

Reducing gunshot victimization in high-risk social networks through direct and spillover effects

George Wood ^{1,2} and Andrew V. Papachristos ^{2,3*}

More than 60,000 people are victimized by gun violence each year in the United States. A large share of victims cluster in bounded and identifiable social networks. Despite a growing number of violence reduction programmes that leverage networks to broaden programmatic effects, there is little evidence that reductions in victimization are achieved through spillover effects on the peers of participants. This study estimates the direct and spillover effects of a gun violence field intervention in Chicago. Using a quasi-experimental design, we test whether a desistance-based programme reduced gunshot victimization among 2,349 participants. The study uses co-arrest network data to further test spillover effects on 6,132 non-participants. Direct effects were associated with a 3.2-percentage point reduction in victimization among seeds over two years, while potential spillover was associated with a 1.5-percentage point reduction among peers. Findings suggest that peer influence and the structure of networks might be leveraged to amplify gun violence reduction efforts.

In 2016, more than 14,000 people in the United States were shot and killed by another person, while another 70,000 were wounded by guns in assaults^{1,2}. Gun violence and its effects are not evenly distributed across the population, however. The trauma and consequences associated with gun violence disproportionately affect young minority men in socially and economically disadvantaged neighbourhoods^{1,3–5}. Recent research demonstrates that gunshot victimization further concentrates in small, circumscribed social networks within high-risk populations^{6–8}. For example, 70% of all victims of non-fatal gunshot injuries in Chicago could be located in a co-arrest network comprising less than 5% of the city's population⁷. A study of a high-crime Boston community found that nearly 85% of all victims of fatal and non-fatal gunshot injuries could be located in a network comprising 763 connected individuals⁸. Importantly, the structure of such social networks and individuals' placement within them can severely elevate the risk of victimization and contribute to the diffusion of violence. Cascades of gun violence within high-risk networks starting with one victim whose associate is subsequently victimized, and so on, accounted for 63% of victimizations over an eight-year period in Chicago⁹.

The concentration of gun violence in social networks has implications for gun violence reduction efforts to the extent that identifying and leveraging such networks might enhance violence prevention and reduction practices. Many violence reduction efforts already implicitly or explicitly rely on this sort of networked logic, by concentrating efforts on high-risk individuals and groups within small geographic areas. The central premise of such programmes is that gun violence might be reduced by dissuading specific individuals or groups from involvement in violence, by providing information, resources, mediation or other services directly to those at the highest levels of involvement in gun crime and violence^{10,11}. Furthermore, some programmes implicitly seek to engender spillover effects on unassigned individuals by placing outreach workers and mediators into the networks of individuals involved in violent disputes^{12,13}, or else by encouraging individuals who are part of interventions to propagate a programme's message within their personal networks¹⁴.

While mounting evidence suggests that gun violence reduction programmes focusing on small geographic areas or a small number of groups are effective in reducing aggregate levels of gunshot violence^{15,16}, we know little about the magnitude of programme effects on treated individuals and even less about whether victimization can be reduced through network spillover effects. To date, most evaluations have analysed aggregate levels of gun violence either in geographic regions or among specific groups or gangs in which a programme has been implemented, comparing the frequency of victimization before and after implementation¹⁷. Such aggregated approaches are limited in estimating the magnitude of the effect on treated individuals because they capture victimization outcomes among individuals unaffected by the programme while being affected by shifts in the incidence of gunshot violence caused by exogenous, non-programme-related factors^{18,19}. In addition, it has been difficult to test whether programmes reduce victimization among untreated but potentially affected individuals through spillover effects, largely because it has been infeasible to identify such individuals and difficult to establish a causal framework for estimating spillover²⁰. As such, a central question of precisely how violence reduction programmes reduce the incidence of gunshot victimization remains open.

The intervention we report attempted to reduce gunshot victimization among high-risk participants, as well as among their unassigned peers, through spillover effects. The programme is an 'induction intervention'^{21,22} that attempted to activate peer-to-peer diffusion of a desistance message by encouraging intervention participants to spread a behavioural stimulus.

For the intervention, a group comprising law enforcement, community members and social service agencies carried out an exercise to map violent conflicts, disputes and episodes of gun violence to identify individuals at high risk of involvement in group-involved gun violence²³. These individuals were subsequently invited to attend a one-hour meeting^{24,25}. At these meetings, which were held in-person at a public location such as a local park district, school or community centre, participants engaged in a four-stage process. First, law enforcement officers delivered a message that the

¹Department of Internal Medicine, Yale University, New Haven, CT, USA. ²Institute for Policy Research, Northwestern University, Evanston, IL, USA.

³Department of Sociology, Northwestern University, Evanston, IL, USA. *e-mail: avp@northwestern.edu

individual is at acute risk of victimization and that further involvement in gun violence would be met with coordinated enforcement action and criminal justice sanctions. Second, community representatives emphasized that the participant is valued by their community and extended an invitation for the participant to become involved in positive community activities to avoid victimization. Third, in a process akin to perspective-taking²⁶, victims or relatives of victims of gunshot violence recounted the effects and trauma associated with gun victimization. Lastly, participants were offered direct links to local social service provision. Throughout the meeting, participants were encouraged to spread the desistance and community message, perspective-taking and service provision information to their peers^{27–29}. See Supplementary Notes for further programme information. Invited individuals may or may not attend the meeting, and are designated compliant or non-compliant accordingly. There are no punitive consequences for non-compliance.

To estimate the effects of these meetings, we conducted a quasi-experimental evaluation using participation data and administrative data. We first identified those individuals who were invited to, and attended, a meeting ($n = 1,642$) and those who were invited, but not did not attend, a meeting ($n = 707$), whom we call compliant and non-compliant seeds, respectively. We then constructed a co-arrest network³⁰ around each compliant and non-compliant seed, where each seed is connected to all non-invited individuals alongside whom they were arrested in the three years before the meeting. We define an exposure mapping^{31,32} in which unassigned individuals are designated as either compliant peers ($n = 3,034$) if they are connected to a compliant seed—and therefore potentially exposed to the intervention message—or non-compliant peers ($n = 3,098$) if they are connected to a non-compliant seed (see Supplementary Table 1 for a summary of seed and peer units' covariate profiles). To estimate the main effect of the meeting, we compared victimization outcomes in the two years following invitation to the programme for the compliant seeds against the non-compliant seeds. To estimate the spillover effect, we compared victimization outcomes in the two years after potential exposure to the intervention message for the compliant peers against the non-compliant peers. Figure 1 shows the identification of peer units and the comparisons for the main effect and spillover effect. For each peer, potential exposure to spillover begins on the day that the seed to whom that peer is connected was invited to attend a meeting. Finally, to test the spillover effect under additional exposure to programme participants, we compared victimization outcomes for peers with connections to two or more compliant seeds and those connected to two or more non-compliant seeds.

