JAMA Internal Medicine | Original Investigation | FIREARM VIOLENCE

Modeling Contagion Through Social Networks to Explain and Predict Gunshot Violence in Chicago, 2006 to 2014

Ben Green, MSc; Thibaut Horel, MSc; Andrew V. Papachristos, PhD

IMPORTANCE Every day in the United States, more than 200 people are murdered or assaulted with a firearm. Little research has considered the role of interpersonal ties in the pathways through which gun violence spreads.

OBJECTIVE To evaluate the extent to which the people who will become subjects of gun violence can be predicted by modeling gun violence as an epidemic that is transmitted between individuals through social interactions.

DESIGN, SETTING, AND PARTICIPANTS This study was an epidemiological analysis of a social network of individuals who were arrested during an 8-year period in Chicago, Illinois, with connections between people who were arrested together for the same offense. Modeling of the spread of gunshot violence over the network was assessed using a probabilistic contagion model that assumed individuals were subject to risks associated with being arrested together, in addition to demographic factors, such as age, sex, and neighborhood residence. Participants represented a network of 138 163 individuals who were arrested between January 1, 2006, and March 31, 2014 (29.9% of all individuals arrested in Chicago during this period), 9773 of whom were subjects of gun violence. Individuals were on average 27 years old at the midpoint of the study, predominantly male (82.0%) and black (75.6%), and often members of a gang (26.2%).

MAIN OUTCOMES AND MEASURES Explanation and prediction of becoming a subject of gun violence (fatal or nonfatal) using epidemic models based on person-to-person transmission through a social network.

RESULTS Social contagion accounted for 63.1% of the 11123 gunshot violence episodes; subjects of gun violence were shot on average 125 days after their infector (the person most responsible for exposing the subject to gunshot violence). Some subjects of gun violence were shot more than once. Models based on both social contagion and demographics performed best; when determining the 1.0% of people (n = 1382) considered at highest risk to be shot each day, the combined model identified 728 subjects of gun violence (6.5%) compared with 475 subjects of gun violence (4.3%) for the demographics model (53.3% increase) and 589 subjects of gun violence (5.3%) for the social contagion model (23.6% increase).

CONCLUSIONS AND RELEVANCE Gunshot violence follows an epidemic-like process of social contagion that is transmitted through networks of people by social interactions. Violence prevention efforts that account for social contagion, in addition to demographics, have the potential to prevent more shootings than efforts that focus on only demographics.

JAMA Intern Med. 2017;177(3):326-333. doi:10.1001/jamainternmed.2016.8245 Published online January 3, 2017. Editorial page 316 and Invited Commentary page 333

+ Supplemental content at jamainternalmedicine.com

Author Affiliations: John A. Paulson School of Engineering and Applied Sciences, Harvard University, Cambridge, Massachusetts (Green, Horel); Department of Sociology, Yale University, New Haven, Connecticut (Papachristos); Yale Institute for Network Science, Yale University, New Haven, Connecticut (Papachristos).

Corresponding Author: Andrew V. Papachristos, PhD, Department of Sociology, Yale University, 493 College St, Room 201, New Haven, CT 06520 (andrew.papachristos@yale .edu).

jamainternalmedicine.com

326

n 2014, a total of 10 945 people in the United States were murdered with a firearm, and approximately 65 106 others were injured in nonfatal gun assaults (an average of more than 200 fatal and nonfatal subjects of gun violence per day).¹ Although mass shootings are often the focus of public attention, most gun murders and assaults occur in everyday incidents involving a small number of people (typically 2 individuals).² Furthermore, gun violence tends to concentrate within socially and economically disadvantaged minority urban communities, where rates of gunshot injuries far exceed the national average^{3,4} and where young black men experience rates of gun homicide 10 times higher than their white counterparts.²

The media, politicians, and academics alike often describe gun violence in the United States as an "epidemic,"^{2,5-9} implying concern over its alarmingly high levels, as well as the possibility of its spread. Although gun violence's stubborn persistence in certain communities might be more accurately described as an endemic,⁶ the public emphasis on epidemics has inspired research on the mechanisms through which violence might be transmitted.9-11 The most common approach measures the spatial diffusion of gun violence from neighborhood to neighborhood.^{3,9,11,12} Although this spatial approach often discusses interpersonal relationships related to gang activity^{9,13} or drug markets¹⁴ as the drivers behind the diffusion of gun violence, the statistical models presume that violence might be conceptualized as an airborne pathogen (eg, influenza) moving between neighborhoods, which can be "caught" by inhabiting locations with high incidence rates.

