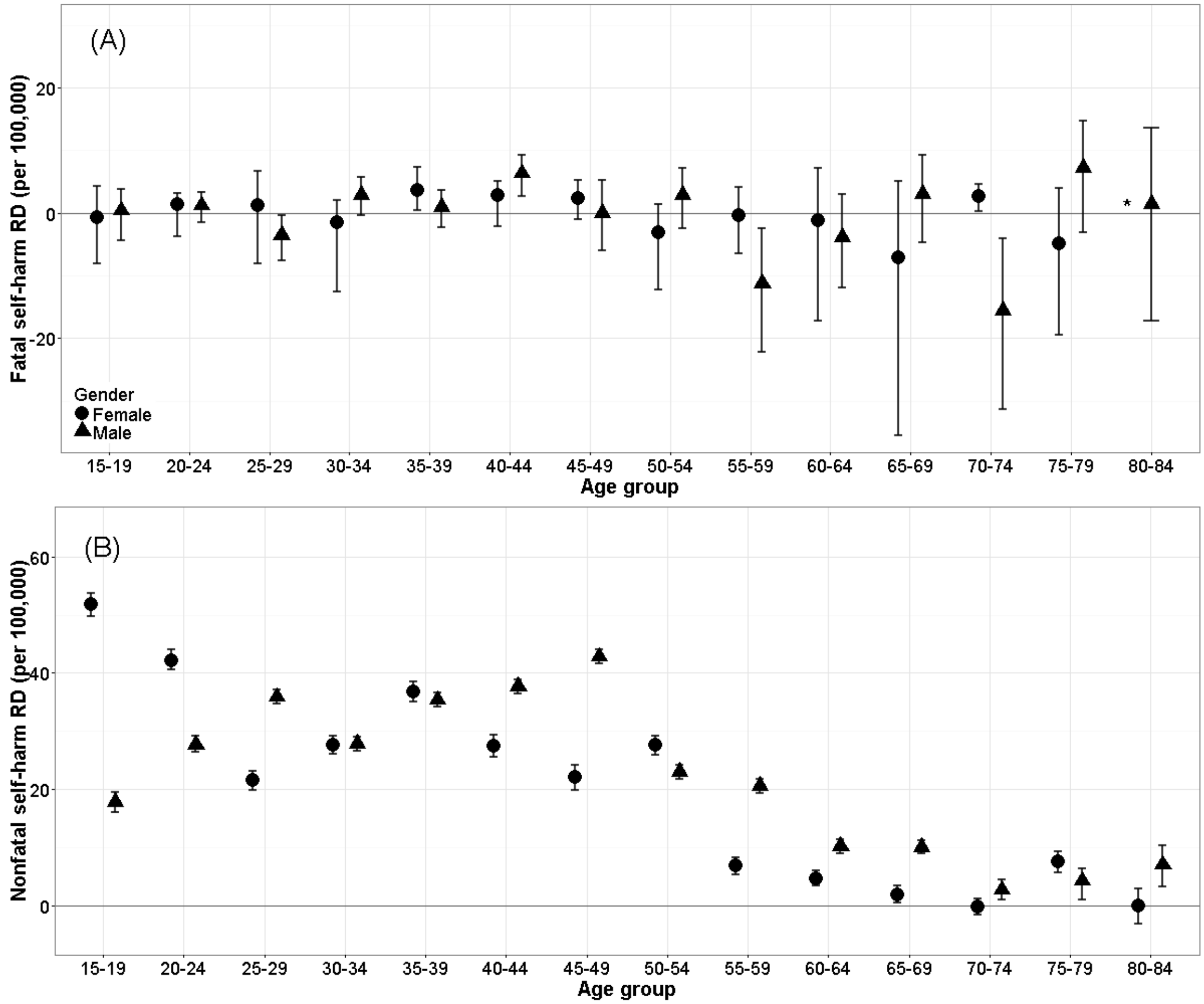
Legend: Estimates of risk of self-harm are weighted to be population representative (see Statistical Analysis). Risks are presented per 100,000 persons per year.

Table 2: Overall adjusted associations between exposure to community violence and risk of fatal and nonfatal self-harm, California, 2005-2013

Self-harm type	Observed risk	Risk of self-harm if all individuals were exposed to high community violence[a] (95% CI)	Risk of self-harm if all individuals were exposed to low community violence[a] (95% CI)	Overall risk difference (95% CI)	Population attributable risk difference (95% CI)	Targeted risk difference (95% CI)
Fatal	21.0	21.0 (19.9, 22.0)	21.1 (20.0, 22.1)	0.0 (-2.0, 1.9)	0.0 (-1.1, 1.0)	0.2 (-0.2, 0.6)
Nonfatal	234.8	267.7 (267.1, 268.2)	204.7 (204.3, 205.1)	62.9 (61.9, 63.7)	30.1 (29.7, 30.6)	10.8 (10.6, 11.0)
Nonfatal, restricted to 2008-2013, controlling for health insurance type	240.0	266.4 (265.5, 267.2)	214.2 (213.5, 214.9)	52.2 (50.7, 53.7)	25.8 (25.0, 26.5)	9.7 (9.4, 10.0)
Nonfatal, inpatient only	76.5	80.2 (80.0, 80.5)	73.6 (73.4, 73.8)	6.7 (6.2, 7.1)	2.9 (2.7, 3.1)	1.4 (1.3, 1.5)

[a] High and low violence are defined as the highest and lowest levels of monthly violence observed within the study participants' communities over the study period (2005-2013).
Risks and risk differences are presented per 100,000 persons per year. The overall risk difference compares the estimated risk of self-harm if all individuals were exposed to 12-month average violence rates equal to the highest versus the lowest monthly violence rate observed within their communities. The population attributable risk difference compares the observed risk of self-harm to the risk of self-harm if all individuals were exposed to 12-month average violence rates equal to the lowest monthly violence rate observed within their communities. The targeted risk difference compares the observed risk of self-harm to the risk of self-harm if individuals in the top quartile of community violence were exposed to 12-month average violence rates equal to the lowest monthly violence rate observed within their communities and exposure for all other individuals were left unchanged. Analyses are adjusted for 5-year age group, gender, race/ethnicity, year of injury or survey, and community-level confounders (see Covariate assessment). Analyses of nonfatal outcomes are also adjusted for individual-level primary language spoken, or primary language spoken and insurance type, as indicated. Analyses of fatal outcomes are also adjusted for individual-level marital status, education, foreign born, military service, and recent immigration to the United States. CI: confidence interval.

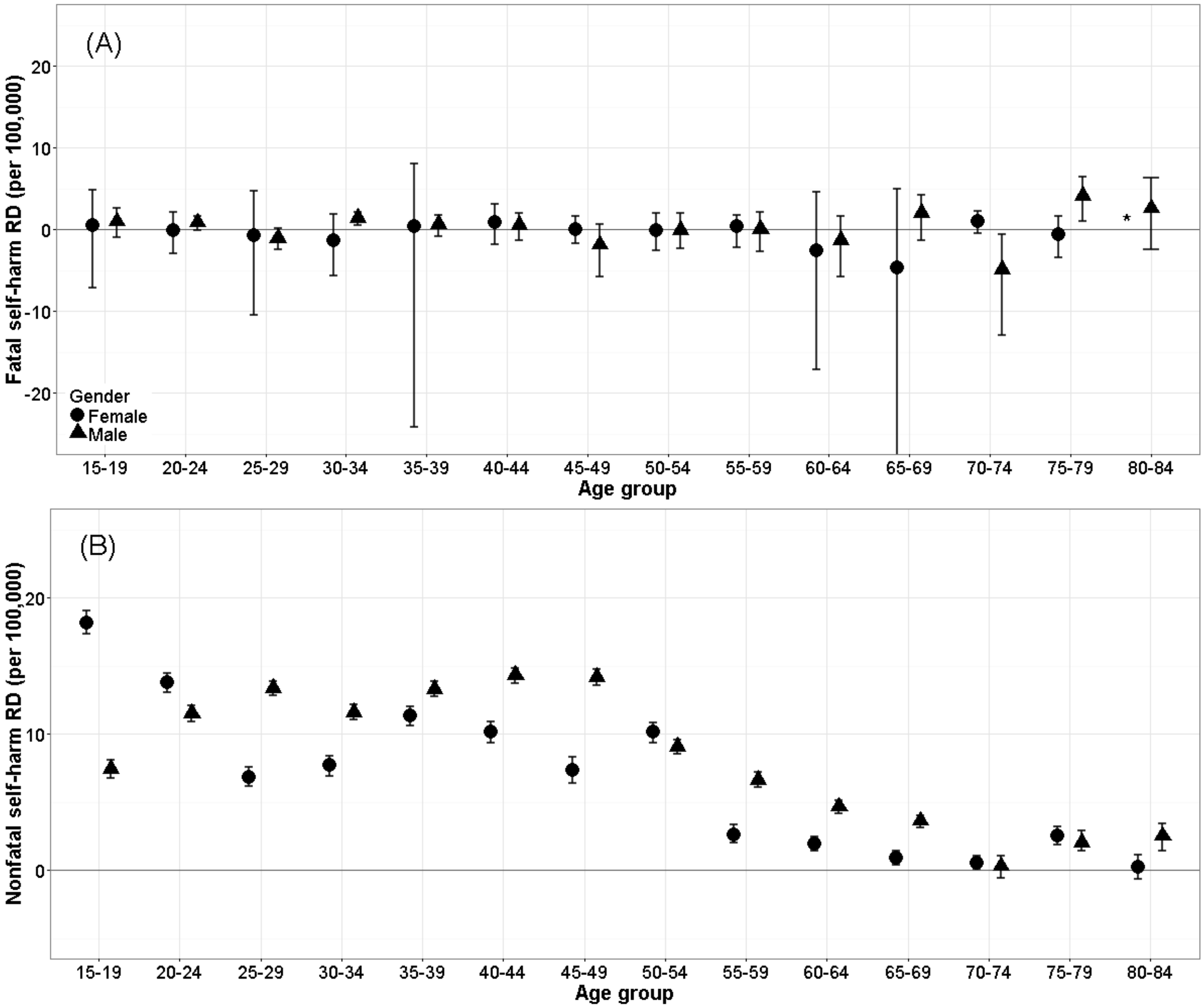
Figure 1: Adjusted population attributable risk difference for fatal and nonfatal self-harm associated with community violence, by age and gender, California, 2005-2013



Risk differences are presented per 100,000 persons per year. (A) Fatal self-harm. (B) Nonfatal self-harm. The population attributable risk difference compares the observed risk of self-harm to the risk of self-harm if all individuals were exposed to 12-month average violence rates equal to the lowest monthly violence rate observed within their communities. Analyses are adjusted for race/ethnicity, year of injury or survey, and community-level confounders (see Covariate assessment). Analyses of nonfatal outcomes are also adjusted for individual-level primary language spoken. Analyses of fatal outcomes are also adjusted for individual-level marital status, education, foreign born, military service, and recent immigration to the United States. Confidence intervals for fatal self-harm are asymmetric due to the small sample size of these subgroups and should be interpreted with caution. RD: risk difference. Bars indicate 95% confidence intervals.

[a] Estimate for women ages 80-84 is unstable due to small sample size and is not presented.

Figure 2: Adjusted targeted risk difference for fatal and nonfatal self-harm associated with community violence, by age and gender, California, 2005-2013



Risk differences are presented per 100,000 persons per year. (A) Fatal self-harm. (B) Nonfatal self-harm. The targeted risk difference compares the observed risk of self-harm to the risk of self-harm if individuals in the top quartile of community violence were exposed to 12-month average violence rates equal to the lowest monthly violence rate observed within their communities and exposure for all other individuals were left unchanged. Analyses are adjusted for race/ethnicity, year of injury or survey, and community-level confounders (see Covariate assessment). Analyses of nonfatal outcomes are also adjusted for individual-level primary language spoken. Analyses of fatal outcomes are also adjusted for individual-level marital status, education, foreign born, military service, and recent immigration to the United States. Confidence intervals for fatal self-harm are asymmetric due to the small sample size of these subgroups and should be interpreted with caution. RD: risk difference. Bars indicate 95% confidence intervals.

[a] Estimate for women ages 80-84 is unstable due to small sample size and is not presented.