By linking programme assignment data with administrative data on the networks of participants and their associates, we are able to map potential pathways of diffusion and thereby determine whether gunshot victimization is reduced among individuals assigned to the programme, and among individuals indirectly exposed to treatment through their network connections. One research design feature warrants further exposition. The programme uses non-random assignment and selects individuals deemed to be at high risk of victimization. As such, we could not compare those assigned to attend a meeting against a random selection from the wider Chicago population (see Supplementary Results for details). Exploiting non-compliance with the programme permits a comparison among units deemed to be similarly high-risk *ex ante*. However, we cannot be certain that the choice to comply or not comply with the intervention is unconfounded with the risk of victimization. In estimating the effects of the intervention on compliant seeds, we must therefore account for the probability of selection into the compliant or non-compliant condition. Moreover, because the risk of victimization among connected peers and seeds is plausibly associated, we must also account for the probability of being connected to a compliant or non-compliant seed.

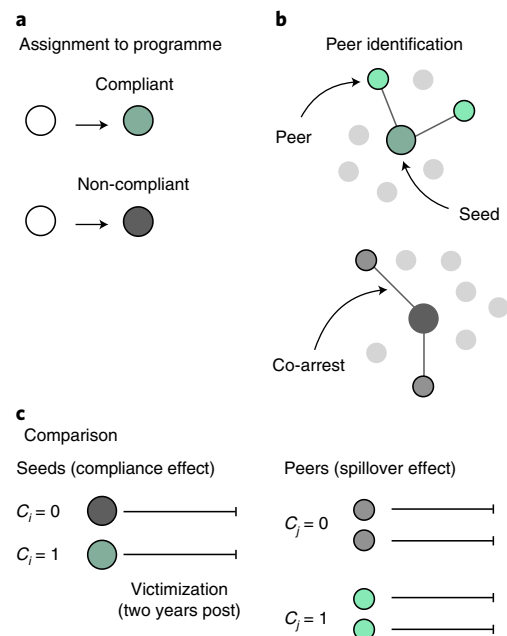


Fig. 1 | Design of the intervention evaluation. **a**, Individuals at high risk of gunshot victimization (seeds) are assigned to the programme and classified as either compliant or non-compliant. **b**, Each seed is identified in the Chicago co-arrest network. We apply our exposure mapping, classifying individuals adjacent to a compliant seed as compliant peers and those adjacent to a non-compliant seed as non-compliant peers. **c**, The compliance effect is based on a comparison of victimization outcomes in the two years after assignment for seeds in the compliant and non-compliant conditions. The spillover effect is based on the equivalent comparison among peers.

To address this problem of confounding, we estimate the effect of compliance on victimization outcomes conditional on observed covariates using Bayesian additive regression trees (BART). BART has been shown to be more accurate than propensity score-based estimators in estimating treatment effects under confounding, particularly when the parametric relationship between the outcome, treatment and confounders is unknown³³. This is in part because BART naturally allows for possible interaction effects and nonlinearities in this relationship^{33–35}. We then compare the BART estimate to a simple difference-in-means (DIM). We use this comparison to assess the proportion of the observed difference in victimization outcomes that is attributed to the compliance and spillover effects after adjusting for confounding due to selection into compliance. Finally, as an extension, we use the flexible BART approach to estimate the heterogeneity of the compliance effect. In Supplementary Results, we show further estimates of the compliance and spillover effects using a more conventional logistic regression model with unit re-weighting based on entropy balancing (EBAL)³⁶. See Methods for further design and estimation details.

Results

Compliance effect on victimization. Compliance reduced the probability of gunshot victimization in the two years after attending an intervention meeting. The share of non-compliant individuals who were victimized in the two years following non-attendance was 18.1%; for compliant individuals, the corresponding share was 10.6%. From the BART model, we estimate that compliance caused a median reduction in the chance of victimization of -3.2 percentage points (Fig. 2; mean estimate, -3.2 , credible interval, -4.9 to -1.4). The magnitude of this reduction is substantially larger in

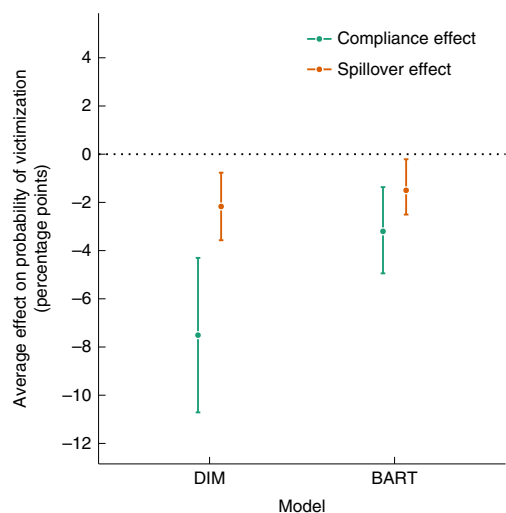


Fig. 2 | The estimated effect of compliance on programme seeds ($n = 2,349$) and the estimated effect of compliance spillover on programme peers ($n = 6,132$). DIM is the mean difference in the two-year probability of victimization for individuals in the compliant versus non-compliant condition. The BART model is a non-parametric estimate of the conditional average treatment effect of compliance, accounting for observed covariates. A 95% confidence interval is shown for the DIM estimate, and a 95% credible interval for the BART estimate.

the DIM model (estimate, -7.5 , confidence interval, -10.7 to -4.3 , $P < 0.001$), which does not adjust for selection into compliance and simply reflects the 7.5-percentage point difference between compliant and non-compliant units in post-intervention victimization outcomes. The reduced effect size estimate in the BART model shows that adjusting for selection into compliance accounts for part of this difference in observed victimization outcomes. The BART estimate attributes 42.6% of the difference in victimization outcomes to the effect of compliance. The estimated reduction in victimization caused by compliance is supported in the EBAL model (see Supplementary Results).