However, recent thinking suggests that many of the processes that we attribute to geography might occur in part because of the interpersonal ties underlying social networks¹⁵ (see the Glossary in the eText in the Supplement for definitions of social network and other technical terms used in this article). Research on gun violence in Chicago, Illinois, Boston, Massachusetts, and Newark, New Jersey, has found that subjects of gun violence are concentrated within networks, along with cross-sectional evidence that such concentration is related to social contagion (ie, the spread of beliefs, attitudes, and behaviors through social interactions).^{10,16-18} Furthermore, social networks are fundamental in diffusion processes related to diverse areas, such as behaviors,¹⁹ opinions,^{20,21} human immunodeficiency virus (HIV),²² obesity,²³ and depression.²⁴ Taken together, the results of these studies suggest that the diffusion of gun violence might occur through person-to-person interactions, in a process akin to the epidemiological transmission of a blood-borne pathogen (eg, HIV). Contagion via social ties, then, may be a critical mechanism in explaining why neighborhoods matter when modeling the diffusion of crime and, perhaps more important, why certain individuals become subjects of gun violence while others exposed to the same high-risk environments do not.

To study the role of social influence in gun violence, we examined a particular interaction between individuals, namely, being arrested together for the same offense, a behavior known as co-offending. Co-offending typically occurs between people who share strong preexisting social ties²⁵ and is driven by social processes that amplify risky behaviors (criminal or delinquent acts that might lead to arrest, including violent episodes and offending).²⁵⁻²⁹ Like other social behaviors, such as needle

Key Points

Question Does gun violence spread over social networks through a process of social contagion?

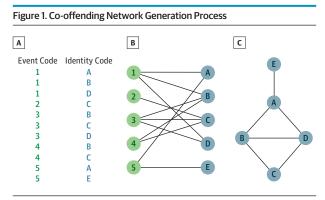
Findings An epidemiological analysis of a network of 138 163 individuals in Chicago, Illinois, determined that social contagion was responsible for 63.1% of the 11123 gunshot violence episodes that occurred between 2006 and 2014. Models incorporating social contagion and demographics (eg, age, sex, and neighborhood residence) predicted future gunshot subjects better than models based on social contagion or demographics alone.

Meaning Violence prevention efforts that account for social contagion, in addition to demographics, have the potential to prevent more shootings than efforts that focus only on demographics.

sharing³⁰ and sex,^{22,31} co-offending may reveal patterns of social interactions that influence how gun violence spreads.^{25,27,32-34} We postulated that a person becomes exposed to gun violence through social interactions with previous subjects of gun violence: someone who has been shot may be more likely to be embedded in the networks and environments in which guns are present and gun violence is likely to erupt. Therefore, associating with subjects of gun violence, and specifically co-engaging in risky behaviors with them, may expose individuals to these same behaviors, situations, and people that in turn increase the probability of becoming a subject of gun violence.

Our study directly assessed the efficacy of treating the diffusion of gunshot violence as an epidemiological process that spreads through social networks. Our central hypothesis was that, when someone in your network becomes a subject of gun violence, your risk of becoming a subject of gun violence temporarily increases. We hypothesized that predictive models incorporating social contagion would outperform models considering only individual and ecological risk factors in predicting future gunshot subjects. Modeling the precise social dynamics of gun violence could represent an important advance in treating gun violence as a public health epidemic. By identifying high-risk individuals and transmission pathways that might not be detected by other means, a contagion-based approach could detect strategic points of intervention that would enable measures to proactively reduce the trauma associated with gun violence rather than just react to past incidents. Most important, such a contagion-based approach is centered on subjects of gun violence and, as such, has the potential to move the larger public dialogue on gun violence away from efforts that rest largely on geographic or group-based policing efforts that tend to disproportionately affect disadvantaged minority communities.

We tested our hypothesis in Chicago, a city whose welldocumented patterns of gun violence are emblematic of the epidemic described above and whose rates of gun violence are more than 3 times the national average (eText and eFigures 1, 2, and 3 in the Supplement).^{15,35-39} Although Chicago does not have the highest urban per capita homicide rate, the city has a long history of violence and consistently tallies a greater number of homicides than any other city in the United States.⁴⁰



A, Example of raw data and its structure, in which event codes mark specific arrest events and identity codes represent unique individuals. Each entry represents a single individual arrested in a specific incident. B, Bipartite (ie, 2 mode) network between offenses (green) and people (blue), generated by using the data from A as an edge list (in which each row represents a pair of nodes that are connected by an edge). C, Person-to-person (ie, 1 mode) co-offending network, generated by performing a bipartite projection on the network from B. Nodes represent unique offenders, and edges connect offenders who were arrested for the same incident. Note that the network shown in this panel is unweighted, meaning that every edge has identical weight, even for pairs who were arrested together multiple times.

As in other major US cities, violent gun crime in Chicago is intensely concentrated in a small number of socially and economically disadvantaged neighborhoods (where homicide rates can be upward of 75 per 100 000 people).^{36,37,41} Furthermore, gun violence is concentrated in small social networks: a recent study¹⁰ by one of us (A.V.P.) of nonfatal gunshot violence episodes in Chicago from 2006 to 2014 found that more than 70% of all subjects of gun violence could be located in networks containing less than 5% of the city's population. The present study examines the extent to which being shot in Chicago might be explained as a process of epidemiological transmission between individuals in these networks.