CHAPTER 4: ACUTE WITHIN-COMMUNITY VARIATION IN VIOLENCE AND RISK OF SELF-HARM IN CALIFORNIA: A POPULATION-BASED CASE-CONTROL AND CASE-CROSSOVER STUDY

Introduction

Self-harm is a leading cause of morbidity and mortality in the United States (US), accounting for over 44,000 deaths and 500,000 injuries in 2015.[1] Rates of self-harm are also increasing,[1] in some cases rapidly,[2] but the reasons for these increases are not well-understood. The influence of social environments on self-harm has been recognized for over a century,[3] but research to disentangle which features of the social environment are most influential is limited and may help shed light on the drivers of self-harm.

Community violence—i.e. experiencing, witnessing, or hearing about violence in one's community— is one potentially modifiable feature of the social environment that may influence self-harm. However, few studies have examined the association of community violence with self-harm,[4–11] and to our knowledge, no research has examined acute within-community variation in violence, as opposed to variation between communities in chronic violence.

Acute within-community variation in violence is directly relevant to common models of self-harm. The stress-diathesis model and its variants posit that incidents of self-harm are the confluence of a long-term predisposition to self-harm (e.g., due to early life adversity) with acute, stressful life events (e.g., loss of a loved one or psychosocial crisis) that trigger brief periods of elevated risk.[12] Acute increases in community violence – for example, having neighbors who were victims of a recent shooting—may trigger self-harm in a vulnerable individual.

Levels of community violence vary over time. Some of this variation follows predictable patterns—for example, community violence tends to be higher in summer months and lower in winter months, patterns that may reflect weather, employment, or school opening/closing cycles.[13] However some of this variation is unpredictable. For example, although July tends to be a high-violence month, within a community, some Julys will have higher or lower violence than others. This study leverages this variation in violence within communities to study the association of community violence with risk of self-harm.

Studying these acute, within-community changes in violence addresses several methodological challenges that have inhibited past research on the association of community violence with self-harm.[4–11] Community violence is strongly associated with other self-harm risk factors such as economic opportunity, making the effects of these factors difficult or impossible to disentangle, a phenomenon known as structural confounding.[14] Investigating acute, within-community changes in violence helps address this challenge by allowing comparison of residents of the same community at times with relatively high and low levels of community violence, thereby controlling for community-level factors that are time-invariant over the study period. Additionally, individual risk factors such as mental and substance use disorders are strong determinants of violence direct towards both self and others in the community, and thus are potential confounders of the relationship between community violence and self-harm. Because self-harm is rare, studies of self-harm are frequently retrospective and lack detailed data to adequately control individual-level confounding. Examining acute, within-community changes in violence enables the use of designs such as the case-crossover, to compare each individual's exposures at different times while controlling for individual risk factors that are time-invariant over the study period. Finally, community violence and self-harm both have long-term trends and seasonal patterning, peaking in summer and plunging in winter. Research that does not account for this patterning may detect associations that are simply artefacts of this temporal patterning. Analyzing acute, within-community changes in violence allows this temporal patterning to be explicitly modeled and removed to isolate the associations of interest.

We examined whether acute variation in community violence is associated with risk of fatal and nonfatal self-harm. To maximize control of individual and community confounders through study design, we utilize both case-control and case-crossover approaches (detailed below) with community-matched controls drawn in close time proximity to cases. We leverage data from statewide population-based registries, surveys, and healthcare utilization data from California, a large and heterogeneous state with self-harm trends similar to those seen nationwide.

Methods

Overall study designs and data sources

We applied case-control and case-crossover study designs to existing population-based data sources. In case-control studies with a primary study base,[15] all cases arising from a defined population are identified, and controls representative of the defined population (i.e., the study base) are sampled (e.g. using population-based sampling frames or random digit dialing).[16] The case-crossover design[17] compares the cases' exposure at a time relevant to case occurrence to exposure at referent non-case times, thereby catering to brief exposures and transient changes in risk for acute-onset outcomes. The case-crossover enhances control of unmeasured individual confounders, and reduces concerns related to control-selection bias. The case-control design provides a useful comparator and, depending on the design, eliminates the need for certain assumptions required by the case-crossover (see "selection of control time periods" below).

We compiled data on self-harm and community violence for 2005-2013 from mortality, emergency department, and inpatient hospitalization discharge records from the California Offices of Vital Records and Statewide Health Planning and Development. Records included all deaths and hospital visits statewide, excluding active duty military hospitals, and captured medical information, demographic characteristics, and decedent address (in vital records) or patient zip code of residence (in hospital records). External cause of injury coding in California's hospital discharge records is compulsory, entails ongoing quality assurance efforts, and considered 100% complete.[18] In mortality records, external cause of mortality codes for homicide and self-harm are also considered valid and complete [19]. Emergency department records were available starting in 2005.

Cases were all deaths and hospital visits due to deliberate self-harm (any external cause of injury code: hospital visits, ICD-9: E95; deaths, ICD-10: X6-X8). Controls were the cases themselves at control time periods (case-crossover), or California resident participants of the American Community Survey (case-control). The ACS is a continuous, national survey conducted by the US Census Bureau. It produces population-representative small-area estimates of demographic, economic, and social indicators, and serves as an efficient, existing, population-representative sampling frame from which to draw population-based controls. From 2005 to 2013, between 170,000 and 220,000 California residents participated in the ACS annually.

In the ACS case-control design, consistent with previous research,[11] we created a representative pseudo-population of California residents by duplicating each ACS record by the corresponding person weight[20] and selected controls from this expanded ACS dataset. For statistical efficiency,[21] ACS controls were matched to cases on confounders that are strongly associated with self-harm: gender, race/ethnicity, 5-year age group, community, and neighboring time unit (see "selection of control time periods" below). For this design, we did not assess self-harm status of ACS controls and thus assume that controls were not also cases at the time they were selected as controls. This is reasonable, because self-harm was very rare (<0.5% in all strata).

Exposure assessment

Community violence was assessed using deaths due to homicide (ICD-10 death codes X85-X99, Y00-Y09, Y35, U01, U02, Y871) and hospital visits due to assault (ICD-9 hospital visit codes E960-E969, E970-E977) in the Consistent Public Use Microdata Area (CPUMA) of residence. CPUMAs are geographic partitions designated by the US Census Bureau that include at least 100,000 residents. The 110 CPUMAs in California are consistently defined over the study period, and correspond to known neighborhoods in urban areas (95% of the California population), and counties or aggregations of small counties in rural areas.

The CPUMA of residence was determined from the geocoded decedent address (mortality records) or the zip code of residence via geographic crosswalk (hospital records).[22] We selected CPUMAs to define communities because they are recognized places of residence, but are large enough for stable estimation of community violence rates. CPUMAs were found to be meaningful geographic units in previous research on community violence and self-harm [11], and are the smallest geographic identifier available in the ACS. Crime data can also be used to measure community violence, but may contain patterns that are artefacts of differences in reporting practices between jurisdictions and over time.[23,24] Objectively measured community violence is strongly correlated with frequency of experiences of direct injury and witnessing violence reported by residents,[25,26] but avoids same-source bias, in which error in self-report of both community violence and nonfatal self-harm may be associated, for example due to respondent temperament (fatal cases cannot self-report their past exposure).

To our knowledge, there is no evidence on the critical exposure period (duration and lag time) for the association of acute community violence with self-harm. Related literature on stressful life events and self-harm varies in the time frames assessed; self-harm has been associated with stressors occurring within a few hours and as much as several weeks.[27] We hypothesized that any effects of acute violence would be immediate and of short duration. Thus, we selected a reasonable time frame of 30 days prior to injury/survey to balance capturing short-term, acute effects with pooling enough data to estimate stable rates of community violence. We used ACS-based population estimates as denominators to calculate community violence rates.

To separate acute variation in community violence from predictable temporal patterning, we de-trended the community violence rates by applying a Kalman smoother with seasonal terms.[28] The

Kalman smoother is an automated, Bayesian procedure that uses an ARIMA model as its first stage. To ensure temporal ordering, we applied the smoother to the unique time series of 30-day units spanning 2005 to 2013 and defined by the community and index day of the case. For example, for a case occurring on April 20 2007 in a given community, we constructed a time series of community violence rates in 30-day time units in the set {…, February 20 2007 – March 21 2007, March 22 2007 – April 20 2007, April 21 2007 – May 20 2007, …}, and applied the smoother to this series. We defined acute community violence, or deviations from expected levels, as the difference between the observed rate and the modeled rate of community violence (i.e. the residuals of these models). Previous simulation work suggests that the Kalman smoother is superior to a range of other time series methods in the separation of acute versus predictable patterning of violence in California populations.[13] Violence residuals created using ARIMA models were highly correlated (Pearson's correlation: 0.95). Figure 1 depicts an example community violence trend and residuals after applying the Kalman smoother.

Confounder assessment

To examine acute, within-community variation in violence exposure, we drew controls from the same community as the cases. Thus, community-level confounders that are time-invariant over the study period are controlled by design. The case-crossover design provides additional strength by also controlling time-invariant individual-level confounders. Remaining potential confounders are time-varying community and individual factors for both designs and time invariant individual factors in the case-control design. These were identified a priori based on scientifically established risk factors for self-harm and factors that affect community violence or its determinants. Individual-level variables controlled in the final case-control analysis depended on availability in death, discharge, and ACS records. For fatal self-harm, these were marital status, education, foreign born, veteran status, and recent immigration to the US. For nonfatal self-harm, we used primary language spoken. Community-level confounders controlled in all analyses were annual or monthly measures of sociodemographic composition, economic factors, firearm access, social organization density, primary care provider density, alcohol outlet density, and weather, among others (see appendix for details).

Selection of control time periods

Each control was drawn from the same community as the corresponding case. As a result, controls could not be matched to cases on time of injury/survey, but were matched with a lag or lead. We selected control periods carefully, because although we removed temporal patterning in community violence, we wanted to minimize the possibility that any residual patterns could lead to spurious associations.[29]

For both fatal and nonfatal outcomes, we drew controls from exactly 30 days after the case occurrence (a 30-day lead with respect to the case), and considered controls with a 30-day lag or bidirectional design as sensitivity analyses as sensitivity analyses (Figure 2). The 30-day lead limits confounding by seasonal patterns, secular trends, and other events affecting both exposure and outcome by being as close in time as possible (in 30-day units) to the case. Referent periods before the case are similarly close in time, but require the assumption that the control's exposure does not carryover beyond 30 days to affect the case. Although we hypothesized that any effects of acute changes in community violence on self-harm would be short-term, longer-lasting effects are possible. In contrast, exposure after the case occurrence cannot influence the occurrence of the case. Relative to control time periods with longer lags or leads, the 30-day lead also provides better control of individual and community confounders that may change systematically over time, and for the case-crossover design, minimizes the risk of exposure misclassification due to residential moves.

In using a 30-day lead as the primary referent period, we must assume that past outcomes do not affect future exposure. This is a reasonable assumption in this study because self-harm is uncommon and rarely publicized, and the exposure is characterized as unpredictable patterning in community violence. Also of note, using controls drawn from after case occurrence is a violation of the Study Base Principle[30] for fatal outcomes in the case crossover design, because the case has died in the index time and thus is not eligible to become a case 30 days later. This concern, along with avoiding the assumption that the control's exposure does not carryover beyond 30 days to affect the case, motivated the inclusion of the case control design with population-based ACS participants as controls. Nevertheless, for acute exposures with transient effects, post-case exposure may be a reasonable proxy for the exposure experience during a referent period not relevant to case occurrence, and excluding post-outcome referent periods may result in an even greater selection bias.[31–33] Thus, we drew controls with a 30-day lead for the main analysis and considered controls with a 30-day lag or bidirectional design as sensitivity analyses. We tested these approaches because controls drawn with a 30-day lag have the benefit of not relying on dead controls, and previous simulation studies suggest that the bidirectional design may be superior in controlling for trends and seasonality.[31–34] We did not consider controls drawn from all or a random selection of control periods (e.g. all 30-day units 2005-2013 except that immediately prior to case occurrence), because these approaches performed poorly in previous simulations.[31–33]

Statistical analysis

We used conditional logistic regression to estimate the conditional odds ratio associated with a community violence residual of 1 per 100,000 versus 0 (approximately the 80^{th} percentile versus the median, expected level of community violence), while accounting for the matched data structure. Continuous variables (exposure, covariates) were entered linearly. To allow for potential non-linearity, we considered quadratic and cubic terms that improved model fit, a priori optimizing the Akaike Information Criterion (AIC). The intraclass correlation coefficients for the occurrence of fatal and nonfatal self-harm across CPUMAs[35] were negligible (<0.001), indicating that no further model adjustment to account for clustering of participant outcomes within communities was necessary (i.e., we assume that outcomes within a community are independent given community).

