



Published in final edited form as:

Curr Epidemiol Rep. 2019 June ; 6(2): 200–207. doi:10.1007/s40471-019-00195-4.

Identifying and Addressing Confounding Bias in Violence Prevention Research

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Abstract

Purpose of review: Violence prevention research has enhanced our understanding of individual and community risk and protective factors for aggression and violence. However, our knowledge of risk and protective factors for violence is highly dependent on observational studies, since there are few randomized trials of risk and protective factors for violence. Observational studies are susceptible to systematic errors, specifically confounding, and may lack internal validity.

Recent findings: Many violence prevention studies utilize methods that do not correctly identify the set of covariates needed for statistical adjustment. This results in unwarranted matching and restriction leading to further confounding or selection bias. Covariate adjustment based on purely statistical criteria generates inconsistent results and uncertain conclusions.

Summary: Conventional methods used to identify confounding in violence prevention research are often inadequate. Causal diagrams have potential to improve the understanding and identification of potential confounding biases in observational violence prevention studies, and methods like sensitivity analysis using quantitative bias analysis can help to address unmeasured confounding. Violence research studies should make more use of these methods.

Keywords

Confounding; directed acyclic graphs; violence prevention

Introduction

Violence prevention research has enhanced our understanding of individual and community risk and protective factors for aggression and violence. However, a weakness of the field is that our knowledge of risk and protective factors for violence is highly dependent on observational studies. There are a few randomized trials of examining the effect of risk and protective factors for violence, but conducting such studies in violence prevention introduces many ethical concerns that most often can only be navigated by using non-randomized observational designs. However, observational studies may lack internal validity. The main challenge to the validity of observational studies is that the observed associations between

risk/protective factors and health outcomes may be biased due to presence of other factors acting as confounders or selection factors [1]. Observational studies may also have information bias that lead to misclassification of exposures and outcomes [1]. These three types of biases (confounding bias, selection bias, and information bias) are collectively referred to as systematic errors [1]. Such biases are widely noted in observational studies examining the association between various risk/protective factors and violence-related outcomes like violence victimization [2–4], sexual and intimate partner violence [5–7], youth violence [8,9], child maltreatment [10,11], elder abuse [12], firearm-related violence [13,14], homicides [2–7], suicides [15], and legal intervention deaths [16]. Yet, methods for addressing these biases are seldom discussed. This review illustrates the use of modern epidemiologic methods for the addressing the most common of these sources of bias, confounding.

Of the three systematic biases noted above, selection and information bias tend to be readily identified and discussed in limitation sections of most peer reviewed published research [17–19]. However, confounding bias is seldom examined or discussed in violence prevention research. In the majority of observational violence research, potential confounders remain unmeasured with little discussion on how this might affect the study results. Some authors discuss additional confounding as a limitation, but do not address it directly in their research and sometimes conclude, with little or no empirical justification, that such confounding may not affect their study substantively [20]. Many authors have gone a step further and stated that causal interpretations cannot be made from their study [21–25]. However, as public health scientists, we must acknowledge that such blanket statements do not absolve us from how our study results are used in informing violence prevention interventions. Despite their limitations, the majority of our understanding today about how risk/protective factors affect violence-related outcomes depends on observational studies, since randomized studies examining risk/protective factors for violent outcomes are not likely to be ethically feasible.

A common analytical strategy in violence research is to control for as many covariates as possible, typically using one or more statistical techniques. Some studies have attempted to refine this approach by only controlling for covariates that have a statistically significant relationship with the study outcome [24,26,27]. Some authors also focus on controlling for covariates that produce an a-priori determined magnitude of change in the relationship between the risk/protective factor of interest and the outcome [27,28]. However, such criteria still do not provide any clarity in identifying potential confounders or deepening our understanding of the confounding processes at play. In fact, adjustment for covariates identified through such criteria may sometimes be unadvisable as they may cause further selection bias in the study [29–31]. In addition, most violence prevention research discount the potential of time-varying confounding and almost never attempt to explore the possibility of unmeasured confounding.

In this review, I will define confounding; discuss the pros and cons of conventional and more definitive methods of identifying confounding using examples from published literature; and discuss methods to explore and address unmeasured confounding in observational violence prevention research.

What is confounding?

Confounding bias occurs when the association between a risk factor and a violent outcome can be completely or partially explained by a third factor (confounder) [1], which predicts both the risk factor and the violent outcome, and the confounder cannot be predicted by the risk factor (Figure 1) [32]. As an example, let us consider greenery (trees and green areas in neighborhoods) in relation to firearm-related violence. In non-randomized studies examining the association of greenery with firearm-related assaults [17], the amount of economic activity in an area (represented by the number of shopping centers, recreational centers, restaurants, movie theatres and other commercial activity) may be associated with both the amount of greenery and violent crimes [18]. Economic activity in an area affects greenery if trees are cut down to make way for shopping centers and malls. Similarly, more economic activity in an area brings more people to the area, and increases the likelihood of assaults related to robbery and gang-related violence [18]. Thus, greenery appears to be positively associated with firearm- and robbery-related assault, however this relationship is confounded by economic activity. An inverse relationship may exist between greenery and intimate partner violence and still confounded by economic activity. Assault related to intimate partner violence is more likely to occur in the privacy of home [33], or residential neighborhoods where greenery is higher and economic activity is lower.