Methods

Data

We examined all recorded fatal and nonfatal gunshot injuries in Chicago from 2006 to 2014 among the population of individuals arrested during this period. Data are from 2 different sources provided by the Chicago Police Department through a nondisclosure agreement (and approved by the Yale Institutional Review Board). These data were considered exempt by the institutional review board because they were secondary deidentified information provided for the study, and informed consent was not necessary. The first source was all 1189225 arrests recorded by the police between January 1, 2006, and March 31, 2014, involving 462 516 people (for comparison, the adult population of Chicago totals approximately 2.1 million). Arrest data are recorded at the incident level and contain social and demographic information on each reported individual, including birth date, race, sex, and gang membership (as identified by the police).

The second source was detailed records for all 16 399 gunshot violence episodes recorded by the police during the

same period, excluding suicides, accidents, and shootings that occurred during legal interventions (ie, shootings involving law enforcement personnel). These records consist of 13 877 nonfatal and 2522 fatal shootings, affecting 14 695 people; 1498 people were shot on more than 1 occasion. Among all shooting subjects, 90.2% were arrested during the study period and could be identified in the arrest data.

Co-offending Network

Figure 1 shows how the co-offending network was created. We generated a social network from the data by identifying all unique individuals arrested during the study period and connecting them via "edges," that is, a relationship between pairs of individuals defined by being arrested together for the same offense (a behavior known as co-offending) at least once during the study period (section 2 of the eMethods in the Supplement). This network contained 462 516 individuals, 467 506 edges, and 13 252 subjects of gun violence. Because co-offending typically occurs between individuals who share preexisting social ties,²⁵ co-arrests represent an association between 2 people but not the date at which this relationship formed. Therefore, we developed a static network with edges between every pair of individuals who had been arrested together at any time during the study period.

We restricted our analysis to the network's largest connected component, which contained 29.9% of all arrested individuals (n = 138 163) and 89.3% of all the co-offending edges (n = 417 635). Consistent with previous research on the concentration of gun violence within co-offending networks,¹⁰ the largest connected component contained 74.5% of gunshot violence episodes of arrested individuals (11 123 violence episodes, affecting 9773 people). We henceforth refer to this component as the network.

Social Contagion Model

We modeled the contagion of violence over the network using a stochastic model in which the probability of future shootings depended on the history of past shootings.^{11,42-45} Individuals are susceptible to gunshot violence through the following 2 means: (1) social contagion, reflecting the increased probability to be shot immediately after a person with whom one associates has been shot, and (2) a seasonal factor that reflects the persistent rate of violence episodes within the network. The model expressed the social contagion component of susceptibility via 2 factors, namely, time and network structure (section 4 of the eMethods and eFigure 4 in the Supplement). Consistent with previous models and epidemiological research, we assumed that gun violence is most likely to spread immediately after another shooting⁴⁴⁻⁴⁶ and between people who are closely linked in the network.^{47,48} Regarding this latter point, we set the influence of contagion to weaken farther away from the source according to the inverse square of network distance and to disappear beyond 3 degrees of separation (ie, >3 edges away in the network).^{10,23}

Using variables calibrated on the observed data, our model calculated each person's exposure to gun violence based on the aggregate influence of social contagion and seasonal facTable. Characteristics of the 138 163 Individuals Arrested in Chicago Between 2006 and 2014 and in the Largest Connected Component of the Network^a

Variable	Largest Connected Component	Subjects of Gun Violence	Not Subjects of Gun Violence
Demographics			
No. of people	138 163	9773	128 390
Age at study midpoint, y	27.5	23.2	27.0
Male, %	82.0	97.0	80.9
Black race/ethnicity, %	75.6	79.8	75.3
White Hispanic race/ethnicity, %	23.3	19.5	23.6
Gang member, %	26.2	52.3	24.3
Network Characteristics			
No. of co-offenders (degree centrality)	6.1	10.2	5.7
Neighbors who are subjects of gun violence (first degree), %	10.4	17.9	9.8
Neighbors who are subjects of gun violence (first and second degree), %	11.1	15.9	10.7
Neighbors who are subjects of gun violence (first, second, and third degree), %	11.8	14.9	11.6

^a The mean characteristics are listed for all individuals arrested in Chicago between 2006 and 2014 and located in the largest connected component, comparing demographic and network statistics between the subjects of gun violence and those who were not. All comparisons between gunshot subjects and those who were not were significant at *P* < .001 (*P* values were calculated using the Welch 2-sample t test).

tors (section 4 of the eMethods and eFigures 5 and 6 in the **Supplement**). For each gunshot subject who was influenced primarily by contagion, we identified which peer (the infector) was most responsible for causing him or her to become infected (ie, a subject of gun violence). We then connected these infections from infector to subject of gun violence to trace cascades of gunshot violence episodes through the network (ie, chains in which one person becomes infected, exposing his or her associates, who then may become infected and spread the infection to their associates, etc) (section 4 of the eMethods in the **Supplement**). It is important to note that the infector is not assumed to be the person who shoots the subject of gun violence but rather the one who exposes him or her to the risk of gun violence.