Cases were restricted to those occurring between March 2, 2005 and December 1, 2013, such that controls could be drawn as early as January 31, 2005 and from as late as December 31, 2013, and exposure could be assessed for the 30 days before these dates. Cases with incomplete individual-level covariate data (2.8%) were excluded from case-control analyses. We restricted the study to California residents (those for whom we had community violence data) and to those aged 15 to 84 at the time of injury because there were few cases of self-harm outside of that range. We stratified analyses by self-harm type (fatal versus nonfatal) because determinants of self-harm differ by type.[1,36,37]

We conducted analyses using R.[38] Exposures modeled with multiple terms (e.g. quadratic or cubic) were combined into a single summary measure of association using the “glht” function in the “multcomp” package. This study was approved by the State of California and University of California, Berkeley Committees for the Protection of Human Subjects.

Sensitivity analyses

Our approach to studying acute changes in community violence assumes that the exposure is brief and transient, and uncorrelated across periods.[32] We thus assessed the autocorrelation function (ACF) of each exposure Kalman smoothed violence residual series (i.e. the acute deviations from expected levels of community violence). Remaining autocorrelation was rare, but to ensure those places were not driving the results we conducted a sensitivity analysis restricting to CPUMAs with an absolute value of the ACF<0.2 at one lag.[39]

Results

There were 30,741 cases of fatal self-harm and 362,508 cases of nonfatal self-harm among adults aged 15 to 84 in California between March 2, 2005 and December 1, 2013, corresponding to crude annual rates of 12.2 per 100,000 and 144.2 per 100,000, respectively. Table 1 presents characteristics of study participants. Fatal cases were 23% female, 69% white non-Hispanic, and 16% Hispanic, with a median age of 48. Nonfatal cases were 59% female, 59% white non-Hispanic, and 25% Hispanic, with a median age of 31. Acute deviations from expected levels of community violence were centered around the expected value of 0 with an interquartile range of approximately -0.90 to 0.90 per 100,000, but varied across study subjects within communities; acute deviations were positively associated with self-harm case occurrence (median difference in acute community violence of 0.008 for fatal and 0.011 for nonfatal).

Figure 3 presents results for the association of acute community violence with fatal and nonfatal self-harm, adjusted for individual and community confounders, for the main and sensitivity analyses. For fatal self-harm, 30-day periods with higher-than-expected levels of community violence were associated with an OR of 1.012 (95% confidence interval [CI]: 1.003, 1.021) in the case-crossover design and 1.000 (CI: 0.985, 1.015) in the case-control design. For nonfatal self-harm, these estimates were 1.007 (CI: 1.004, 1.009) for case-crossover design and 1.006 (CI: 1.004, 1.009) for case-control. Associations for sensitivity analyses with controls drawn with both a 30-day lag and lead were generally attenuated. Analyses with controls drawn with a 30-day lag showed null or slightly protective associations.

Assessment of autocorrelation in the violence residuals after applying the Kalman smoother suggested that this approach successfully removed autocorrelation, secular trends, and seasonal patterning from most series (see appendix for details). There were 11 CPUMAs (10%) in which the absolute value of the autocorrelation at one lag was greater than 0.2. Exclusion of these CPUMAs did not alter the results.

Discussion

We assessed whether acute variation in community violence was associated with risk of fatal and nonfatal self-harm using comprehensive population-based data from California and several population-based case-control and case-crossover approaches. Results from this investigation varied by study design, each with differing assumptions, strengths, and weaknesses in control of confounding by common temporal patterning and individual- and community-level factors, and potential for control-selection bias. We preferred the case-crossover design with controls drawn from 30 days after each case. Compared to the others we considered, this design provides better control of measured and unmeasured individual-level confounders, minimizes concerns about confounders, seasons, and places of residence changing over time, and does not require assuming that the control exposure does not have carryover effects beyond 30 days. Results from this design suggested that higher-than-expected levels of 30-day community violence are associated with an increased risk of self-harm, particularly nonfatal self-harm, but that the level of increased risk is small (approximately 1%).

There are several reasons why the associations detected in this study may be small. First, we only assessed acute deviations from expected levels of community violence. It therefore does not capture the entire relationship between community violence and self-harm. Overall levels and regular patterns of community violence may be stronger determinants of self-harm than unexpected changes. Indeed, results presented in Chapter 3 of this dissertation suggest that the association of overall levels of community violence, at least with nonfatal self-harm, is strong. Sudden or unexpected increases in community violence may be sufficient to influence anxiety or substance use,[40] which can contribute to self-harm, but not sufficient to influence self-harm resulting in hospital visits or death. Larger increases in community violence (e.g. mass shootings, wars, or terrorism) may also be more likely to produce bigger effects and have been associated with self-harm in previous research (for example[41]).

Second, this study improves on previous research by limiting same-source bias, structural confounding, and confounding due to time-invariant individual confounders such as genetics and family history. This greater control may also explain the smaller-than-expected associations.

Third, the exposure measure in this study–30-day deviation from expected levels of community violence in the CPUMA of residence–may not be the optimal characterization. Previous research has identified strong associations between long-term CPUMA-level community violence and self-harm,[11] and 30-day time units are long enough to estimate stable rates and allow for variability in acute exposure but small enough to minimize the likelihood that other factors are changing over the time period. However, the most salient geographic scope and time frame for elevated risk remain uncertain. This is an area for future research. More certainty about the relevant time of increased risk would also help in interpreting differences in results between the main analysis and sensitivity analyses. In particular, findings for the sensitivity analysis with controls drawn with a 30-day lag showed null or slightly protective effects. These findings may be the result of associations between acute community violence and self-harm last longer than one 30-day time unit (i.e. the control's exposure influences the case occurrence), but more investigation of critical risk periods is needed.

Fourth, self-harm is a heterogeneous condition with different drivers and manifestation in different groups.[1,36,37] It is possible that acute community violence is associated with self-harm for some groups but not others; these patterns may not be detectable at the population level. This is an area for future work that we are actively pursuing.

Fifth, as with all observational studies, there may be residual confounding or selection bias. Any causes of both community violence and self-harm that change unpredictably in time within communities are uncontrolled. Given the small magnitude of the measured associations and that not all study designs showed harmful associations, these biases very well may explain the findings. However, through measured covariates and the study design, we controlled for a substantial range of individual- and community-level confounders. Future research examining the impacts of violence prevention programs aiming to limit acute increases in community violence (e.g. the District of Columbia's Summer Crime Prevention Initiative[42] or summer employment programs[43]) may provide more conclusive evidence.

This study has several other limitations. First, all of the approaches we considered assume that individuals in our study have independent, non-overlapping exposure histories. Although not strictly true in our study, previous research suggests the impacts of violation of this assumption in the presence of a rare disease are small, and alternatives are likely to cause greater selection bias [33,32]. Second, our measure of community violence does not capture violence not resulting in hospital visits or deaths, and thus may be underestimating the overall level of community violence.

Third, neither the case-control nor the case-crossover design control factors that are specific to individuals or communities and vary acutely in time, such as individual distress or hopelessness, regional closing of a major employment center, or other compositional or structural changes of communities. These risk factors can bias the measured association if they if they affect both risk of self-harm and unexpected, short-term variation in community violence. For individual-level factors, this is plausible, because fluctuations in mental state could relate to fluctuations in both self-harm and violence within a given 30-day period. For example, an individual's mental state might lead them to participate in both community violence and self-harm or make them more vulnerable to both violent victimization and self-harm. We minimized the impact of these factors by removing predictable patterning in community violence in the exposure characterization and by drawing controls as close as time as possible to the cases, but, as with all observational studies, residual confounding is possible.

Fourth, it is possible that the detected associations are artefacts of "harvesting".[44] Harvesting might occur if an increase in community violence led the most vulnerable individuals to commit fatal self-harm and thus be removed from the risk pool, such that self-harm in subsequent time periods would be abnormally infrequent, even if community violence continued to be elevated. This pattern could cause bias in either direction. Fifth, death, discharge, and ACS records did not link unique individuals over time. Thus, it is possible that individuals were both cases and controls, or appeared as cases of nonfatal self-harm multiple times. This non-independence was not taken into account in the analysis, but its impacts are likely to be small. However, the outcomes were extremely rare, so any bias in variance estimation is likely to be small.

Despite these limitations, our study improves on prior work in this field and the methodology provides a template for future research. We used large, existing databases from California to study social ecological drivers of self-harm, an outcome for which previous research has been limited by small sample sizes. Recent increases in the size, scope, and availability of large health data facilitate epidemiologic studies that combine different data sources in efficient ways and leverage the high degree of geographic and temporal precision available in these data. This study is one application in which such data are particularly useful–the case of population-based case-control studies with transient ecological exposures. Our findings suggest that acute, within-community variation in violence has a small association with self-harm. Future research should further assess critical time periods of increased risk of self-harm and the impacts of violence prevention programs and policies.

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Tables and figures

Figure 1: Example community violence trend and Kalman smoother residuals

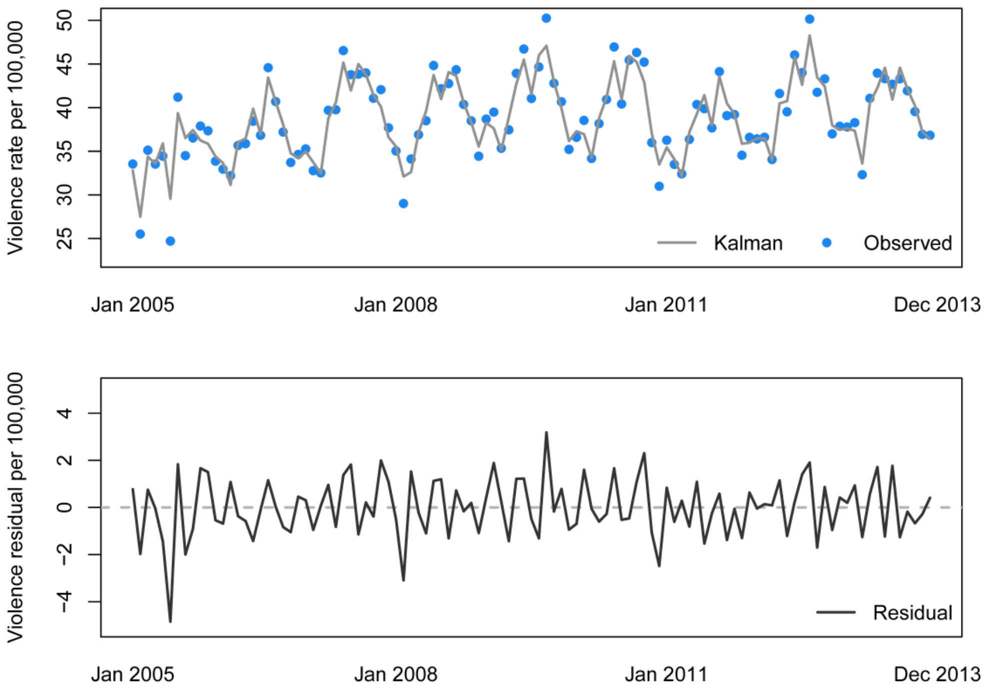


Table 1: Study participant characteristics, California, 2005-2013

<table>
<tr><th rowspan="2">Characteristic</th><th rowspan="2">Design</th><th colspan="2">Fatal</th><th colspan="2">Nonfatal</th></tr>
<tr><th>Cases</th><th>Controls</th><th>Cases</th><th>Controls</th></tr>
<tr><td rowspan="2">Sample size</td><td>Case-control</td><td>28,933</td><td>28,933</td><td>353,132</td><td>353,132</td></tr>
<tr><td>Case-crossover</td><td>30,741</td><td>30,741</td><td>362,508</td><td>362,508</td></tr>
<tr><td rowspan="2">Acute deviations from expected levels of community violence* (median [IQR])</td><td>Case-control</td><td>-0.01 (-0.91, 0.91)</td><td>-0.03 (-0.94, 0.89)</td><td>0.00 (-0.92, 0.93)</td><td>-0.02 (-0.94, 0.92)</td></tr>
<tr><td>Case-crossover</td><td>0.00 (-0.91, 0.92)</td><td>-0.03 (-0.95, 0.90)</td><td>0.00 (-0.92, 0.94)</td><td>-0.01 (-0.94, 0.93)</td></tr>
<tr><th colspan="2">Characteristics of case-crossover cases</th><th colspan="2">Fatal</th><th colspan="2">Nonfatal</th></tr>
<tr><td colspan="2">Age (median [IQR])</td><td colspan="2">48 (34, 59)</td><td colspan="2">31 (21, 45)</td></tr>
<tr><td colspan="2">Gender (% female)</td><td colspan="2">23.3%</td><td colspan="2">58.5%</td></tr>
<tr><td colspan="2">Race/ethnicity (%):</td><td colspan="2"></td><td colspan="2"></td></tr>
<tr><td colspan="2">White, NH</td><td colspan="2">69.4%</td><td colspan="2">58.6%</td></tr>
<tr><td colspan="2">Black, NH</td><td colspan="2">4.0%</td><td colspan="2">8.2%</td></tr>
<tr><td colspan="2">American Indian, NH</td><td colspan="2">0.5%</td><td colspan="2">0.4%</td></tr>
<tr><td colspan="2">Asian or Pacific Islander, NH</td><td colspan="2">8.3%</td><td colspan="2">4.4%</td></tr>
<tr><td colspan="2">Other/multi-race, NH</td><td colspan="2">1.4%</td><td colspan="2">3.4%</td></tr>
<tr><td colspan="2">Hispanic</td><td colspan="2">16.3%</td><td colspan="2">24.8%</td></tr>
</table>

IQR: interquartile range. NH: non-Hispanic.
Sample sizes vary slightly for case-control versus case-crossover due to missingness in individual-level covariates which are used in the case-control but not case-crossover design. We present characteristics of cases for the case-crossover study only, because this group is more inclusive and descriptive statistics are nearly identical for the case-control study.
*Acute community violence was defined as the difference between the observed rate and the Kalman smoothed rate of community violence.