Spillover effect on victimization. Analysis of peer outcomes showed that units with a social connection to a compliant seed had a lower rate of victimization than those with a connection to a non-compliant seed. In the two years following potential exposure to programme spillover effects, the share of non-compliant peers victimized was 9.7% compared to 7.5% among compliant peers. From the BART model, we estimate that compliance spillover caused a median reduction in the probability of victimization of 1.5 percentage points (Fig. 2; mean estimate, -1.4 , credible interval, -2.5 to -0.2). The magnitude of the spillover effect is approximately 0.47 as large as the primary compliance effect. The effect size in the unadjusted DIM model (estimate, -2.2 , confidence interval, -3.6 to -0.8 , $P = 0.0024$) reflects the raw 2.2-percentage points difference in victimization outcomes. The effect size is tempered in the BART model, which attributed 69% of the difference in outcomes to the effects of intervention spillover. The attribution of some of the difference in victimization to pre-intervention differences between compliant and non-compliant peers, rather than to spillover effects, implies that differential selection into compliance among seeds is informative for characterizing the risk of peer victimization. That is, both non-compliant seeds and the peers of non-compliant seeds appear to have a higher baseline risk of victimization than their compliant counterparts. Notably, although similar in magnitude to the BART estimate, there is considerably greater uncertainty around the

spillover effect estimates (estimate, -1.2 , confidence interval, -2.7 to 0.3 , $P = 0.1178$) in the EBAL model (see Supplementary Results).

Heterogeneity of compliance and spillover effects. To assess the extent to which the compliance and spillovers effect resulted in similar reductions in the risk of victimization among all compliant seeds and compliant peers, we used the BART models to estimate the effect for each seed and peer as a function of their baseline covariate profile. This is a natural extension of BART, which allows for variation in the treatment effect across covariates^{33,35}. The primary effect of compliance on the probability of victimization ranges from -5.8 to -1.0 percentage points, at the extremes (Fig. 3a). Overall, however, our analysis shows little heterogeneity in the compliance effect, with 46.1% of units falling within ± 0.5 percentage points of the median estimated compliance effect and 74.9% of units falling within ± 1 percentage point. Similarly, the spillover effect of compliance is largely homogenous (Fig. 3b), ranging from -3.6 to -0.0 with 61.7 and 87.0% falling within ± 0.5 and ± 1 percentage points of the median spillover effect, respectively. Thus, there is little evidence to suggest that the primary compliance and spillover effects are moderated by the covariate profile of compliant seeds or peers to a degree that would warrant restructuring of the intervention for particular subgroups of assigned individuals.

Spillover effect of multiple exposures. Our results so far show that compliance reduces the probability of victimization among seeds and that compliance spillover effects reduce the probability of victimization among peers. As a further test of the spillover effects of compliance, we compared victimization outcomes among the subset of peers with exposure to either two compliant seeds ($n = 200$) or two non-compliant seeds ($n = 350$). In the two years following exposure, 12% of peers with connections to two compliant seeds and 10.6% of peers connected to two non-compliant seeds were victimized (see Supplementary Results). Thus, the victimization rate was higher among peers with a double exposure to compliant seeds than among those with a double exposure to non-compliant seeds. Additionally, the incidence of victimization was higher among peers with exposure to two seeds than among those with a single exposure (Supplementary Fig. 3). Peers with two or more exposures exhibit a different pre-intervention covariate profile in terms of age, gender and gang membership, as well as having a higher incidence of victimization and arrest among their co-arrestees than peers with a single exposure. From BART Model 1, we estimate that peers with a double-compliant exposure had a median -0.1 percentage points lower probability of victimization (mean, 0.0, credible interval, -0.6 to 1.8). However, the credible interval for the estimated effect includes zero with considerable posterior mass at both positive and negative values (Supplementary Fig. 4). We therefore have insufficient information to ascertain whether double exposure to compliant seeds increases, reduces or does not affect the probability of victimization relative to double exposure to non-compliant seeds.

Expected victimizations under the counterfactual. The parameter estimates support the idea that the violence reduction programme reduced the probability of victimization among both seeds and peers. However, the magnitude of the reduction among peers that is attributable to spillover effects is small in absolute terms: a median reduction in the probability of victimization of 1.5 percentage points (Fig. 2), with 25.7% of peers experiencing a spillover effect that is smaller in magnitude (that is, closer to zero) than one percentage point (Fig. 2). Moreover, the P value for the spillover effect estimate is > 0.05 in the EBAL model, which imposes a stronger parametric form on the relationship between victimization, compliance and the observed covariates than the BART model (see Supplementary Results). To put the magnitude of this effect into context, 174 of the 1,642 seeds who complied with the intervention were victimized