Model Evaluation for Predicting Subjects of Gun Violence

An important application of modeling gun violence is to identify who might be shot in the future: predicting gunshot subjects might provide information that can be used for intervention and violence prevention efforts, especially if we could precisely identify a small population that faces the most immediate risk. Therefore, we applied our model to predict who would be shot. We compared the model described above (referred to henceforth as the social contagion model) with 2 others. The first model was a demographics model that assumes becoming a subject of gun violence is determined by traits, such as age, sex, and neighborhood residence, and correspondingly predicts future subjects of gun violence based on demographic similarities to previous subjects of gun violence. This model approximated traditional risk factor theories of gun violence. The second model combined the social contagion and demographics models in a weighted average (with weights found by optimizing over all possible linear combinations) to account for both potential explanations of gun violence.

For every day of the study period, based on the data up to that day, we calculated the risk of every person in the network to be shot according to each model and selected the highest-risk individuals identified by each (section 5 of the eMethods in the Supplement). We defined 3 high-risk populations as those people identified with the top 0.1% (n = 138), 0.5% (n = 691), and 1.0% (n = 1382) of risk to be shot on a given day. Although larger populations of high-risk individuals could potentially include more subjects of gun violence, larger populations could also include too many people to be reasonably suited for targeted interventions. We evaluated and ranked our models based on the number of subjects of gun violence they identified within these high-risk populations. To evaluate the reliability of the models, we performed tests on simulated data for which we knew the true variable values and showed that for the duration of our study period the method could accurately estimate the variables within 12.7% (section 4 of the eMethods and eFigure 6 in the Supplement).

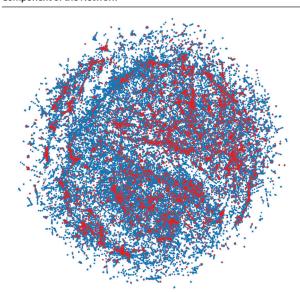
Results

Characteristics of the Network

The Table lists characteristics of the 138 163 individuals in the network. Figure 2 shows a graphical representation of the network, illustrating the relative locations of subjects of gun violence and those who were not. Individuals were on average 27 years old at the midpoint (in 2010) of the study and predominantly male (82.0%) and black (75.6%). According to police estimates, 26.2% were members of street gangs. Compared with those who were not subjects of gun violence, the subjects of gunshots were 3.8 years younger (23.2 vs 27.0 years) and more likely to be male (97.0% vs 80.9%), black (79.8% vs 75.3%), and involved in a gang (52.3% vs 24.3%). Consistent with prior research,¹⁰ subjects of gunshot violence were concentrated within the network. Gunshot subjects were socially close to other gunshot subjects in the network: 17.9% of first-degree associates of subjects of gun violence were also subjects compared with 9.8% for subjects who were not. This pattern was similar for second-degree and third-degree associates as well (Table), indicating that there were clusters in the network with many subjects of gun violence and other parts with few subjects of gun violence.

To analyze the concentration of subjects of gun violence within the network, we sought to distinguish between 3 potential explanations, namely, homophily, confounding, and social contagion.⁴⁹⁻⁵² Homophily is when individuals associate with similar people, confounding occurs when individuals are exposed to the same environmental factors, and social contagion refers to individuals influencing one another's behavior; homophily and confounding can generate situations that appear to be due to social contagion. To explore the ability of homophily and confounding to generate the observed data, we

Figure 2. Graphical Representation of the Largest Connected Component of the Network



Each node represents a unique individual (N = 138 163). Red nodes identify subjects of a fatal or nonfatal gunshot injury (n = 9773); blue nodes represent people who were not subjects of gun violence (n = 128 390). Data are from the Chicago Police Department, as described in the Data subsection of the Methods section.

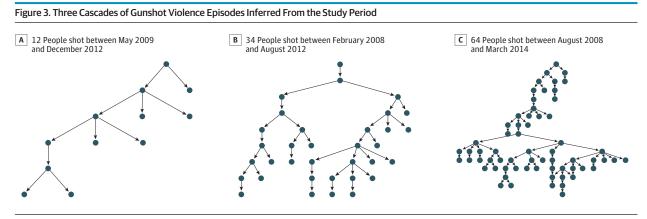
performed simulation experiments based on demographics and the dates of violence episodes. The results of these experiments suggested that homophily and confounding were insufficient explanations for the data, leaving social contagion as a more likely explanation (section 3 of the eMethods and eFigure 7 in the Supplement).