Figure 2: Case and control exposure periods for case-crossover and case-control designs

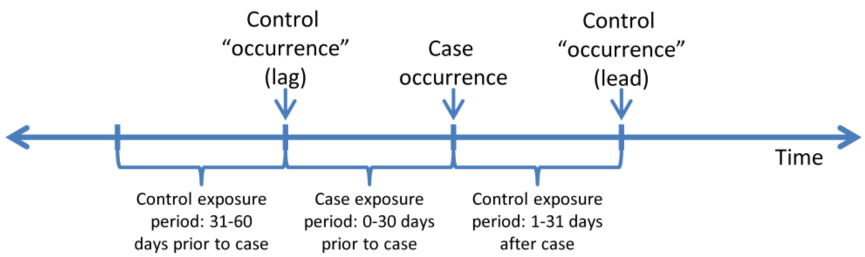
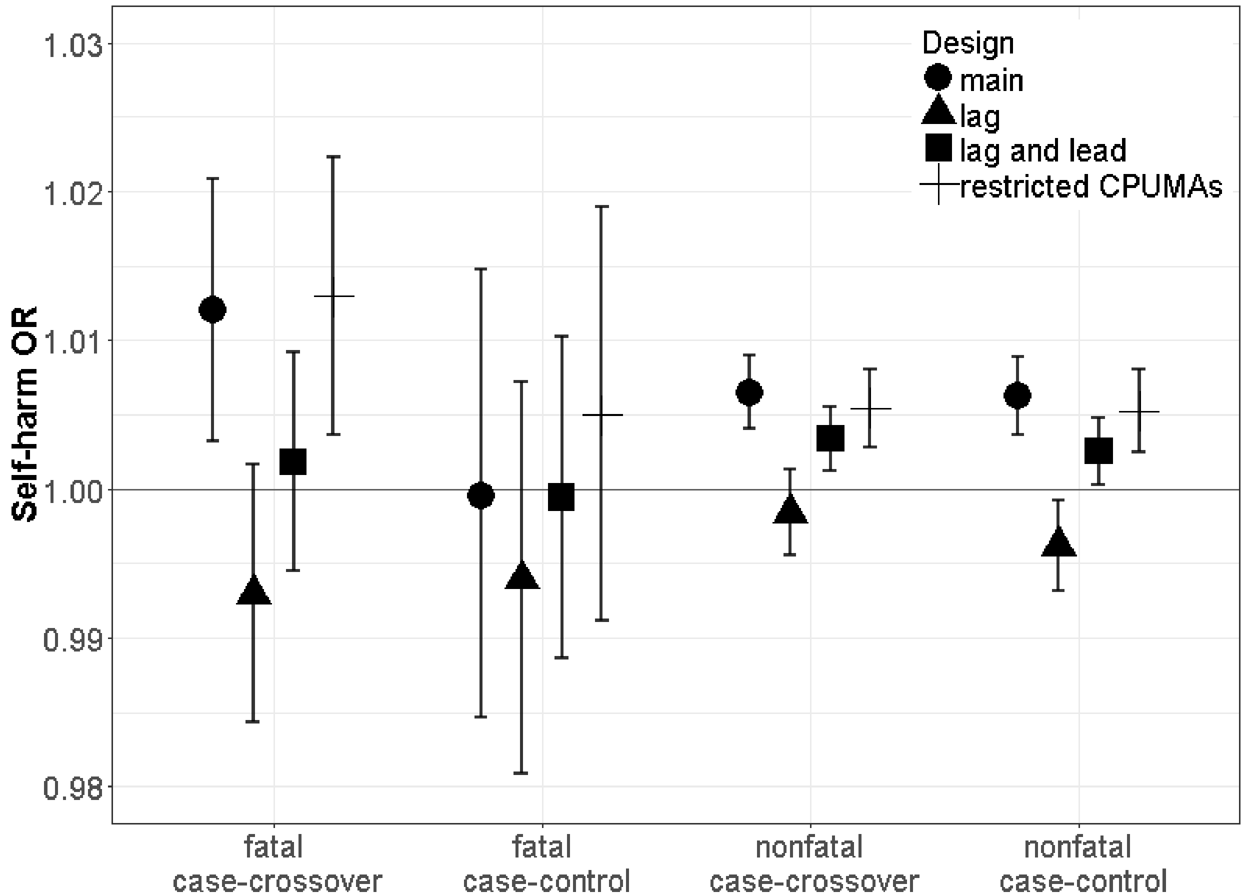


Figure 3: Relative risk of fatal and nonfatal self-harm associated with higher-than expected levels of 30-day community violence, California, 2005-2013



X-axis labels indicate the outcome type (fatal vs. nonfatal self-harm) and the study design (case-crossover vs. case-control). Point shapes indicate the design. Main: main analysis, controls drawn with 30-day lead. Lag: sensitivity analysis, controls drawn with 30-day lag. Lag and lead: sensitivity analysis, controls draw with both 30-day lag and lead. Restricted CPUMAs: sensitivity analysis, controls drawn with 30-day lead, analysis restricted to CPUMAs with absolute value violence residual autocorrelation less than 0.2. Acute community violence was defined as the difference between the observed rate and the Kalman smoothed rate of community violence.

CHAPTER 5: CONCLUSIONS

In this dissertation, I investigated epidemiologic trends in rates and means of fatal and nonfatal self-harm in California between 2005 and 2013. I then examined the association of overall levels and acute variation in community violence—an important and potentially modifiable feature of the social environment—as one possible contributor to self-harm risk and to changing patterns in self-harm in California.

In Aim 1, I detected concerning trends in self-harm in certain demographic groups that indicate the need for increased efforts to mitigate rising rates of self-harm. This research identified current and emerging high-risk groups that may benefit from targeted intervention and suggested that who is at highest risk is changing rapidly, motivating population-level interventions that do not require identifying high-risk individuals.

In Aims 2 and 3, I found that community violence is a risk factor for self-harm in the California population. In Aim 2, I focused on overall levels of community violence, as a long-term pre-disposing risk factor, and estimated population-level intervention parameters that correspond to hypothetical interventions. In Aim 3, I focused on acute, within-community deviations in violence from expected levels, as a potential acute trigger of self-harm. Both overall levels of community violence and acute increases from expected levels were associated with an increased risk of nonfatal self-harm. Acute increases from expected levels were also associated with fatal self-harm.

There are several possible reasons why fatal self-harm may be associated with acute deviations from expected levels but not overall levels of community violence. First, these differences may reflect distinctions in the study design and analytic approaches—and thus differences in the parameters estimated. Approaches to both Aims 2 and 3 were selected with the goal of minimizing likely sources of bias in each application, including individual-level and structural confounding, same-source bias, and control selection bias, but the methodology in Aim 2 produced additive, population-level parameters while the methodology in Aim 3 produced conditional, within-community multiplicative parameters. Using the same approach for both Aims would eliminate discrepancies due to differing analytic methods, but may introduce other, even greater biases. Second, these differences may result from differing levels of confounding control achieved with each approach. For example, analysis of fatal versus nonfatal self-harm controlled for different covariates depending on availability in death, discharge, and survey records, the case-crossover design better-controls individual-level confounders, and the smaller effect sizes identified in Aim 3 may be more vulnerable to small amounts of residual confounding. Finally, these differences may reflect differing mechanisms by which community violence influences different forms of self-harm. For example, acute deviations from expected levels of community violence, perhaps as an acute trigger, may matter more for fatal self-harm risk than long-term, perhaps predisposing, levels. As discussed throughout this work, the distribution and determinants of self-harm differ by type, and thus, we would not necessarily expect their association with overall levels and acute deviations in community violence to be similar. Disentangling these distinct explanations is an area for future research.

This work reflects several challenges and tradeoffs in studying social ecological determinants of self-harm. For example, the rarity of self-harm motivated the use of population-wide records-based data, but such data are generally cross-sectional and lack detailed covariate information.

The case-crossover design used in Aim 3 provides greater control of individual-level covariates but requires that the data be analyzed as matched pairs using conditional logistic regression which inhibits estimating population-level (marginal), additive parameters which may be more informative for public health. The final analytic methods and results I present in this dissertation are those that I believe minimize the most important sources of bias present in research on social ecological determinants of self-harm, but other approaches weighing these concerns differently may also be valid. In this regard, a formal assessment of how the results differ by approach or a simulation study to compare the performance of different design and analytic methods may be valuable in future research.

Future research directly examining the health impacts of violence prevention programs and policies would also be valuable. Exposure to community violence is both very common and very unequally distributed, with the majority of community violence occurring in poor, racially and ethnically segregated urban neighborhoods. Therefore, the health impacts of exposure to community violence have important implications for public health and health equity. Community violence, though not fully understood, is the product of a confluence of factors including poverty, lack of opportunity, and historical disadvantage. Together with community violence, these factors play a substantial role in propagating health disparities among communities that are already vulnerable. While we cannot intervene on historical disadvantage, to the extent that effective community violence prevention programs exist, reducing community violence may be one valuable strategy for mitigating the negative health consequences of historical disadvantage.

There is still much we do not understand about why self-harm occurs, and how it can be prevented. Although not definitively causal, the results of the preceding analyses suggest that rates and means of self-harm in California are changing in ways that merit greater targeted and population-level efforts to address self-harm, and that features of the social environment such as community violence, in various forms, may influence self-harm. Results from this work have implications for public health, public policy, and mental health professionals seeking to design and implement self-harm prevention efforts in California in terms of both who is targeted and the types of interventions selected. Aims 2 and 3 add to the existing literature on the health consequences of exposure to community violence and further motivate violence prevention programs and policies.

This dissertation also has implications for investigators seeking to generate rigorous evidence on social ecological determinants of self-harm, and motivates several areas for future research. This work strengthens evidence on the epidemiology of self-harm in California, the role of community violence as a risk factor for self-harm, and the interconnections between self-directed and interpersonal violence. It overcomes a variety of limitations of previous investigations, and the unique combinations of data sources and carefully selected design and analytic methods used in this work may serve as a model for future research. Self-harm is a complex phenomenon, and a continued focus on methodologically rigorous research will be necessary to better-understand and address its fundamental causes.

APPENDICES

Appendix to Chapter 2

Supplemental methods

Table 1 presents the International Classification of Diseases (ICD) 9th and 10th revision codes used to classify means of self-harm into the nine categories used in this study.

Table 1: International Classification of Diseases 9th and 10th revision codes used to classify means of self-harm

	ICD-9 (hospitalization records)	**ICD-10 (mortality records)**
Poisoning by medicinal substances or drugs	E9500 – 9505	X60-X65
Poisoning by non-medicinal substances or drugs	E9506-E9509, E951-E952	X66-X69
Hanging, strangulation, or suffocation	E953	X70
Drowning	E954	X71
Handgun	E9550	X72
Other firearm or explosive	E9551-E9554	X73
Sharp object, cutting, or piercing	E956	X78
Falls	E957	X80
Other	E958-E959, E9555-E9559	X74-X77, X79, X81-X84

Supplemental results

Figure 1 investigates potential age, period and cohort effects. For total self-harm, within each of the youngest age groups, cohorts born in more recent years experienced notably higher rates of total self-harm, while other age groups showed no cohort effects. In contrast, for fatal self-harm, cohort effects (variation in rates across groups of the same age who were born in different years) were observed in almost all age groups, but the direction of effects varied substantially depending on the age group. In the youngest and oldest age groups, cohorts born more recently experienced lower rates of self-harm, while middle age groups show the opposite pattern, or reversal of this pattern partway through the study period. This figure highlights that the heterogeneous pattern of self-harm rates cannot be explained by differences in self-harm risk by age alone. Reasons for changing risk of self-harm within age groups over time should be investigated further to help identify drivers of changing rates of self-harm.