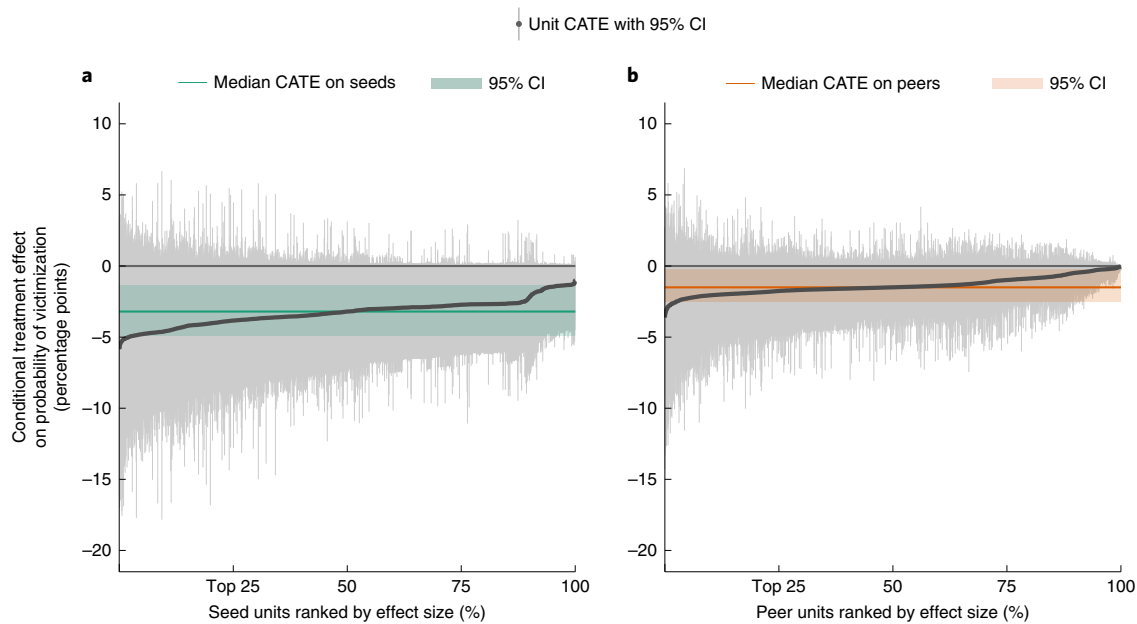


Fig. 3 | a,b, The CATE of compliance on the probability of victimization for programme seeds ($n = 2,349$) (**a**) and compliance spillover on the probability of victimization for peers ($n = 6,132$) (**b**). For each individual, the CATE is estimated as a function of their covariate profile using the fitted BART models for the compliance and spillover effects; 95% credible intervals (CI) are shown for each CATE, and the median CATE and associated 95% CI are also shown.

in the two years after compliance. In the counterfactual condition in which these seeds did not comply, our BART effect estimate implies that 227 (lower bound, 196, upper bound, 255) might have been victimized. For the compliance spillover effect, 228 of the 3,034 peers of compliant seeds were victimized in the two years after potential exposure to the programme. In the counterfactual condition in which those peers were connected to non-compliant seeds, our BART effect estimate implies that 273 (lower bound, 234, upper bound, 304) might have been victimized by gunshot violence. Our estimates therefore indicate that 53 victimizations were averted as a product of the primary effect and a further 45 through spillover effects, albeit with considerable uncertainty around these estimates. In summary, these findings imply that, by holding call-in meetings with 1,642 individuals in high-risk social networks for whom we could identify 3,034 peers, the programme contributed to approximately 98 fewer gunshot victimizations.

Discussion

Gun violence prevention, intervention and treatment programmes are increasingly directing their efforts toward the small networks and geographical areas disproportionately impacted by gunshot victimization and associated trauma. Although a networked logic drives many such programmes, most evaluations to date have analysed changes in aggregate rates of victimization or crime before and after programme implementation, paying little attention to the direct effects on individuals who are assigned to such programmes let alone the spillover effects that are theoretically deemed to be responsible for reductions in gun violence.

This study analysed a field intervention in Chicago that focused on high-risk individuals who were actively involved in ongoing disputes. Using a quasi-experimental design and data on co-arrest network ties, we evaluated the direct effect on those participating in the intervention as well as the spillover effect on their unassigned co-arrest associates. Our findings show that participation in the intervention reduced gunshot victimization by 3.2 percentage points over two years. At the same time, potential spillover reduced victimization by 1.5 percentage points among the unassigned associates of intervention participants. However, we did not detect a

spillover effect among the subset of peers who were connected to two or more compliant intervention participants. While the absence of a detectable effect may be due to the relatively small number of peers in this subset, it could also be due to a difference between individuals with exposure to two or more seeds and those with a single exposure. For example, individuals with greater seed exposure may be involved in higher-risk networks, as indicated by the greater incidence of victimization among peers with two exposures compared to those with one, and they may be less amenable to a behavioural shift in response to the intervention (see Supplementary Results).

Unlike many public health interventions³⁷, gun violence reduction programmes seldom utilize formal network metrics for the selection of participants. Consistent with several recent evaluations of networked interventions^{31,38–40}, our findings suggest that engaging the social networks of intervention participants can yield strong direct effects as well as a potential amplification of programme effects via treatment spillover. While the intervention directly treated 1,642 individuals, the structure of the co-arrest network expanded the potential reach of the programme to an additional 3,034 indirectly treated individuals—a 1.8-fold expansion of the affected population. In total, our findings imply that direct and spillover effects resulted in approximately 98 fewer gunshot victimizations over two years. However, it is worth noting that co-arrests are unlikely to capture all peers of programme participants, which could result in the total reduction being underestimated. Moreover, because it is not possible to test whether the observed covariates fully account for selection into non-compliance, we cannot rule out the possibility that observed differences in victimization between compliant and non-compliant seeds could be due to factors outside of the programme effects. Such factors could extend to the difference in victimization between compliant and non-compliant peers if peers are more likely to associate with seeds who have a similar latent risk of victimization⁴¹. Our findings nevertheless imply that further resources should be allocated to non-compliant individuals who exhibit considerably higher rates of victimization.

Programmes like the one studied here are not a panacea for gun violence. Based on the mean two-year incidence of gunshot victimization in Chicago during the period 2010–2016, the programme

effects equate to a reduction of roughly 1.5% in city-wide gun victimization. However, the results suggest that violence reduction programmes can have a substantial impact on the incidence of victimization among targeted high-risk individuals and their peers. The programme is scalable and, importantly, it minimizes traditional law enforcement responses that can have a negative impact on communities⁴², especially incarceration. Our evidence suggests that gun violence interventions might amplify programmatic effects if designed more explicitly around maximizing network diffusion^{21,43,44}. For example, street outreach¹³ or hospital intervention programmes¹¹ might use formal network analytics to guide their efforts and place workers into those parts of a network or neighbourhood experiencing acute rates of gun violence.

Field interventions aimed at high-risk social networks have become a key policy tool for reducing gunshot violence. Studying peer effects provides an avenue for enhancing the efficacy of these interventions and improving our understanding of the role of social influence in the emergence and perpetuation of gun violence and violent conflict.

Methods

Network exposure mapping. To measure exposure to the programme through social ties, we used data on 868,607 arrests in the jurisdiction of the Chicago Police Department from the period 2007–2017. We created a co-arrest network in which an edge is present between two individuals if they had been arrested together. We located each programme seed in the network and classified adjacent units as peers if those units were arrested alongside the seed at least once in the three-year period before the date that the seed was assigned to an intervention meeting. We define the exposure mapping $f(z, g)$, where $z_i = \{0, 1\}$ indicates whether a unit was assigned to the programme, and $g_i = \{0, 1\}$ indicates whether a unit has a tie to a seed^{32,45}. For units with $z_i = 1$ —that is, the seeds—we denote a compliance indicator C_i that is 1 if the unit was compliant and 0 if the unit was non-compliant. For units with $z_i = 0$, $g_i = 1$ —that is, the peers—we denote a compliance indicator C_j that is 1 if the seed to whom the peer is connected was compliant and 0 if the seed was non-compliant.