Modeling Contagion

After calibrating our model to the data, we found that 63.1% (n = 7016) of the 11123 gunshot violence episodes in the network during the study period were attributable to social contagion (section 4 of the eMethods and eFigure 8 in the Supplement). In total, 60.8% of fatal violence episodes and 62.6% of nonfatal violence episodes were attributable to social contagion. Subjects of gun violence were shot on average 125 days after their infector (the person most responsible for exposing the subject to gunshot violence), with a median time difference of 83 days. From tracing gunshot violence episodes through the network, we detected 4107 separate cascades (connected chains of infection through the network), ranging in size from cascades with a single subject to a cascade involving 469 subjects, with 680 cascades involving multiple subjects and a mean cascade size of 2.7 subjects (eFigure 9 in the Supplement). Figure 3 shows 3 representative cascades, containing 12 people, 34 people, and 64 people, all shot during the study period, and illustrating the pathways of diffusion between individuals. These cascades visually reinforce how gunshot violence spreads through a co-offending network, connecting individuals who initially may have had no connections to one another. They also help to explain the concentration of subjects of gun violence, as summarized in the Table and Figure 2, because violence episodes in one part of the network generate further violence episodes in that same region.

Predicting Subjects of Gun Violence

Figure 4 shows a comparison of the 3 models to predict the subjects of gun violence, namely, a model based on demographics, a model based on social contagion, and a model based on both social contagion and demographics. The social conta-



Each edge (a line with an arrow showing the direction) represents the transmission of gunshot violence from one individual to another. The originators of each cascade are on top.

330 JAMA Internal Medicine March 2017 Volume 177, Number 3

jamainternalmedicine.com

gion model outperformed the demographics model at estimating an individual's risk to be shot (Figure 4 and eFigure 10 in the Supplement). During the study period, the social contagion model identified 5.3% (589 of 11123) of the network's subjects of gun violence among the 1.0% of the population it deemed highest risk each day compared with 4.3% (475 of 11123) identified by the demographics model (24.0% increase). The combined model performed best, identifying 6.5% (728 of 11123) of subjects of gun violence when selecting the 1.0% highest-risk population daily. Compared with the demographics model, across the 3 daily high-risk population sizes considered (0.1%, 0.5%, and 1.0%), the combined model correctly identified 71.7%, 65.5%, and 53.3% more subjects of gun violence, respectively.

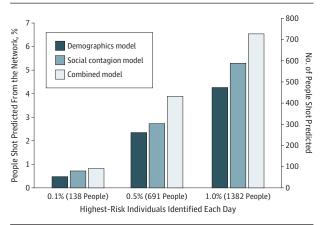
Discussion

Comparing levels of gun violence in the United States and its concentration within communities with an epidemic garners wide appeal but, scientifically, often stops at descriptive and spatial analyses. Whereas previous research has been cross-sectional, the present study advances understanding of gun violence by modeling it as social contagion and by directly tracking the contagion's spread. Our findings suggest not only that gunshot violence concentrates within certain populations but also that the diffusion of violence follows an epidemic-like process of social contagion that is transmitted through networks by social interactions. Violence prevention efforts that account for contagion, in addition to demographics, to identify likely subjects of gun violence have the potential to prevent more shootings than efforts that focus on only demographics.

Our research suggests that a holistic public health approach to gun violence should be developed in at least 2 ways.⁵³ First, violence prevention efforts should consider the social dynamics of gun violence: tracing the spread of violence episodes through social networks could provide valuable information for public health and medical professionals, in addition to law enforcement, looking to intervene with the people and communities at highest risk. Given that public health and epidemiology are founded on studying pathways of transmission, approaches from these domains may readily extend to gun violence prevention efforts. For example, information on the timing and pathways of gunshot cascades might provide street outreach workers of campaigns (eg, Cure Violence, a violence prevention model used in more than 50 US cities that draws on public health methods to mediate conflicts before they become violent⁵⁴) with a more accurate assessment of the people who would most benefit from their program. Likewise, hospital-based violence intervention programs^{55,56} might follow such network models to extend their services beyond the emergency department to others within a social network who are also at risk of becoming gunshot subjects.

Second, concerted efforts should focus on making gun violence prevention efforts subject focused rather than offender

Figure 4. Predictions of Gunshot Violence Among High-Risk Populations



Comparison of the ability of the 3 models to identify subjects of gun violence as one of the highest-risk individuals in the network on the day that the individual was shot; predictions for the 0.1%, 0.5%, and 1.0% of individuals at highest risk are shown.

focused by prioritizing the health and safety of those in harm's way. Although mounting evidence from multiple cities suggests that small place-, group-, and network-based interventions can effectively reduce gun violence,⁵⁷⁻⁶⁰ these network-based approaches have often relied heavily or solely on law enforcement activities. The individuals identified in our study are not in contact just with the criminal justice system: they are also deeply embedded within the public health, educational, housing, and other governmental systems. A fully realized public health approach centered on subjects of gun violence includes focused violence reduction efforts that work in concert with efforts aimed at addressing the aggregate risk factors of gun violence, namely, the conditions that create such networks in the first place or otherwise determine which individuals are in such networks (eg, neighborhood disadvantage and failing schools).