Figure 1: Cohort effects in age-specific rates of total self-harm and fatal self-harm in California, 2005-2013

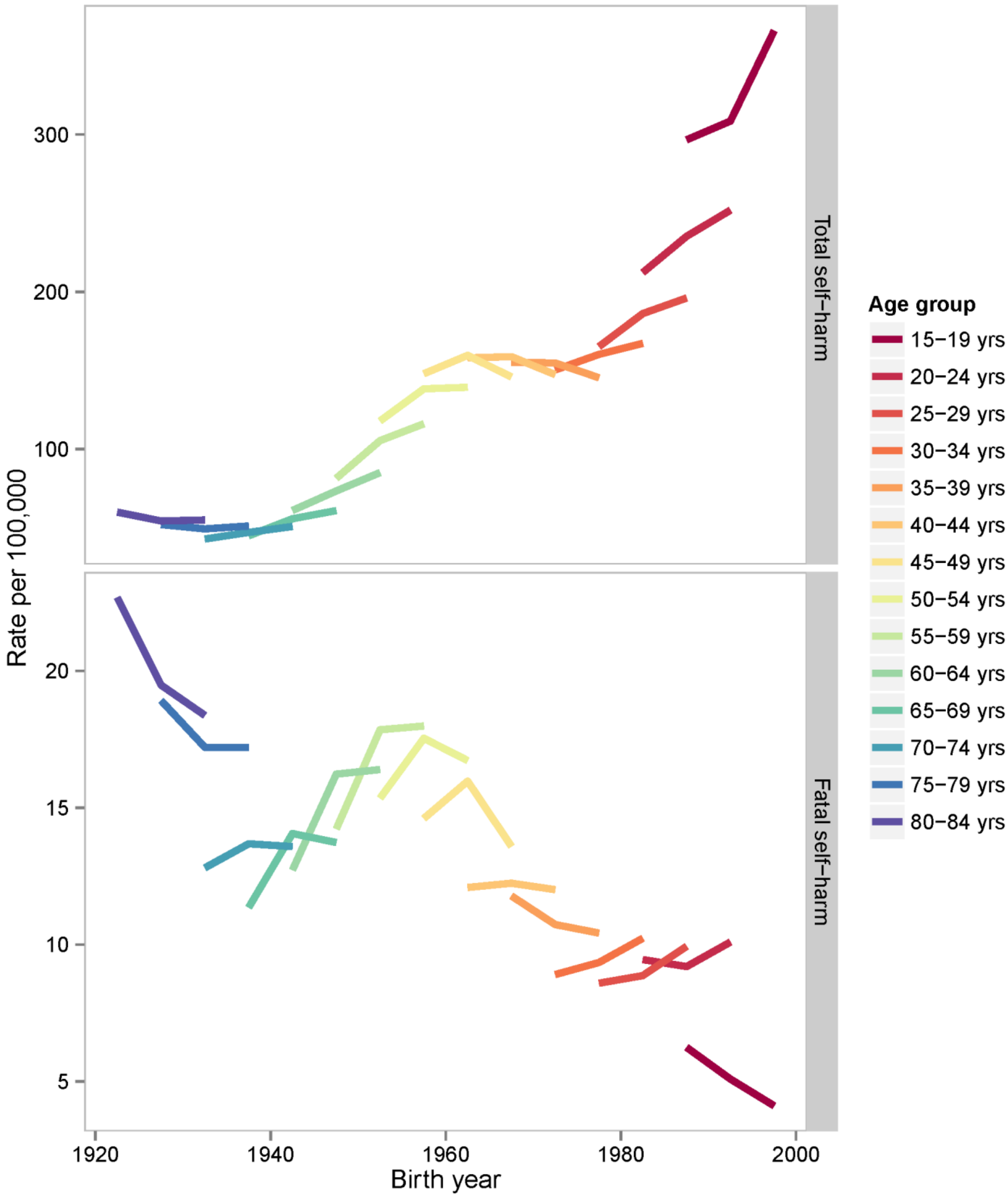


Figure 2 shows levels and trends in age-adjusted rates of total and fatal self-harm by means and urbanicity (urban, suburban and rural). Disparities in total and fatal self-harm rates are immediately apparent, with rural regions generally experiencing much higher rates than urban regions. Increases and decreases in total and fatal self-harm were most pronounced for suburban regions. We present maps displaying the rates, counts, and absolute changes in rates of self-harm by county, which highlight important disparities and spatial patterning of self-harm. In particular, most cases of self-harm occur in urban areas where rates are generally lower, while rural regions have smaller caseloads but manifold greater rates. Additionally, there is wide variation in the trends in self-harm by county, indicating the potential to learn from county level variation in prevention programming and other services what approaches are effective. We also present plots of variation in the dominant means of self-harm by county and the relationship between baseline rates and change in rates during the study period.

Figure 2: Trends in age-adjusted rates of total self-harm and fatal self-harm, by means and urbanicity, California, 2005-2013

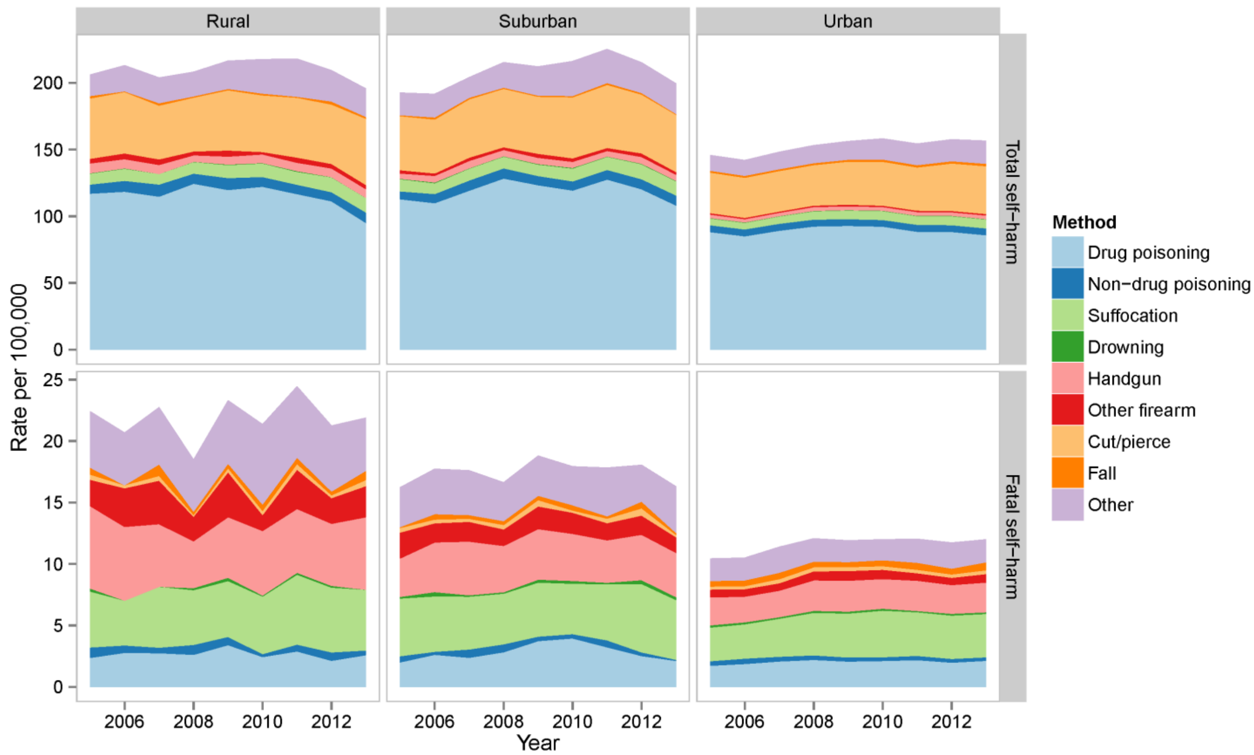


Figure 3 presents age-adjusted rates and unadjusted counts of total self-harm and fatal self-harm in California in 2013 by county. Of note, the spatial patterning of counties with high and low rates is the inverse of that for counts, highlighting that most cases occur in urban areas where rates are generally lower, while rural regions have smaller caseloads but manifold greater rates. A key tension moving forward will be to balance prioritizing intervention in urban areas where the most cases occur with prioritizing rural areas where rates are highest. Arguments for efficiency would point to the former while arguments for social justice would point towards the latter.

Figure 3: Age-adjusted rates and unadjusted counts of total self-harm and fatal self-harm in 2013 in California counties

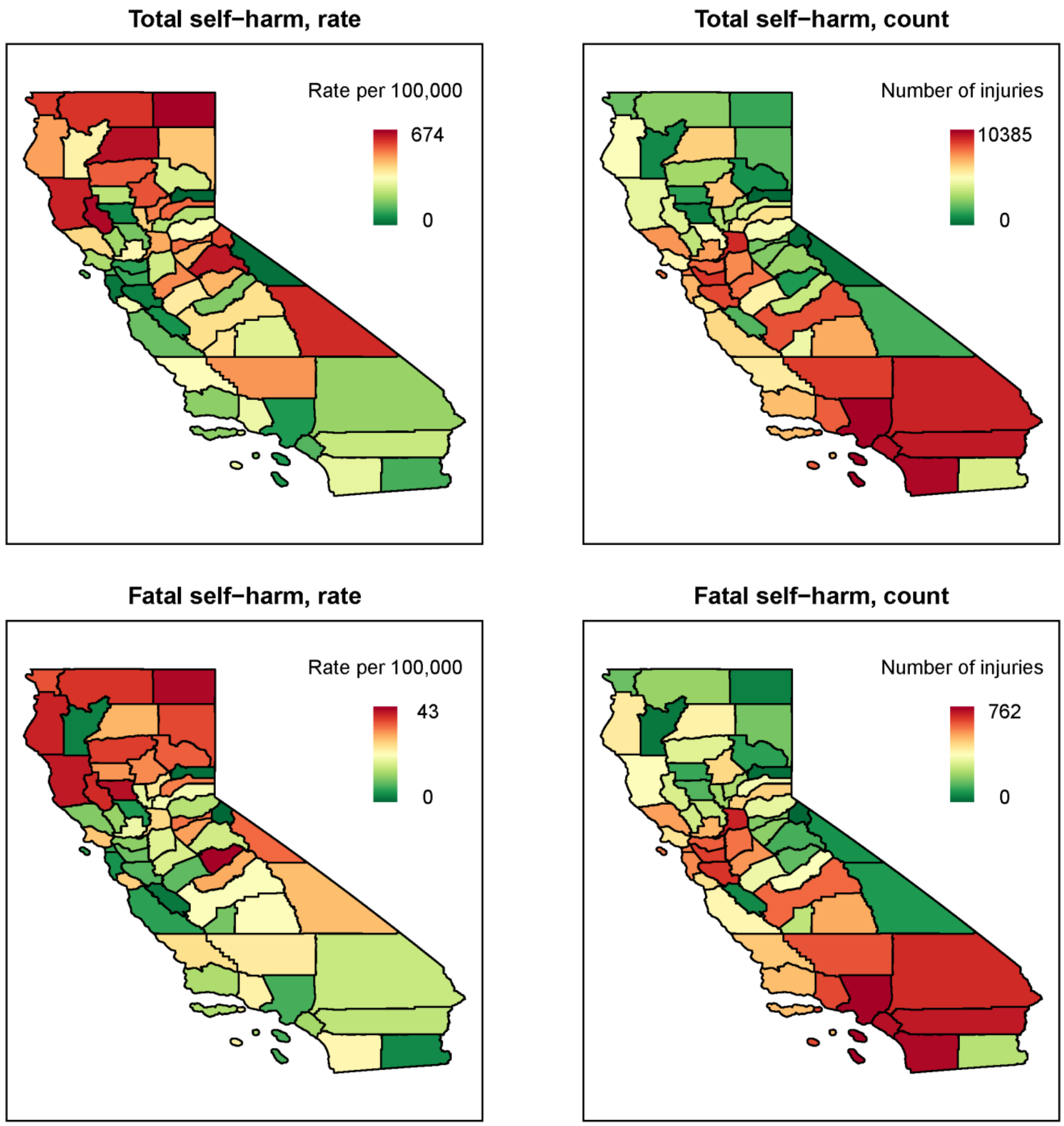


Figure 4 presents the dominant means of total self-harm and fatal self-harm by county in 2013. Drug poisoning was the dominant means of total self-harm in all but three counties, but the dominant means of fatal self-harm varies substantially by county. Thus, efforts targeting the use of particular means in particular places may be more effective in reducing fatal self-harm than efforts that cater less to the individual circumstances in each region. Uncolored counties indicate that there were no cases of total self-harm or fatal self-harm for that county in 2013.