For the estimate of the effect of additional spillover exposure on victimization, we performed a subset analysis in which we identified 550 peers with exposure to two seeds, $z_i = 0$, $g_i \geq 2$. For units connected to three or more seeds, we considered only the first two exposures to maintain a suitably large n for estimation. We denote the compliance indicator C_j , which is 1 if the peer was connected to two compliant seeds and 0 if the peer was connected to two non-compliant seeds.

Identification strategy. Identifying the effect of the intervention is challenging, due to the non-randomized assignment protocol. As a strategy for overcoming this challenge, we exploited compliance and non-compliance with the programme (a recent study found that non-compliers and control units assigned by randomization had nearly identical post-assignment outcomes in a similar deterrence-based intervention in St. Louis⁴⁶) to identify the effect of compliance with the programme among assigned seeds who, by virtue of their assignment, were deemed by the administrators of the programme to have a similar baseline risk of victimization *ex ante*⁴⁷. To identify the effect of compliance with the programme, we compared two-year victimization outcomes among seeds $z_i = 1$ with $C_i = 1$ and $C_i = 0$. To identify the effect of compliance spillover, we compared two-year victimization outcomes among peers $z_i = 0$, $g_i = 1$ with $C_j = 1$ and $C_j = 0$. We denote the potential outcome $Y_i(1)$, which is the outcome that would be observed if unit i was in the compliant condition $C_i = 1$, and $Y_i(0)$ if unit i was in the non-compliant condition $C_i = 0$.

To estimate the compliance effect, we rely on the conditional ignorability assumption $Y_i(0), Y_i(1) \perp C_i | X_i$ (ref. ⁴⁸). That is, we assume independence between the potential outcomes and compliance status conditional on observed covariates X_i . We define the effect of compliance, τ_c , as the average of the conditional expectation,

$$\tau_c = E[E[Y(1)|C_i = 1, X_i] - E[Y(0)|C_i = 0, X_i]] \quad \forall z_i = 1 \quad (1)$$

For the spillover effect, we made the analogous assumption $Y_i(0), Y_i(1) \perp C_j | X_i$, that is independence between the potential outcomes and the compliance status of the seed j to whom the peer unit i is connected conditional on the peer's observed covariates X_i . We defined the spillover effect of compliance, $\tau_{s=1}$, as the average of the conditional expectation,

$$\tau_{s=1} = E[E[Y(1)|C_j = 1, X_i] - E[Y(0)|C_j = 0, X_i]] \quad \forall z_i = 0, g_i = 1 \quad (2)$$

Finally, we defined the spillover effect of double exposure to compliance $\tau_{s=2}$ as in equation (2), substituting $Z_i = 0$, $g_i \geq 2$ to restrict analysis to the subset of peers with connections to at least two seeds.

Suitability of the observed covariates. The identification strategy is valid if the observed covariates X_i provide an admissible back-door adjustment from the victimization outcome to compliance status⁴⁹. The back-door adjustment is admissible if there are no unmeasured confounders of victimization and compliance. In practice, it is not possible to demonstrate this admissibility. As our outcome is gunshot victimization, we included several covariates related to gunshot violence⁵⁰ and violence more generally, all measured pre-intervention. These include gunshot victimizations; arrests on violence-related charges, including first- and second-degree assault; and arrests on weapons-related charges, including carrying weapons without a permit and ownership of illegal firearms. To account for the risk level in the co-arrest network surrounding each unit, we included counts of victimizations, arrests, arrests on violence-related charges and arrests on weapons-related charges among adjacent units. To account for the connectivity of each unit, we also included degree (that is, the number of co-arrestees). Finally, we included age, race, gender and gang status, which are related to the incidence of gunshot victimization^{51–54}. We assumed that this broad array of covariates adequately accounts for confounding due to selection into the compliant or non-compliant conditions. However, we cannot rule out unobserved factors that could bias the estimates, although our adjustment may partly reduce bias due to any unmeasured confounders that are correlated with the observed covariates^{55,56}.

Estimation strategy. To estimate the compliance effect τ_c and spillover effect of compliance $\tau_{s=1}$ we used both BART and DIM.

BART is a sum-of-trees model that allows a flexible relationship between the victimization outcome Y_i , compliance indicator C_i (or C_j in the case of spillover) and observed covariates X_i . We use this non-parametric approach to avoid imposing a structural form on the relationship between the compliance indicator and the observed covariates, avoiding researcher-imposed choices regarding the ways in which the covariates might be associated with selection into compliance and, instead, allowing automatic detection of interactions and nonlinearities in this relationship³⁵. We fit the BART model using the `dbarts` R package³⁴ with the default settings (`ntrees = 200`, $\alpha = 0.95$, $\beta = 2$), 1,000 burn-in Markov chain Monte Carlo iterations and 5,000 posterior samples.

We contrast this approach with a simple DIM,

$$Y_i = \alpha + C_i \quad \forall Z_i = 1 \quad (3)$$

where α is an intercept and C_i is the compliance indicator. Similarly, for the spillover effect we estimate,

$$Y_i = \alpha + C_j \quad \forall Z_i = 0, g_i = 1 \quad (4)$$

where C_j is the indicator for the compliance status of the seed to whom the peer is connected. This model simply compares the average probability of victimization within the compliant and non-compliant conditions without adjustment for confounding. By contrasting the results between BART and the DIM model, we are able to gauge the extent to which the relationship between the victimization outcomes Y_i and compliance status C_i (C_j for spillover) is moderated by the covariates X_i . Reported P values for the compliance and spillover DIM estimates are based on two-tailed tests.

To further assess the effect estimates, we compared the results from BART to those of a model that weights units based on covariate balancing propensity scores. See Supplementary Information for further details.

Estimating heterogeneous effects. The BART model naturally detects interactions between the compliance indicator and observed covariates³³. To estimate the heterogeneity of the compliance and spillover effects, we used the fitted BART model to estimate the conditional average treatment effect (CATE) of compliance on each unit as a function of that unit's covariate profile. For the main effect we duplicated the seed covariate data into two $N \times K$ matrices, where N is the number of seeds and K is the number of covariates. We set the compliance indicator to 1 in the first matrix and 0 in the second. We then took 1,000 posterior draws for each matrix from the fitted model and subtracted the second matrix from the first. The CATE is calculated by taking the mean of the rows, while the 95% uncertainty bounds are calculated by taking the 0.025 and 0.975 quantiles³⁵. We repeated this process for the spillover effect, substituting in the peer covariate data and the fitted spillover BART model.