Limitations

Several limitations of our study should be noted. First, we lacked additional data that might have been relevant to understanding individual and neighborhood risk factors, such as substance abuse, employment, and police activity. Therefore, our models may have underestimated the predictive ability of demographic and ecological risk factors. Second, although our descriptive findings of the Chicago co-offending network were similar to those from Boston and Newark,^{16,17} additional research is needed to understand how city-specific factors like segregation, public housing policies, street gangs, and the availability of guns might influence the structure of social networks and the transmission process of gun violence within them. Third, our study relied on a single behavioral tie, co-offending, and thus failed to capture other social ties (eg, kinship, friendship, employment, and gang membership) that might also facilitate the contagion process or protect individuals from infection. Specifically, we were unable to assess why some individuals in the social network (indeed, the vast majority) never became gunshot subjects. Understanding resilience in networks is an important next step for research and practice, and future research should expand its focus on the types of networks that foster and abate the contagion of violence. Developing our understanding of resilience in networks might advance a preventive approach to mitigating the effects of gun violence that looks not simply to respond to shootings that have already happened but also to bolster networks that might inoculate from the potential for future shootings.

Conclusions

We analyzed administrative records to show how modeling gun violence as an epidemic that spreads through social networks via interpersonal interactions can improve violence prevention strategies and policies. Our results suggest that an epidemiological approach, modeled on public health interventions developed for other epidemics, can provide valuable information and insights to help abate gun violence within US cities.

ARTICLE INFORMATION

Accepted for Publication: October 5, 2016.

Published Online: January 3, 2017. doi:10.1001/jamainternmed.2016.8245

Author Contributions: Dr Papachristos had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Green, Papachristos. Acquisition, analysis, or interpretation of data: All authors.

Drafting of the manuscript: All authors. Critical revision of the manuscript for important intellectual content: Green, Papachristos. Statistical analysis: Green, Horel. Obtained funding: Papachristos. Administrative, technical, or material support: Papachristos.

Conflict of Interest Disclosures: None reported.

Funding/Support: This research was supported by the John D. and Catherine T. MacArthur Foundation and by CAREER award SES-1151449 from the Sociology and Law and Social Sciences programs at the National Science Foundation (both to Dr Papachristos).

Role of the Funder/Sponsor: The funding sources had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Disclaimer: The findings of this article represent the opinions of the authors and not those of the City of Chicago or the Chicago Police Department.

Additional Contributions: Alan Altshuler, MA, PhD (Harvard University), Nicholas Christakis, MD, MPH, PhD (Yale University), Manuel Gomez-Rodriguez, MS, PhD (Max Planck Institute for Software Systems), David Kirk, PhD (Oxford University), Michael Morse, BS (Harvard University), Christopher Muller, PhD (University of California, Berkeley), Yaron Singer, PhD (Harvard University), Alexander Tsai, PhD, MD (Harvard Medical School), and Christopher Wildeman, PhD (Cornell University) provided helpful comments. None received compensation.

REFERENCES

 Centers for Disease Control and Prevention.
Welcome to WISQARS (Web-Based Injury Statistics Query and Reporting System). Nonfatal injury data:
2001 to 2013. https://www.cdc.gov/injury/wisqars.
Published 2015. Accessed November 19, 2016. 2. Wintemute GJ. The epidemiology of firearm violence in the twenty-first century United States. *Annu Rev Public Health*. 2015;36:5-19.

 Morenoff JD, Sampson RJ, Raudenbush SW. Neighborhood inequality, collective efficacy, and the spatial dynamics of urban violence. *Criminology*. 2001;39(3):517-559.

4. Peterson RD, Krivo LJ. *Divergent Social Worlds: Neighborhood Crime and the Racial-Spatial Divide*. New York, NY: Russell Sage; 2010.

5. Braga AA. Serious youth gun offenders and the epidemic of youth violence in Boston. *J Quant Criminol.* 2003;19(1):33-54.

6. Christoffel KK. Firearm injuries: epidemic then, endemic now. *Am J Public Health*. 2007;97(4):626-629.

7. Slutkin G. Violence is a contagious disease. In: Patel DM, Simon MA, Taylor RM, eds. *Contagion of Violence: Workshop Summary*. Washington, DC: National Academy of Sciences; 2013:94-111.

8. Cook PJ, Laub JH. After the epidemic: recent trends in youth violence in the United States. *Crime Justice*. 2002;29:1-37.

9. Zeoli AM, Pizzaro JM, Grady SC, Melde C. Homicide as infectious disease: using public health methods to investigate the diffusion of homicide. *Justice Q*. 2014;31(3):609-632.

10. Papachristos AV, Wildeman C, Roberto E. Tragic, but not random: the social contagion of nonfatal gunshot injuries. *Soc Sci Med*. 2015;125(1): 139-150.

11. Mohler GO, Short MB, Brantingham PJ, Schoenberg FP, Tita GE. Self-exciting point process modeling of crime. *J Am Stat Assoc.* 2011;106 (493):100-108.

12. Cohen J, Tita G. Diffusion in homicide: exploring a general method for detecting spatial diffusion processes. *J Quant Criminol*. 1999;15(4):451-493.

13. Tita GE, Cohen J, Engberg J. An ecological study of the location of gang "set space." *Soc Probl.* 2005; 52(2):272-299.

14. Cohen J, Cork D, Engberg J, Tita G. The role of drug markets and gangs in local homicide rates. *Homicide Stud.* 1998;2(3):241-262.