Figure 4: Dominant means of total self-harm and fatal self-harm in 2013 in California counties

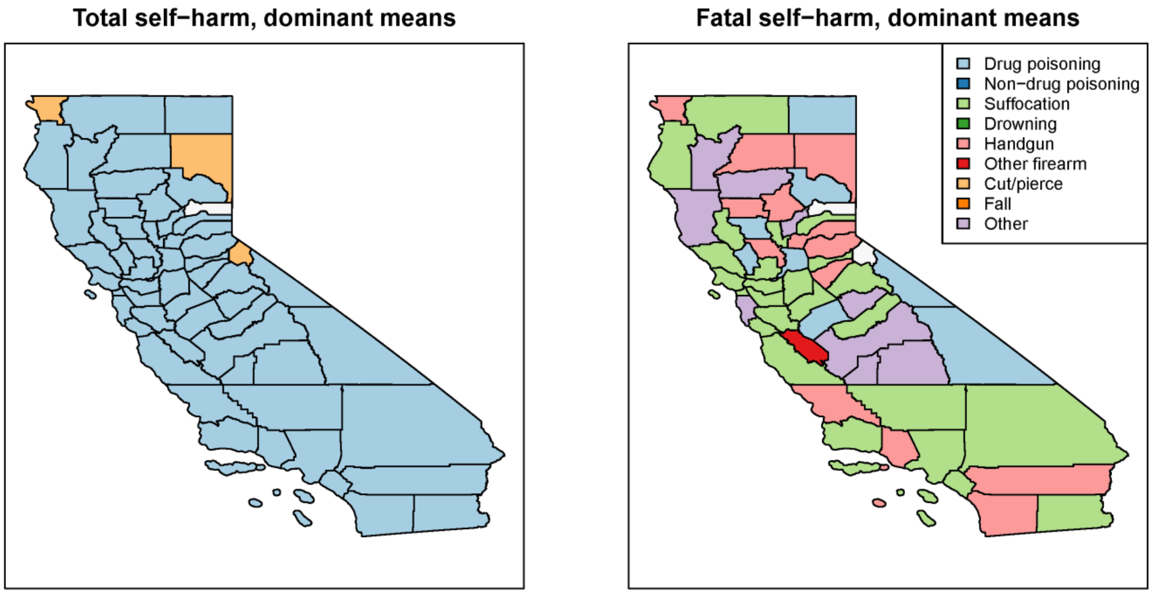
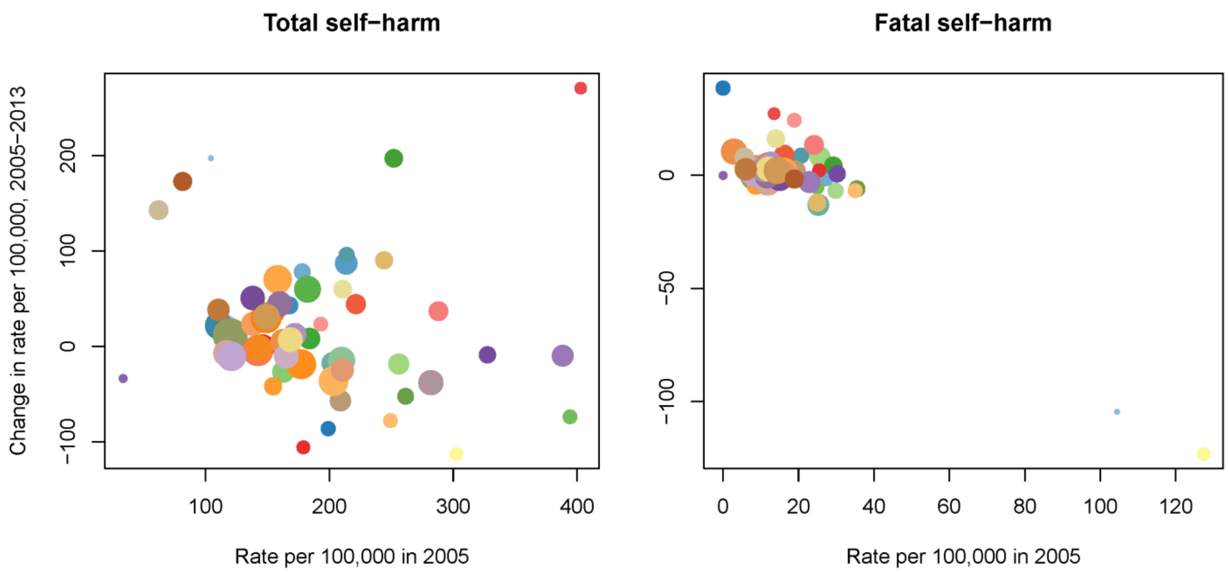


Figure 5 presents the relationship between county-level baseline age-adjusted rates of total self-harm and fatal self-harm in 2005 and the absolute change in age-adjusted rates of total self-harm and fatal self-harm between 2005 and 2013. The size of each point is proportional to logarithm of the 2010 population in the corresponding county. Of interest here is the lack of a systematic association. With the exception of two counties in the lower right plot for fatal self-harm, these plots indicate that the observed increases and decreases in self-harm are not a function of random variation followed by regression to the mean. Some counties are high and remain high, while some counties start out high and decline dramatically; some counties are low and remain low while others are low and increase dramatically. These plots suggest that explanations for the variation in trends in rates of self-harm across counties is complex, and individual case studies of particular counties may be necessary to uncover the drivers of these trends.

Figure 5: Absolute change in age-adjusted rates of total self-harm and fatal self-harm between 2005 and 2013 versus baseline rates in 2005 by county, California



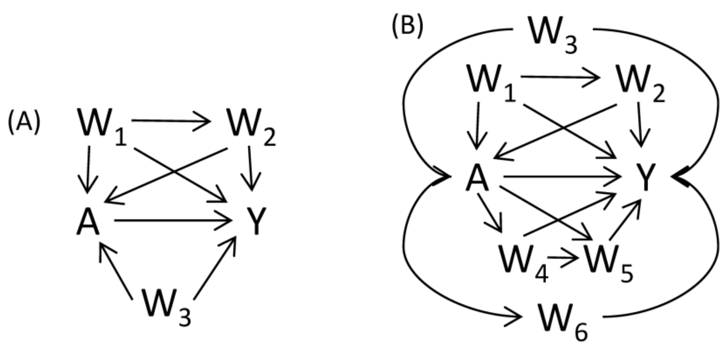
Appendix to Chapter 3

Directed Acyclic Graphs

Figure 1 presents two versions of the directed acyclic graph (DAG) considered for this study. A represents the exposure, community violence. Y represents the outcome, self-harm, and the W variables are the covariates considered. We present two versions of the DAG because several of the covariates may both influence and be influenced by community violence. Figure 1A presents the DAG in which these factors are confounders. Figure 1B presents the DAG in which these factors are mediators. Figures1A and 1B represent the extreme scenarios where either all covariates are confounders, or all potential mediators are mediators, respectively. Intermediate DAGs where some potential mediators are mediators, but others are confounders are also possible. To be conservative, we used the DAG from Figure 1A, which indicates more adjustment, to inform the covariates used in the final analysis. Several of the covariates are unmeasured.

Several covariates also operate at both the community and individual levels, and these different levels influence each other. For example, community-level drug use may influence individual-level drug use through mechanisms such as social norms, and individual-level drug use may contribute to community-level drug use because the individual is a member of the community and individuals using drugs may be more likely to live in high-drug use communities. Thus, there may be pathways both from the individual level measure to the community level measure and from the community-level measure to the individual-level measure. In both DAGs, we represent the individual-level measures as influencing the community-level measures, because we believe residential selection is the strongest of the forces at work, but alternative formulations are possible. The implications for confounding control do not change.

Figure1: Directed acyclic graphs



The covariates for each DAG are as follows:

<u>Figure 1A</u>

W1 represents individual-level confounders that may also operate at the community-level. W2 represents community-level confounders that may also operate at the individual level. W3 represents other confounders not fitting this structure.

W1 and W2 (individual and community level, respectively): drug and alcohol use (acute intoxication and use disorders); history, type, extent, and treatment of mental disorders and symptoms (personal, family, or community); early life adversity; adverse life events (e.g. loss of a loved one or a job); psychosocial crises; socioeconomic status and disadvantage (poverty, income, occupation, education); physical health (chronic conditions, functional ability, pain); access, receipt, and quality of physical and mental health services; social support and isolation; ownership of and access to firearms and other lethal means; social capital.

W3: previous self-harm and violence towards others; personality traits (emotion regulation, aggression, impulsivity); sexual orientation and gender identity; natural disasters; genetic factors; neighborhood disorder; community engagement; macroeconomic trends; month/season; characteristics of the physical environment (parks, heat, sunlight, rain, etc.).

Figure1B
W1 represents individual-level confounders that may also operate at the community-level. W2 represents community-level confounders that may also operate at the individual level. W3 are other confounders not fitting this structure. W4 are individual-level mediators that may also operate at the community-level, and W5 are community-level mediators that may also operate at the individual level, and W6 are other mediators not fitting this structure.

W1 and W2 (individual and community level, respectively): socioeconomic status and disadvantage (poverty, income, occupation, education). Note that these factors are plausible mediators – community violence may influence disinvestment in communities and lack of job opportunities, for example. However, we believe their influence as confounders is predominant.

W3: previous self-harm and violence towards others; personality traits (emotion regulation, aggression, impulsivity); sexual orientation and gender identity; natural disasters; genetic factors; macroeconomic trends; month/season; characteristics of the physical environment (parks, heat, sunlight, rain).

W4 and W5 (individual and community level, respectively): drug and alcohol use (acute intoxication and use disorders); history, type, extent, and treatment of mental disorders and symptoms (personal, family, or community); early life adversity; adverse life events (e.g. loss of a loved one or a job); psychosocial crises; physical health (chronic conditions, functional ability, pain); access, receipt, and quality of physical and mental health services; social support and isolation; ownership and access to firearms and other lethal means; social capital.

W6: neighborhood disorder; community engagement.

Confounder assessment

Individual- and community-level confounders were identified *a priori* based on the scientific literature and the development of a directed acyclic graph.[1] We considered established risk factors for self-harm and factors that affect community violence or share common causes with community violence. Variables controlled in the final analysis depended on availability in death, discharge, and ACS records.

Individual-level confounders included in analyses of fatal self-harm were marital status (married/partnered, divorced/widowed, or single never-married), education (high school and 4-year-college completion), foreign born (yes/no), history of military service (yes/no), and recent immigration to the United States (years of residence in US is more/less than 5 years). Analyses of nonfatal self-harm controlled for individual-level primary language spoken (English/not English). Health insurance type (Medicaid, other insurance, or none), a proxy for socioeconomic status, was available in the ACS after 2008 and was included in sensitivity analyses restricted to this time period.

Community-level confounders controlled for in both analyses included the following: percent male, percent Hispanic, percent non-Hispanic Black, percent non-Hispanic Asian or Pacific Islander, percent non-Hispanic American Indian/Alaskan Native, percent non-Hispanic multiracial, percent renters, percent single-parent households, percent foreign born, percent separated, divorced, or widowed, percent males aged 15 to 29, percent unaffiliated youth, percent moving residence in previous year, percent with a cognitive, ambulatory, independent living, self-care, vision, or hearing difficulty (source: ACS; time frame: annual estimates); population (US Census; annual); alcohol outlet density and social organization density (a proxy for social cohesion) (US Census Zip Code Business Patterns; annual); a validated proxy for firearm ownership constructed from percent firearm suicides and hunting licenses per capita (California Vital Records and California Department of Fish and Wildlife, and US Census; annual);[2] mean self-reported mentally unhealthy days per month (California Health Interview Survey; bi-annual); primary care providers per capita (Health Resources and Services Administration Area Resource File; annual); unemployment (Bureau of Labor Statistics; monthly); and average temperature and average precipitation (WestMap Climate Analysis PRISM Climate Mapping Program; monthly).