Reporting Summary. Further information on research design is available in the Nature Research Reporting Summary linked to this article.

Data availability

The original data used in this study were provided to the corresponding author as part of a data-sharing agreement with the City of Chicago and the Chicago Police Department, and are prohibited from being shared directly. De-identified replication data generated and analysed in this study are available from the corresponding author upon request.

Code availability

All analyses were carried out in R. Code for reproducing the results of this study is available from the corresponding author upon request.

Received: 19 October 2018; Accepted: 12 July 2019;

Published online: 19 August 2019

References

- Wintemute, G. J. The epidemiology of firearm violence in the twenty-first century United States. *Annu. Rev. Public Health* **36**, 5–19 (2015).
- Web-based injury statistics query and reporting system. *National Center for Injury Prevention and Control* <http://www.cdc.gov/injury/wisqars/index.html> (2019).
- Peterson, R. D. & Krivo, L. J. *Divergent Social Worlds: Neighborhood Crime and the Racial-Spatial Divide* (Russell Sage, 2010).
- Harper, S., Lynch, J., Burris, S. & Davey Smith, G. Trends in the black-white life expectancy gap in the United States, 1983–2003. *JAMA* **297**, 1224–1232 (2007).
- Sharkey, P. The acute effect of local homicides on children's cognitive performance. *Proc. Natl Acad. Sci. USA* **107**, 11733–11738 (2010).
- Tracy, M., Braga, A. A. & Papachristos, A. V. The transmission of gun and other weapon-involved violence within social networks. *Epidemiol. Rev.* **38**, 70–86 (2016).
- Papachristos, A. V., Wildeman, C. & Roberto, E. Tragic, but not random: the social contagion of nonfatal gunshot injuries. *Soc. Sci. Med.* **125**, 139–150 (2015).
- Papachristos, A. V., Braga, A. A. & Hureau, D. Social networks and the risk of gunshot injury. *J. Urban Health* **89**, 992–1003 (2012).
- Green, B., Thibaut, H. & Papachristos, A. V. Modeling contagion through social networks to explain and predict gunshot violence in Chicago, 2006 to 2014. *JAMA Intern. Med.* **177**, 326–333 (2017).
- Cooper, C., Eslinger, D. M. & Stolley, P. D. Hospital-based violence intervention programs work. *J. Trauma Acute Care Surg.* **61**, 534–540 (2006).
- Purtle, J., Rich, J. A., Fein, J. A., James, T. & Corbin, T. J. Hospital-based violence prevention: progress and opportunities. *Ann. Intern. Med.* **163**, 715–717 (2015).
- Butts, J. A., Roman, C. G., Bostwick, L. & Porter, J. R. Cure violence: a public health model to reduce gun violence. *Annu. Rev. Public Health* **36**, 39–53 (2015).
- Whitehill, J. M., Webster, D. W., Frattaroli, S. & Parker, E. M. Interrupting violence: how the ceasefire program prevents imminent gun violence through conflict mediation. *J. Urban Health* **91**, 84–95 (1994).
- Crandall, V. & Wong, S. L. *Group Violence Reduction Strategy: Call-in Preparation and Execution* (The Office of Community Oriented Policing Strategies, 2012).
- Braga, A. A. & Weisburd, D. The effects of focused deterrence strategies on crime: a systematic review and meta-analysis of the empirical evidence. *J. Res. Crime Delinq.* **49**, 323–358 (2012).
- Braga, A. A., Papachristos, A. V. & Hureau, D. The effects of hot spots policing on crime: an updated systematic review and meta-analysis. *Justice Q.* **31**, 633–663 (2014).
- Braga, A. A., Weisburd, D. & Turchan, B. Focused deterrence strategies and crime control: an updated systematic review and meta-analysis of the empirical evidence. *Criminol. Public Policy* **17**, 205–250 (2018).
- Heckman, J. J. The scientific model of causality. *Sociol. Methodol.* **35**, 1–97 (2006).
- Durlauf, S. N., Navarro, S. & Rivers, D. A. Understanding aggregate crime regressions. *J. Econ.* **158**, 306–317 (2010).
- Gravel, J. & Tita, G. E. With great methods comes great responsibilities. *Criminol. Public Policy* **14**, 559–572 (2015).
- Valente, T. W. Network interventions. *Science* **337**, 49–53 (2012).
- Proestakis, A. et al. Network interventions for changing physical activity behaviour in preadolescents. *Nat. Hum. Behav.* **2**, 778–787 (2018).
- Kennedy, D. M., Braga, A. A. & Piehl, A. M. The (un)known universe: mapping gangs and gang violence in Boston. in *Crime Mapping and Crime Prevention* (ed. Weisburd, D. & McEwan, T.) 219–262 (Criminal Justice Press, 1997).
- Papachristos, A. V. & Kirk, D. S. Changing the street dynamic: evaluating Chicago's group violence reduction strategy. *Criminol. Public Policy* **14**, 525–558 (2015).
- National Network for Safe Communities. *Group Violence Intervention: An Implementation Guide* (US Department of Justice, Office of Community Oriented Policing Services, 2016).
- Broockman, D. & Kalla, J. Durably reducing transphobia: a field experiment on door-to-door canvassing. *Science* **352**, 220–224 (2016).
