Exposure to Community Violence and Self-harm in California

A Multilevel, Population-based, Case–Control Study

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Background: Self-harm is a leading cause of morbidity and mortality. Exposure to community violence is an important and potentially modifiable feature of the social environment that may affect self-harm, but studies to date are limited in the samples and outcomes examined.

Methods: We conducted a population-based, nested case–control study. Cases were all deaths and hospital visits due to self-harm in California, 2006–2013. We frequency-matched California resident population-based controls from the American Community Survey to cases on age, gender, race/ethnicity, and year of survey/injury. We assessed past-year community violence using deaths and hospital visits due to interpersonal violence in the community of residence. We estimated risk difference parameters that were defined to avoid extrapolation and to capture associations between changes in the distribution of community violence and the population-level risk of self-harm.

Results: After adjustment for confounders, setting past-year community violence to the lowest monthly levels observed within each community over the study period was associated with a 30.1 lower risk of nonfatal self-harm 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 nonfatal self-harm at the population level. In sensitivity analyses, results were robust.

Conclusions: This study strengthens evidence on the relationship between community violence and self-harm. Future research should investigate reasons for differential associations by age and gender and whether community violence prevention programs have meaningful impacts on self-harm.

Keywords: Case–control; Community violence; Epidemiologic methods; Positivity; Self-harm; Social epidemiology; Social environment; Suicide

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Self-harm is a leading cause of morbidity and mortality in the United States, accounting for more than 44,000 deaths and 500,000 injuries in 2015. Between 2001 and 2015, rates of fatal self-harm (suicide) increased 24% and rates of nonfatal self-harm increased 39%. 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. Rising rates have drawn attention to self-harm as an important population health issue. Additional research is needed to understand the drivers of self-harm and to identify effective interventions.

Aspects of the social environment such as social fragmentation and inequality are key risk factors for self-harm. Community violence is an important and modifiable feature of the social environment that may contribute to the burden of self-harm, particularly in the United States where levels of community violence are high and rising. Exposure to community violence, meaning witnessing, hearing about, or directly experiencing violence in one’s community, may increase the risk of self-harm in several ways. Increased stress, depressed mood, anxiety, symptoms of posttraumatic stress, and mental disorders can result from exposure to community violence.
and are strong risk factors for self-harm.15–20 Similarly, exposure to community violence can lead to substance use or social isolation (e.g., staying inside),21 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 methodologic 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 (e.g., 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 statewide, except active duty military hospitals, and captured the external cause of death or injury, 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 (International Classification of Diseases (ICD), Ninth Revision, Clinical Modification hospital visit code: E95 and E95 subtypes; ICD-10 death codes: X6–X8 and corresponding subtypes). Self-harm outcomes were included starting in 2006 so that data on community violence were available for the relevant preinjury exposure period (see Exposure Assessment). We made efficient use of an existing population-representative sampling frame by sampling population-based controls42 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 170,000 to 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 weight44 and drew controls from this expanded dataset. For statistical efficiency,43 we matched four 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, Ninth Revision, Clinical Modification 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

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, 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. Studies also indicate completeness and validity of external cause of mortality codes for homicide and self-harm in mortality data. Emergency department records are not available prior to 2005.
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. 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 public ACS microdata. 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 but avoid 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. 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). Crime data may also be used to measure community violence, but differences in reporting practices between jurisdictions and over time may introduce bias.

**Confounding Assessment**

Individual- and community-level confounders were identified a priori based on the scientific literature and development of a directed acyclic graph (eAppendix 1; http://links.lww.com/EDE/B370). 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 birth, 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 eAppendix 2; http://links.lww.com/EDE/B370 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 the following:

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.

**Statistical Analysis**

To estimate these marginal parameters, we used g-computation, 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 subgroups, 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. We used cubic smoothing splines for all continuous independent variables, including community violence, to capture potential nonlinear 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 (1 - \(q_i\))/\(J\) to cases and weights equal to (1 - \(q_i\))/\(J\) to controls, where \(J\) is the ratio of controls to cases. We estimated 95% confidence intervals (CIs) using the nonparametric bootstrap.

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. 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, and we hypothesized that these groups would respond differently to community violence.