We excluded community-level covariates (median income, percent below poverty, racial/ethnic composition, percent English speaking, percent veterans, marital status composition, educational composition, percent employed, percent searching for work, percent living alone, population density, average number of physically unhealthy days in previous month, percent of suicides completed with firearms, alcohol outlet count, health food establishments count and density, social organizations count) that were excessively correlated with other covariates in the control set. When necessary, we used the Missouri Census Data Center Geographic Correspondence Engine to crosswalk covariate values from other geographic units to CPUMAs.[3] Self-harm cases and corresponding controls occurring in the first half of each year were assigned annual covariates from the previous year; self-harm cases and corresponding controls occurring in the second half of each year were assigned annual from the same year.

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Sample sizes

The final analytic sample for this study included 27,027 fatal self-harm injuries, with 108,108 corresponding controls, and 331,203 nonfatal self-harm injuries, with 1,324,812 corresponding controls. Table 1 summarizes the study sample sizes by age and sex strata.

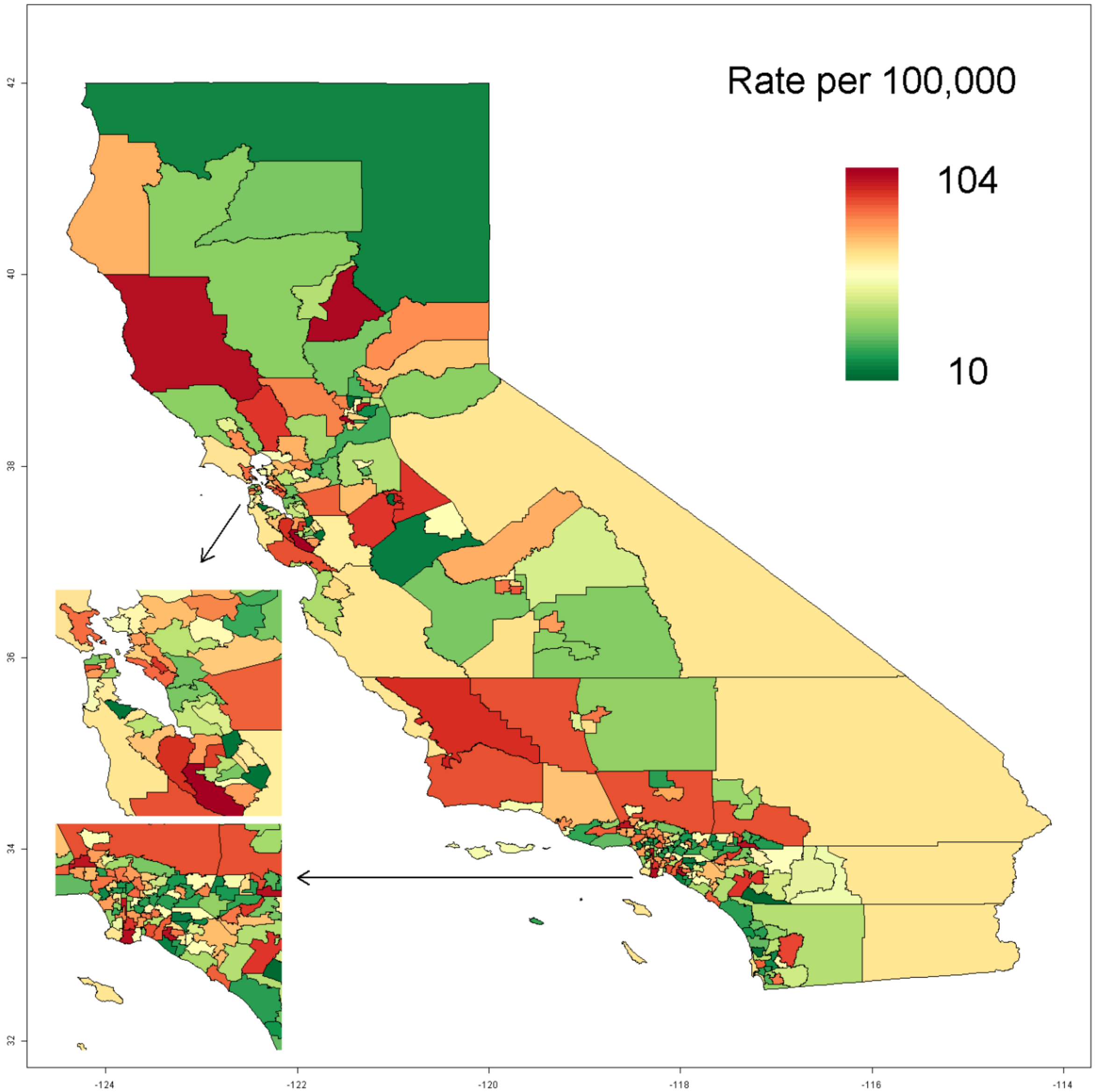
Table 1: Study sample sizes overall and by strata

Characteristic		Fatal				Nonfatal			
		Female		Male		Female		Male	
		Cases	Controls	Cases	Controls	Cases	Controls	Cases	Controls
All		6,344	25,376	20,683	82,732	192,573	770,292	138,630	554,520
Age group	15-19	263	1,052	851	3,404	44,760	179,040	22,824	91,296
	20-24	388	1,552	1,671	6,684	27,065	108,260	21,908	87,632
	25-29	396	1,584	1,540	6,160	20,354	81,416	17,688	70,752
	30-34	382	1,528	1,534	6,136	16,664	66,656	13,909	55,636
	35-39	507	2,028	1,641	6,564	16,473	65,892	12,344	49,376
	40-44	647	2,588	1,816	7,264	17,381	69,524	12,495	49,980
	45-49	777	3,108	2,265	9,060	17,119	68,476	12,146	48,584
	50-54	884	3,536	2,355	9,420	13,701	54,804	9,956	39,824
	55-59	726	2,904	2,110	8,440	8,576	34,304	6,691	26,764
	60-64	521	2,084	1,540	6,160	4,556	18,224	3,801	15,204
	65-69	317	1,268	1,008	4,032	2,470	9,880	1,923	7,692
	70-74	192	768	785	3,140	1,455	5,820	1,195	4,780
	75-79	184	736	840	3,360	1,051	4,204	960	3,840
	80-84	160	640	727	2,908	948	3,792	790	3,160

Geographic distribution of exposure

Figure2 displays a map of California with the median 12-month average community violence rate for each CPUMA over the study period 2005 to 2013.

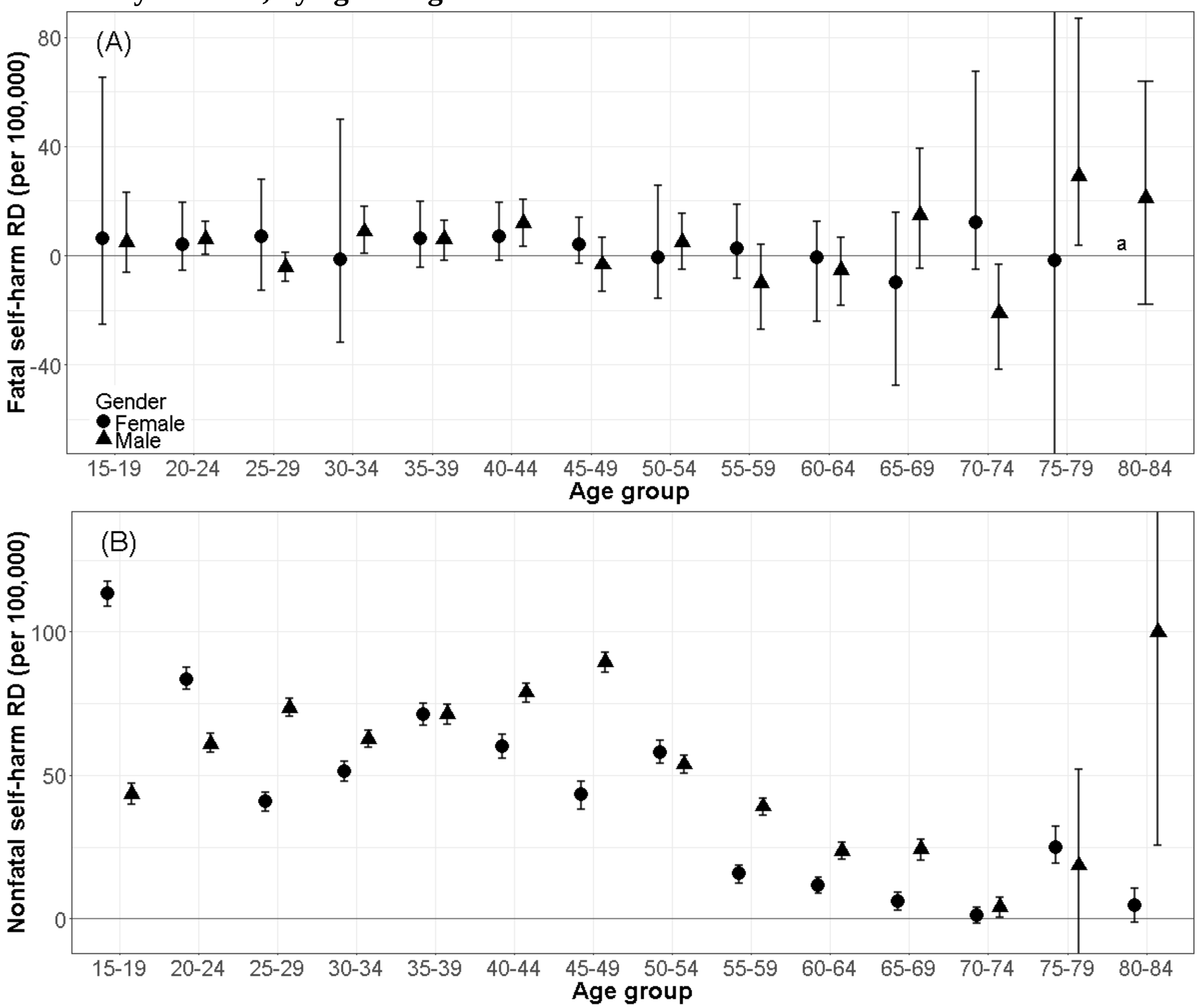
Figure 2: Median monthly community violence rate, 2005-2013



Supplemental results

Figure 3 presents the adjusted overall risk difference for fatal and nonfatal self-harm associated with community violence, stratified by age and gender. Community violence was associated with increased risk of nonfatal self-harm predominantly among the young and middle-aged groups (ages 15-59), with the strongest relationships for women ages 15-24 and men ages 40-49, but was generally not meaningfully associated with fatal self-harm. These estimates are similar in age and gender patterns to those of the RD_{PA} and $RD_{targeted}$, but are larger in magnitude.

Figure 3: Adjusted overall risk difference for fatal and nonfatal self-harm associated with community violence, by age and gender



Risk differences are presented per 100,000 persons per year. (A) Fatal self-harm. (B) Nonfatal self-harm. The overall risk difference compares the estimated risk of self-harm if all individuals were exposed to 12-month average violence rates equal to the highest versus the lowest monthly violence rate observed within their communities. Analyses are adjusted for race/ethnicity, year of injury or survey, and community-level confounders (see Covariate assessment). Analyses of nonfatal outcomes are also adjusted for individual-level primary language spoken. Analyses of

fatal outcomes are also adjusted for individual-level marital status, education, foreign born, military service, and recent immigration to the United States. Confidence intervals for fatal self-harm are asymmetric due to the small sample size of these subgroups and should be interpreted with caution. RD: risk difference. Bars indicate 95% confidence intervals.
[a] Estimate for women ages 80-84 is unstable due to small sample size and is not presented.

Bias analysis

To assess the potential role of confounding due to unmeasured factors, we conducted a quantitative bias analysis for the primary association of interest identified in this study: the overall risk difference for the association of community violence with nonfatal self-harm. We estimated the characteristics of an unmeasured confounder that would yield the observed association between community violence and nonfatal self-harm in California, if (a) the true effect were null or (b) the confidence interval included the null. To do this, we used the bias equation presented by VanderWeele and Arah for the risk difference (RD)[1] and applied it to the estimated overall RD of the association between community violence and nonfatal self-harm.