- Clarke, R. V. & Weisburd, D. Diffusion of crime control benefits: observations on the reverse of displacement. *Crime Prev. Stud.* **2**, 165–184 (1994).
- Kennedy, D. M., Piehl, A. M. & Braga, A. A. Youth violence in Boston: gun markets, serious youth offenders, and a use-reduction strategy. *Law Contemp. Probl.* **59**, 147–196 (1996).
- Braga, A. A. & Weisburd, D. Focused deterrence and the prevention of violent gun injuries: practice, theoretical principles, and scientific evidence. *Annu. Rev. Public Health* **36**, 55–68 (2015).
- Charette, Y. & Papachristos, A. V. The network dynamics of co-offending careers. *Soc. Netw.* **51**, 3–13 (2017).
- Paluck, E. L., Shepherd, H. & Aronow, P. M. Changing climates of conflict: a social network experiment in 56 schools. *Proc. Natl Acad. Sci. USA* **113**, 566–571 (2016).
- Aronow, P. M. & Samii, C. Estimating average causal effects under general interference, with application to a social network experiment. *Ann. Appl. Stat.* **11**, 1912–1947 (2017).
- Hill, J. L. Bayesian nonparametric modeling for causal inference. *J. Comput. Graph. Stat.* **20**, 217–240 (2011).
- Chipman, H. A., George, E. I. & McCulloch, R. E. Bart: Bayesian additive regression trees. *Ann. Appl. Stat.* **4**, 266–298 (2010).
- Green, D. P. & Kern, H. L. Modeling heterogeneous treatment effects in survey experiments with Bayesian additive regression trees. *Public Opin. Q.* **76**, 491–511 (2012).
- Hainmueller, J. Entropy balancing for causal effects: a multivariate reweighting method to produce balanced samples in observational studies. *Polit. Anal.* **20**, 25–46 (2012).
- Palinkas, L. A. et al. Influence network linkages across implementation strategy conditions in a randomized controlled trial of two strategies for scaling up evidence-based practices in public youth-serving systems. *Implement. Sci.* **8**, 133 (2013).
- Christakis, N. A. & Fowler, J. H. Social network sensors for early detection of contagious outbreaks. *PLoS One* **5**, e12948 (2010).
- Centola, D. The spread of behavior in an online social network experiment. *Science* **329**, 1194–1197 (2010).
- Kim, D. A. et al. Social network targeting to maximise population behavior change: a cluster randomised controlled trial. *Lancet* **386**, 145–153 (2015).
- Shalizi, C. R. & Thomas, A. C. Homophily and contagion are generically confounded in observational social network studies. *Sociol. Methods Res.* **40**, 211–239 (2011).
- Legewie, J. & Fagan, J. Aggressive policing and the educational performance of minority youth. *Am. Sociol. Rev.* **84**, 220–247 (2019).
- Valente, T. W. Social network thresholds in the diffusion of innovations. *Soc. Netw.* **18**, 69–89 (1996).
- Rogers, E. M. Diffusion of preventative innovations. *Addict. Behav.* **27**, 989–993 (2002).
- Forastiere, L., Airoidi, E. M. & Mealli, F. Identification and estimation of treatment and interference effects in observational studies on networks. Preprint at *arXiv* <https://arxiv.org/abs/1609.06245> (2018).
- Hamilton, B., Rosenfeld, R. & Levin, A. Opting out of treatment: self-selection bias in a randomized controlled study of a focused deterrence notification meeting. *J. Exp. Criminol.* **14**, 1–17 (2018).
- Rosenbaum, P. R. Choice as an alternative to control in observational studies. *Stat. Sci.* **14**, 259–304 (1999).
- Rubin, D. B. Bayesian inference for causal effects: the role of randomization. *Ann. Stat.* **6**, 34–58 (1978).
- Pearl, J. Causal inference in statistics: an overview. *Stat. Surv.* **3**, 96–146 (2009).
- Steinman, K. J. & Zimmerman, M. A. Episodic and persistent gun-carrying among urban African-American adolescents. *J. Adolesc. Health* **35**, 356–364 (2003).
- Cook, P. J. & Laub, J. H. After the epidemic: recent trends in youth violence in the United States. *Crime Justice* **29**, 1–37 (2002).
- Peterson, R. D. & Krivo, L. J. Macrostructural analyses of race, ethnicity, and violent crime: recent lessons and new directions for research. *Annu. Rev. Sociol.* **31**, 331–356 (2005).
- Jones-Webb, R. & Wall, M. Neighborhood racial/ethnic concentration, social disadvantage, and homicide risk: an ecological analysis of 10 U.S. cities. *J. Urban Health* **8**, 662–676 (2008).
- Papachristos, A. V., Braga, A. A., Piza, E. & Grossman, L. S. The company you keep? The spillover effects of gang membership on individual gunshot victimization in a co-offending network. *Criminology* **53**, 624–649 (2015).
- Fewell, Z., Davey Smith, G. & Sterne, J. A. C. The impact of residual and unmeasured confounding in epidemiologic studies: a simulation study. *Am. J. Epidemiol.* **166**, 646–655 (2007).
- Greenwood, R. H. H. et al. Sensitivity analysis for the effects of multiple unmeasured confounders. *Ann. Epidemiol.* **26**, 601–605 (2016).