15. Sampson RJ. *Great American City: Chicago and the Enduring Neighborhood Effect*. Chicago, IL: University of Chicago Press; 2012.

16. Papachristos AV, Braga AA, Hureau DM. Social networks and the risk of gunshot injury. *J Urban Health*. 2012;89(6):992-1003.

17. Papachristos AV, Braga AA, Piza E, Grossman L. The company you keep? the spillover effects of gang membership on individual gunshot victimization in a co-offending network. *Criminology*. 2015;53(4):624-649.

18. Tracy M, Braga AA, Papachristos AV. The transmission of gun and other weapon-involved violence within social networks. *Epidemiol Rev.* 2016;38(1):70-86.

19. Centola D. The spread of behavior in an online social network experiment. *Science*. 2010;329 (5996):1194-1197.

20. Bakshy E, Messing S, Adamic LA. Political science: exposure to ideologically diverse news and opinion on Facebook. *Science*. 2015;348(6239): 1130-1132.

21. Bond RM, Fariss CJ, Jones JJ, et al. A 61-million-person experiment in social influence and political mobilization. *Nature*. 2012;489(7415): 295-298.

22. Adams J, Moody J, Morris M. Sex, drugs, and race: how behaviors differentially contribute to the sexually transmitted infection risk network structure. *Am J Public Health* 2013;103(2):322-329

23. Christakis NA, Fowler JH. The spread of obesity in a large social network over 32 years. *N Engl J Med*. 2007;357(4):370-379.

24. Rosenquist JN, Fowler JH, Christakis NA. Social network determinants of depression. *Mol Psychiatry*. 2011;16(3):273-281.

25. Warr M. *Companions in Crime: The Social Aspects of Criminal Conduct*. New York, NY: Cambridge University Press; 2002.

26. Turanovic JJ, Young JTN. Violent offending and victimization in adolescence: social network mechanisms and homophily. *Criminology*. 2016;54 (3):487-519.

27. Haynie DL. Friendship networks and delinquency: the relative nature of peer delinquency. *J Quant Criminol*. 2002;18(2):99-134.

28. Haynie DL. Delinquent peers revisited: does network structure matter? *Am J Sociol*. 2001;106 (4):1013-1057.

29. Young JT. How do they "end up together"? a social network analysis of self-control, homophily, and adolescent relationships. *J Quant Criminol*. 2011;27(3):251-273.

30. Koester S, Glanz J, Barón A. Drug sharing among heroin networks: implications for HIV and hepatitis B and C prevention. *AIDS Behav.* 2005;9 (1):27-39.

31. Bearman PS, Moody J, Stovel K. Chains of affection: the structure of adolescent romantic and sexual networks. *Am J Sociol*. 2004;110(1):44-91.

32. Sutherland EH. *Principles of Criminology*. 4th ed. Philadelphia, PA: J B Lippincott; 1947.

 Osgood DW, Wilson JK, O'Malley PM, Bachman JG, Johnston LD. Routine activities and individual deviant behavior. Am Social Rev. 1996;61(4):635-655.

34. Felson RB. Routine activities and involvement in violence as actor, witness, or target. *Violence Vict*. 1997;12(3):209-221.

35. Morenoff JD, Sampson RJ. Violent crime and the spatial dynamics of neighborhood transition: Chicago, 1970-1990. *Soc Forces*. 1997;76(1):31-64.

36. Block R, Block CR. Street gang crime in Chicago. In: Klein MW, Maxson CL, Miller J, eds. *The Modern Gang Reader*. Thousand Oaks, CA: Roxbury; 1995.

37. Papachristos AV, Meares TL, Fagan J. Attention felons: evaluating Project Safe Neighborhoods in Chicago. *J Empir Leg Stud*. 2007;4(2):223-272.

38. Griffiths E, Chavez JM. Communities, street guns, and homicide trajectories in Chicago, 1980-1995: merging methods for examining homicide trends across space and time. *Criminology*. 2004:42(4):941-978.

39. US Department of Justice. Crime in the U.S. https://ucr.fbi.gov/crime-in-the-u.s/2015/crime-in -the-u.s.-2015. Published 2015. Accessed February 9, 2016.

40. DeSilver D. Despite recent shootings, Chicago nowhere near US "murder capital." http://www.pewresearch.org/fact-tank/2014/07/14 /despite-recent-shootings-chicago-nowhere-nearu-s-murder-capital. Published July 14, 2014. Accessed September 21, 2016.

41. Schnell C, Braga AA, Piza EL. The influence of community areas, neighborhood clusters, and street segments on the spatial variability of violent crime in Chicago. *J Quant Criminol*. doi:10.1007 /s10940-016-9313-x

42. Hawkes AG. Spectra of some self-exciting and mutually exciting point processes. *Biometrika*. 1971; 58(1):83-90.

43. Marsan D, Lengliné O. Extending earthquakes' reach through cascading. *Science*. 2008;319 (5866):1076-1079.

44. Linderman SW, Adams RP. Discovering latent network structure in point process data. In: Proceedings from the 31st International Conference on Machine Learning; June 21-24, 2014; Beijing, China.