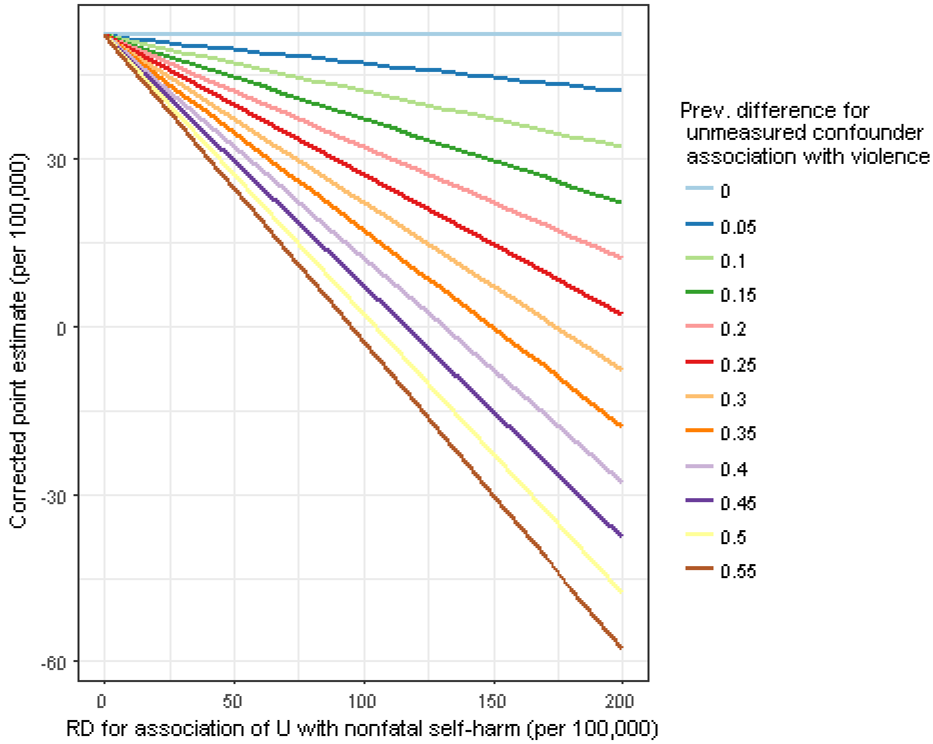
We defined the following random variables: Let A be a continuous measure of community violence exposure, and let a_1 and a_0 represent the highest and the lowest level of violence observed within each study subject's community during the period 2005 to 2013. Let Y be a binary indicator of self-harm, X be the measured covariates controlled in the corresponding analysis, and U be an unmeasured confounder. Consistent with VanderWeele and Arah, we made three assumptions: first, that the association between U and Y does not vary between strata of A; second, that the association between U and A does not vary between strata of X; and third, that U is binary. Under these conditions, the bias in the marginal causal RD is defined as the difference between the observed RD, adjusted for X, and the true marginal causal RD, and is computed as $d_{+a}^{RD}(x) = \gamma\delta$ where γ is the association between U and Y, defined as $\gamma = E(Y|a, x, U = 1) - E(Y|a, x, U = 0)$, and δ is the association between U and A, defined as $\delta = P(U = 1|A = a_1, x) - P(U = 1|A = a_0, x)$.

We estimated (a) the corrected point estimate and (b) the corrected lower confidence bound of the RD for the association of community violence with nonfatal self-harm across a range of bias scenarios. We tested a broad range of plausible values of γ (the RD for the association of U with Y) and δ (the prevalence difference [PD] for the association of U with A), with γ ranging from 0 to 200 per 100,000 and δ ranging from 0 to 0.55. These analyses tell us how strong the U-A and U-Y relationships would have to be, for an uncontrolled confounder to explain the association observed in our study. For both (a) and (b), we used the result from the analysis restricted to 2008-2013 that controlled for health insurance type (observed $RD_{overall}$: 52.2 [95% CI: 50.7, 53.7]), because in addition to better confounding control, it showed a smaller association (thus, more sensitive) compared to the analysis for 2006-2013 that did not control for insurance type (observed $RD_{overall}$: 62.9 [95% CI: 61.9, 63.7]). The observed risk of nonfatal self-harm for this analytic sample was 240 per 100,000.

Figures 4 and 5 present the results for bias analyses (a) and (b). In each plot, the x-axis measures RD for the association of the unmeasured confounder with nonfatal self-harm, the color of each line measures the PD for the association of the unmeasured confounder with community violence, and the y-axis displays the corrected point estimate (Figure 4) or corrected lower confidence bound (Figure 5) for the given bias scenario. For example, when the RD for the U-self-harm association is 100 per 100,000, and the PD for the U-community violence is 0.45, the association of community violence with self-harm would still be meaningfully above the null, with a corrected point estimate of 7.2 per 100,000 and a corrected lower confidence bound of 5.7 per 100,000. Across all of the scenarios we considered, an unmeasured confounder would need to be associated with community violence with a PD of at least 0.5 and be associated with nonfatal self-harm with an RD of at least 100 per 100,000 to yield the observed association, if the true effect were null and non-statistically significant. These U-A and U-Y associations are very large, given that the PD has theoretical maximum of 1.0 (in the extreme case where prevalence is 100% in one group and 0% in the other) and given that the observed nonfatal self-harm risk of 240 per 100,000.

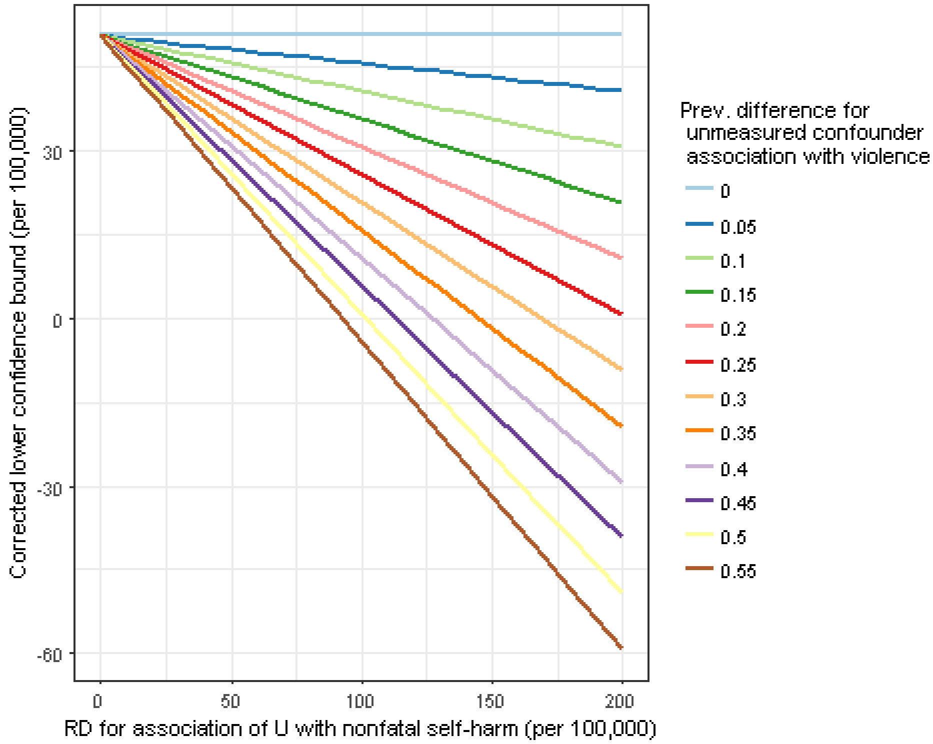
This analysis informs our interpretation of the results. For the association between community violence and nonfatal self-harm in California to be spurious, there would have to be an unmeasured factor that strongly affects both community violence and self-harm. Identifying a factor that fits these criteria is possible. For example, psychiatric disorder strongly increases the risk for self-harm, and also makes one more likely to live in a high-violence community. Confounders of particular concern include the type, extent, and history of mental and substance use disorders, personality traits, early life adversity, and precipitating life circumstances such as the loss of a loved one. However, exposure to community violence may causally precede these (e.g. incite substance use; contribute to the loss of a loved one). If these factors are on the causal pathway, adjusting for them would be inappropriate. Other explanations may also exist.

Figure 4: Bias analysis results for association between community violence and nonfatal self-harm, with corrected point estimate



Risk differences are presented per 100,000 persons per year.

Figure 5: Bias analysis results for association between community violence and nonfatal self-harm, with corrected lower confidence bound



Risk differences are presented per 100,000 persons per year.

References

1. Vanderweele TJ, Arah OA. Bias formulas for sensitivity analysis of unmeasured confounding for general outcomes, treatments, and confounders. *Epidemiol Camb Mass*. 2011;22(1):42-52. doi:10.1097/EDE.0b013e3181f74493

Appendix to Chapter 4

Confounder assessment

To examine acute, within-community variation in violence exposure, we drew controls from the same community as the cases. Thus, community-level confounders that are time-invariant over the study period are controlled by design. The case-crossover design provides additional strength by also controlling time-invariant individual-level confounders. Remaining potential confounders are time-varying community and individual factors for both designs and time invariant individual factors in the case-control design. These were identified a priori based on scientifically established risk factors for self-harm and factors that affect community violence or its determinants.

Individual-level variables controlled in the final case-control analysis depended on availability in death, discharge, and ACS records. Individual-level confounders included in case-control analyses of fatal self-harm were marital status (married/partnered, divorced/widowed, or single never-married), education (high school and 4-year-college completion), foreign born (yes/no), history of military service (yes/no), and recent immigration to the United States (years of residence in US is more/less than 5 years). Case-control analyses of nonfatal self-harm controlled for individual-level primary language spoken (English/not English).

Community-level confounders in both case-control and case-crossover analyses included the following: percent male, percent Hispanic, percent non-Hispanic Black, percent non-Hispanic Asian or Pacific Islander, percent non-Hispanic American Indian/Alaskan Native, percent non-Hispanic multiracial, percent renters, percent single-parent households, percent foreign born, percent separated, divorced, or widowed, percent males aged 15 to 29, percent unaffiliated youth, percent moving residence in previous year, percent with a cognitive, ambulatory, independent living, self-care, vision, or hearing difficulty (source: ACS; time frame: annual estimates); population (US Census; annual); alcohol outlet density and social organization density (a proxy for social cohesion) (US Census Zip Code Business Patterns; annual); a validated proxy for firearm ownership constructed from percent firearm suicides and hunting licenses per capita (California Vital Records and California Department of Fish and Wildlife, and US Census; annual) [1]; mean self-reported mentally unhealthy days per month (California Health Interview Survey; bi-annual); primary care providers per capita (Health Resources and Services Administration Area Resource File; annual); unemployment (Bureau of Labor Statistics; monthly); and average temperature and average precipitation (WestMap Climate Analysis PRISM Climate Mapping Program; monthly).

We excluded covariates (median income, percent below poverty, racial/ethnic composition, percent English speaking, percent veterans, marital status composition, educational composition, percent employed, percent searching for work, percent living alone, population density, average number of physically unhealthy days in previous month, percent of suicides completed with firearms, alcohol outlet count, health food establishments count and density, social organizations count) that were excessively correlated with other covariates in the control set. We used the Missouri Census Data Center Geographic Correspondence Engine as needed to crosswalk covariate values from measured geographic units to CPUMAs [2]. To ensure correct temporal ordering, monthly covariates were assigned for the month prior to injury/survey.

Autocorrelation

To determine whether the Kalman smoother approach successfully isolated acute variation in community violence rates from predictable temporal patterning, including secular trends and seasonality, we assessed the remaining autocorrelation in the model residuals which constitute the measure of acute community violence in this study. Remaining autocorrelation was generally very low. However, to ensure that autocorrelation was not a contributor to our findings, we conducted sensitivity analyses restricted to CPUMAs with |ACF|<0.2 at one lag.

Table 1. Autocorrelation Functions (ACF) for Community Violence Exposure at Different Lags across CPUMAs, California 2005-2013

	Median	% > 0.20 or < -0.20
ACF at 30-day lag	0.08	10%
ACF at 60-day lag	0.07	5%
ACF at 90-day lag	0.08	7%
ACF at 360-day lag	0.08	13%

CPUMA: consistent public use microdata area

References

1. Siegel M, Ross CS, King C. A new proxy measure for state-level gun ownership in studies of firearm injury prevention. *Inj Prev J Int Soc Child Adolesc Inj Prev*. 2014;20(3):204-207. doi:10.1136/injuryprev-2013-040853

2. MABLE/Geocorr12 Version 1.2 - MIssouri Census Data Center. http://mcdc.missouri.edu/websas/geocorr12.html. Accessed July 21, 2017.