We excluded case records with incomplete covariate data (2.8%) 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; see eAppendix 7; http://links.lww.com/EDE/B370 for code), 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, 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 study characteristics. 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 incidence 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 eFigure 2; http://links.lww.com/EDE/B370 for geographic distribution). eAppendix 3; http://links.lww.com/EDE/B370 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. For nonfatal self-harm, the RD overall was 62.9 per 100,000 (CI = 62.0, 63.9) or approximately a 27% reduction in self-harm relative to the observed risk. The RD PA was 30.1 per 100,000 (CI = 29.6, 30.5) or a 13% reduction. The RD targeted was 10.8 per 100,000 (CI = 10.6, 11.0) or a 5% reduction. The median differences in community violence for affected communities were 21.2 per 100,000 for the RD overall and 9.9 per 100,000 for the RD PA and 14.8 per 100,000 RD targeted.

 Associations for the overall population masked substantial subgroup heterogeneity. Figure 1 presents the RD PA by age and gender and shows that community violence was...
TABLE 2. Overall Adjusted Associations Between Exposure to Community Violence and Risk of Fatal and Nonfatal Self-harm

<table>
<thead>
<tr>
<th>Self-harm Type</th>
<th>Risk of Self-harm if All Individuals Were Exposed to High Community Violencea (95% CI)</th>
<th>Risk of Self-harm if All Individuals Were Exposed to Low Community Violencea (95% CI)</th>
<th>Overall Risk Difference (95% CI)</th>
<th>Population Attributable Risk Difference (95% CI)</th>
<th>Targeted Risk Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatal</td>
<td>21.0 (20.1, 22.2)</td>
<td>21.1 (20.0, 22.2)</td>
<td>0.0 (−2.0, 2.1)</td>
<td>0.0 (−1.1, 1.1)</td>
<td>0.2 (−0.2, 0.6)</td>
</tr>
<tr>
<td>Nonfatal</td>
<td>234.8 (267.7, 267.1, 268.3)</td>
<td>204.7 (204.3, 205.2)</td>
<td>62.9 (62.0, 63.9)</td>
<td>30.1 (29.6, 30.5)</td>
<td>10.8 (10.6, 11.0)</td>
</tr>
<tr>
<td>Nonfatal, restricted to 2008–2013, controlling for health insurance type</td>
<td>240.0 (266.4, 265.5, 267.3)</td>
<td>214.2 (213.4, 214.9)</td>
<td>52.2 (50.6, 53.8)</td>
<td>25.8 (25.1, 26.5)</td>
<td>9.7 (9.4, 10.0)</td>
</tr>
<tr>
<td>Nonfatal, inpatient only</td>
<td>76.5 (80.0, 80.5)</td>
<td>73.6 (73.3, 73.8)</td>
<td>6.7 (6.2, 7.1)</td>
<td>2.9 (2.7, 3.1)</td>
<td>1.4 (1.3, 1.5)</td>
</tr>
</tbody>
</table>

aHigh- 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).

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,

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

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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.29,32,56 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-directed and externally-directed violence. Further investigation is needed to disentangle these factors, particularly for nonadolescents for whom existing research is particularly 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 psychologic distress or other psychologic 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,57 whereas fatal self-harm may be a means of escaping distressing environments.5 These are fundamentally different responses, and community violence may

FIGURE 1. Adjusted population attributable 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 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 birth, military service, and recent immigration to the United States. RD indicates risk difference. Bars indicate 95% confidence intervals. aEstimate for women of ages 80–84 years is unstable due to small sample size and is not presented.
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.58,59 There is less research on psychologic vulnerability to stressors among middle-aged men, but this group is less likely to seek or receive needed mental health care.60,61 Thus, untreated mental or substance use disorders or psychologic 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

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**FIGURE 2.** Adjusted targeted 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 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 birth, military service, and recent immigration to the United States. RD indicates risk difference. Bars indicate 95% confidence intervals.

"Estimate for women of ages 80–84 years is unstable due to small sample size and is not presented."
community violence, 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. Differences in the magnitudes of the overall risk difference (RDoverall), population attributable risk difference (RDPA), and targeted risk difference (RDtargeted) reflect differences in the levels of community violence contrasted and the proportion of people affected. The RDoverall intervenes on everyone maximally, the RDPA intervenes on individuals exposed to higher-than-minimum violence to varying degrees, and the RDtargeted intervenes most restrictively on only individuals in high-violence communities. The RDtargeted 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 and mentoring programs for delinquent youth are examples of scientifically supported, locally targeted programs to reduce community violence that would fit this hypothetical scenario.

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. 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 before injury/survey. Third, we used distinct data sources to draw cases and controls, which may generate differences in the measurement of covariates. 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 8-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, 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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REFERENCES