45. Farajtabar M, Du N, Gomez-Rodriguez M, Valera I, Zha H, Song L. Shaping social activity by incentivizing users. In: Proceedings from the 28th Annual Conference on Neural Information Processing Systems; December 8-13, 2014; Montreal, Canada.

46. Gomez-Rodriguez M, Leskovec J, Krause A. Inferring networks of diffusion and influence. *ACM Trans Knowl Discov*. February 2012;5(4).

47. Viboud C, Bjørnstad ON, Smith DL, Simonsen L, Miller MA, Grenfell BT. Synchrony, waves, and spatial hierarchies in the spread of influenza. *Science*. 2006;312(5772):447-451.

48. Sen A, Smith T. *Gravity Models of Spatial Interaction Behavior*. New York: NY: Springer Science & Business Media; 2012.

49. Aral S, Muchnik L, Sundararajan A. Distinguishing influence-based contagion from homophily-driven diffusion in dynamic networks. *Proc Natl Acad Sci U S A*. 2009;106(51):21544-21549.

50. Cohen-Cole E, Fletcher JM. Detecting implausible social network effects in acne, height, and headaches: longitudinal analysis. *BMJ*. 2008; 337:a2533.

51. Shalizi CR, Thomas AC. Homophily and contagion are generically confounded in

Invited Commentary

observational social network studies. *Sociol Methods Res.* 2011;40(2):211-239.

52. Anagnostopoulos A, Kumar R, Mahdian M. Influence and correlation in social networks. In: Proceedings from the 14th ACM SIGKDD International Conference on Knowledge Discovery and Data Mining; August 24-27, 2008; Las Vegas, Nevada.

53. Hemenway D. *Private Guns, Public Health*. Ann Arbor: University of Michigan Press; 2004.

54. Butts JA, Roman CG, Bostwick L, Porter JR. Cure Violence: a public health model to reduce gun violence. *Annu Rev Public Health*. 2015;36(1):39-53.

55. Chong VE, Smith R, Garcia A, et al. Hospital-centered violence intervention programs: a cost-effectiveness analysis. *Am J Surg*. 2015;209 (4):597-603.

56. Purtle J, Dicker R, Cooper C, et al. Hospital-based violence intervention programs save lives and money. *J Trauma Acute Care Surg.* 2013;75(2):331-333.

57. Braga AA, Weisburd DL. The effects of focused deterrence strategies on crime: a systematic review and meta-analysis of the empirical evidence. *J Res Crime Delinq*. 2011;49(3):323-358.

58. Corsaro N, Engel RS. Most challenging of contexts: assessing the impact of focused deterrence on serious violence in New Orleans. *Criminol Public Policy*. 2015;14:471-505.

59. Papachristos AV, Kirk DS. Changing the street dynamic: evaluating Chicago's Group Violence Reduction Strategy. *Criminol Public Policy*. 2015;14 (3):525-558.

60. Kennedy DM, Braga AA, Piehl AM. The (un)known universe: mapping gangs and gang violence in Boston. In: Weisburd D, McEwen T, eds. *Crime Mapping and Crime Prevention*. Monsey, NY: Criminal Justice Press; 1997.

FIREARM VIOLENCE

Firearm Violence as a Disease—"Hot People" or "Hot Spots"?

Charles C. Branas, PhD; Sara Jacoby, PhD; Elena Andreyeva, PhD

In this issue of *JAMA Internal Medicine*, Green and colleagues¹ report on firearm violence in Chicago, Illinois, from 2006 to 2014 and show how the violence is transmitted by social interaction through networks of people. The study establishes that the spread of firearm violence can be understood with parameters that have been used for more than half a century

←

Related article page 326

to model the spread of infectious diseases. This important finding helps put to rest the mistaken idea that epi-

demiology, medicine, and public health somehow have no place in the prevention of firearm violence, a disease process that affects roughly 100 000 people in the United States each year.^{2,3}

Firearm violence is a problem that many fields, including criminology, sociology, and law enforcement, have contributed to better understanding and preventing. However, when a person is shot with a firearm in the United States, there is more than just a police and justice system response. If the person survives long enough, he or she will be treated by emergency medical services professionals and then at a trauma or other medical center; if the person dies, a medical examiner or coroner will likely conduct an autopsy. Substantial medical and public health resources are expended in responding to firearm violence. Correspondingly substantial biomedical research resources are also needed to better understand and prevent this acute and often fatal pathophysiological process.³

Although the study by Green and colleagues¹ provides innovative quantitative evidence, modeling the transmission of firearm violence as an epidemiological phenomenon is not new. Most prior studies, however, have documented ecological transmission between neighborhoods or other groups of people (eg, gangs and peer groups). A few studies have applied mathematical models to person-to-person transmission of firearm violence⁴; however, these studies have been simulations. In contrast, Green and colleagues¹ took an important next step

jamainternalmedicine.com