Acknowledgements

This research was supported by a CAREER award (No. SES-1151449) from the Sociology and Law and Social Science Programs at the National Science Foundation. We thank

Y. Charette and D. Kirk for providing valuable feedback. The funder had no role in the conceptualization, design, data collection, analysis, decision to publish or preparation of the manuscript.

Author contributions

G.W. and A.V.P. designed research. A.V.P. obtained data. G.W. performed research and analysed data. G.W. and A.V.P. wrote the paper.

Competing interests

The authors declare no competing interests.

Additional information

Supplementary information is available for this paper at <https://doi.org/10.1038/s41562-019-0688-1>.

Reprints and permissions information is available at www.nature.com/reprints.

Correspondence and requests for materials should be addressed to A.V.P.

Peer review information: Primary Handling Editor: Aisha Bradshaw.

Publisher's note: Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

© The Author(s), under exclusive licence to Springer Nature Limited 2019

Reporting Summary

Nature Research wishes to improve the reproducibility of the work that we publish. This form provides structure for consistency and transparency in reporting. For further information on Nature Research policies, see [Authors & Referees](#) and the [Editorial Policy Checklist](#).

Statistics

For all statistical analyses, confirm that the following items are present in the figure legend, table legend, main text, or Methods section.

n/a Confirmed

- | | | |
|-------------------------------------|-------------------------------------|--|
| <input type="checkbox"/> | <input checked="" type="checkbox"/> | The exact sample size (n) for each experimental group/condition, given as a discrete number and unit of measurement |
| <input type="checkbox"/> | <input checked="" type="checkbox"/> | A statement on whether measurements were taken from distinct samples or whether the same sample was measured repeatedly |
| <input type="checkbox"/> | <input checked="" type="checkbox"/> | The statistical test(s) used AND whether they are one- or two-sided
<i>Only common tests should be described solely by name; describe more complex techniques in the Methods section.</i> |
| <input type="checkbox"/> | <input checked="" type="checkbox"/> | A description of all covariates tested |
| <input type="checkbox"/> | <input checked="" type="checkbox"/> | A description of any assumptions or corrections, such as tests of normality and adjustment for multiple comparisons |
| <input type="checkbox"/> | <input checked="" type="checkbox"/> | A full description of the statistical parameters including central tendency (e.g. means) or other basic estimates (e.g. regression coefficient) AND variation (e.g. standard deviation) or associated estimates of uncertainty (e.g. confidence intervals) |
| <input type="checkbox"/> | <input checked="" type="checkbox"/> | For null hypothesis testing, the test statistic (e.g. F , t , r) with confidence intervals, effect sizes, degrees of freedom and P value noted
<i>Give P values as exact values whenever suitable.</i> |
| <input type="checkbox"/> | <input checked="" type="checkbox"/> | For Bayesian analysis, information on the choice of priors and Markov chain Monte Carlo settings |
| <input checked="" type="checkbox"/> | <input type="checkbox"/> | For hierarchical and complex designs, identification of the appropriate level for tests and full reporting of outcomes |
| <input checked="" type="checkbox"/> | <input type="checkbox"/> | Estimates of effect sizes (e.g. Cohen's d , Pearson's r), indicating how they were calculated |

Our web collection on [statistics for biologists](#) contains articles on many of the points above.

Software and code

Policy information about [availability of computer code](#)

Data collection

N/A

Data analysis

All data wrangling and analysis conducted in R. Primary packages used: broom, cobalt, dbarts, ebal, igraph, tidygraph, tidyverse, twang, weightit.

For manuscripts utilizing custom algorithms or software that are central to the research but not yet described in published literature, software must be made available to editors/reviewers. We strongly encourage code deposition in a community repository (e.g. GitHub). See the Nature Research [guidelines for submitting code & software](#) for further information.

Data

Policy information about [availability of data](#)

All manuscripts must include a [data availability statement](#). This statement should provide the following information, where applicable:

- Accession codes, unique identifiers, or web links for publicly available datasets
- A list of figures that have associated raw data
- A description of any restrictions on data availability

The original data used in this study were provided to the corresponding author as part of a data-sharing agreement with the City of Chicago and the Chicago Police Department and are prohibited from direct sharing. De-identified replication data generated and analyzed in this study are available from the corresponding author upon request.

Field-specific reporting

Please select the one below that is the best fit for your research. If you are not sure, read the appropriate sections before making your selection.

Life sciences Behavioural & social sciences Ecological, evolutionary & environmental sciences

For a reference copy of the document with all sections, see nature.com/documents/nr-reporting-summary-flat.pdf

Behavioural & social sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description	This is a quantitative quasi-experimental study. We compare gunshot victimization outcomes for units who complied with an intervention against non-compliant units. We also compare victimization outcomes among the network peers of compliant units against the peers of non-compliant units.
Research sample	The research sample is individuals at high-risk of victimization in the jurisdiction of the Chicago Police Department (CPD). The intervention aimed to reduce victimization among such individuals. The intervention effects are not generalizable to the general population. The sample represents particularly high-risk individuals in Chicago. The differences between the sample and the broader population of individuals with arrest records in Chicago is summarized in the Supplementary Results.
Sampling strategy	The sampling procedure is a community-led partnership with law enforcement to identify individuals at high-risk of victimization. The authors did not design or participate in the sampling strategy.
Data collection	Three primary data sources are used: CPD arrest records, CPD gunshot victimization records, and intervention assignment and compliance data. The arrest records and gunshot victimization records are routinely collected by the CPD and were supplied to the researchers directly by the CPD. The assignment data were collected by the intervention team and supplied directly to the researchers. Being routinely collected by the CPD, the outcome data (victimization) is not influenced by the research hypothesis.
Timing	The arrest data covers the period August 2007 to May 2017. The victimization data covers the period August 2007 to November 2018. Assignment and compliance data collected August 2010 to June 2016.
Data exclusions	No data were excluded from the analysis.
Non-participation	Non-compliant units are included in our research design.
Randomization	Units were not randomly assigned. Participants were assigned to the program based on an evaluation of their risk of victimization. The risk evaluation was not conducted by the authors of this study.

Reporting for specific materials, systems and methods

We require information from authors about some types of materials, experimental systems and methods used in many studies. Here, indicate whether each material, system or method listed is relevant to your study. If you are not sure if a list item applies to your research, read the appropriate section before selecting a response.

Materials & experimental systems

n/a	Involved in the study
<input checked="" type="checkbox"/>	<input type="checkbox"/> Antibodies
<input checked="" type="checkbox"/>	<input type="checkbox"/> Eukaryotic cell lines
<input checked="" type="checkbox"/>	<input type="checkbox"/> Palaeontology
<input checked="" type="checkbox"/>	<input type="checkbox"/> Animals and other organisms
<input type="checkbox"/>	<input checked="" type="checkbox"/> Human research participants
<input checked="" type="checkbox"/>	<input type="checkbox"/> Clinical data

Methods

n/a	Involved in the study
<input checked="" type="checkbox"/>	<input type="checkbox"/> ChIP-seq
<input checked="" type="checkbox"/>	<input type="checkbox"/> Flow cytometry
<input checked="" type="checkbox"/>	<input type="checkbox"/> MRI-based neuroimaging

Human research participants

Policy information about [studies involving human research participants](#)

Population characteristics	See above.
Recruitment	Participants were invited to an intervention by a collaborative group who determined the participants to be at high-risk of victimization. We compared units who complied (attended the intervention) against units who did not comply. There is a possibility for selection bias if compliance or non-compliance is associated with the risk of victimization. Our estimation strategy was designed to mitigate this selection bias. We adjusted for a broad array of covariates which may confound victimization and compliance. We re-weighted units to provide an estimate of the effects conditional on the observed covariates. There is the

possibility for remaining selection bias if there are unobserved covariates which confound victimization and compliance, particularly if these are uncorrelated with the observed covariates adjusted for in the study. This is untestable using available data. We discuss this in more detail in the manuscript and Supplementary Information.

Ethics oversight

Yale University IRB and Northwestern University IRB.

Note that full information on the approval of the study protocol must also be provided in the manuscript.