Note that in thinking about confounding and examining associations between risk and protective factors and violent outcomes, the concept of time and temporality are very important [34]. A confounder always occurs temporally prior to the exposure and outcome (Figure 1). A factor that occurs after the exposure has already taken place cannot be a confounder because it cannot retroactively modify the exposure.

The ideal method for confounding control in an experimental study is randomization [35]. Randomization allows assignment of intervention randomly so that the intervention and control arms are balanced [34,35]; that is, all potential confounders are equally distributed between the intervention and the control arms [34]. This balance ensures comparability between the two groups, such that, if we were to switch the groups, so that the control group now gets the intervention and the intervention group ends up getting the control treatment, the observed effect of the intervention on the violent outcomes will be the same. In other words, randomization affords creation of a surrogate for the true counterfactual group for purposes of making an experimental comparison [34]. Note, however, that randomization controls for confounding on average, meaning for large sample sizes or over many studies.

In observational studies, where the risk factors are not random and individuals choose their exposures, or get exposed through various non-randomized and correlated mechanisms, we have to employ other means of confounding control. Commonly used methods for confounding control in observational studies include restriction, matching, stratification, and statistical adjustment including direct adjustment of variables in regression analyses, direct standardization (e.g., inverse probability weighting) and indirect standardization.

Identifying covariates to control confounding in observational studies

In an attempt to address the problem of measured and unmeasured confounders, a common – but flawed – analytical strategy in violence research is to control for as many covariates as possible, typically by adjusting for these covariates in a regression-based analysis [29]. These covariates might include not only potential confounders for which control would be advisable, but others for which control would be unnecessary or could even result in selection bias (e.g. due to missing data on a confounder). Other conventionally used methods to identify confounders include, 1) adjusting for all factors that have a p -value lower than 0.05 in the statistical model, i.e., adjusting for all statistically significant predictors of the outcome, regardless of the predictors' association with the risk/ protective factor under study [24,26,27]; 2) adjusting for covariates that produce an *a-priori* determined amount of change (e.g., 10% or 15%) in the effect estimate representing the relationship between the outcome and risk/protective factor under study [27,28]; 3) adjusting for all covariates that produce a greater change in the effect estimate as compared to the potential inflation of the standard error of the effect estimate representing the relationship between the risk/protective factor and the outcome (seldom used in violence prevention studies) [36]. These methods are frequently inadequate to address confounding (further discussed in section on traditional methods for confounding control below).

Another well-known method of identifying the covariates needed to control confounding is drawing a directed acyclic graphs (DAGs). However, the use of this method in violence research to date has been limited [4,7,32,37–40]. A DAG allows the investigator to identify causal and non-causal paths of association between a risk/protective factor and an outcome of interest. Considering Figure 1, the arrow starting from the exposure (E) and ending into the outcome (O) is considered a causal path, which can be denoted by $E \rightarrow O$. However, the path where the confounder (C) affects both the exposure and outcome is the non-causal path, denoted as $E \leftarrow C \rightarrow O$. The goal is to block the non-causal path (by adjusting for the confounder) to assess the causal association between the exposure and the outcome. In essence, Figure 1 is a simple form of a DAG. A DAG usually only includes the variables that an investigator observes and includes in her/his analysis. This means that there is always some level of unmeasured or unknown confounding that may not have been addressed. Hence, there is typically a preference for the use of the word “association” rather than “effect” in reporting results from observational studies.

In addition to the causal and non-causal paths observed in Figure 1, there are other types of causal and non-causal paths. A path, that starts from the exposure affecting another variable (also known as the intermediate variable or I), which in turn affects the outcome, is termed as an indirect causal path. Such a path can be denoted as $E \rightarrow I \rightarrow O$; note that all the arrows point towards the outcome. An example of an indirect causal path can be seen in Figure 2a, where the exposure, police reporting, affects an intermediate variable, change in behavior, which further predicts the outcome, future victimization. Naturally, the kind of causal path we see in Figure 1 ($E \rightarrow O$) is called a direct causal path. Similarly non-causal paths can also go through many other covariates. It is important to note that, to block a non-causal path, we only need to control for one well-measured covariate on that path. Ultimately, the purpose of a DAG is to identify a minimally sufficient set of well-measured covariates that control for

all known confounding in the relationship between a risk factor and an outcome of interest [32,37].

Identifying covariates to control confounding using a DAG

To demonstrate the utility of DAGs to identify a minimally sufficient set of control variables, I will use data from a previously published violence study where the authors utilized a DAG to identify the minimal set of control covariates to include in a regression model (the DAG was not published) [4]. The research question was, “does police reporting of crime victimizations affect the incidence of future victimizations?” Figure 2a is the final DAG used in that study. Note that DAGs can be subjective, that is different researchers may write different DAGs to address the same research questions. DAGs that can be supported by previously published literature and developed with consensus among research team members are likely to be more reliable [32]. The DAG presented here (Figure 2a) was similarly developed using prior literature and with consensus from all co-authors listed on the original study [4].

The minimally sufficient set of covariates to control confounding in Figure 2a includes some variables that meet the definition of a confounder (affects both exposure and outcome and is not affected by the exposure). These included type of baseline victimization (interpersonal violence/ burglaries/ thefts), victim demographics (age, sex, race, income, education), victim offender relationship (stranger/ non-stranger), and offender sex (male/female). However, the authors also adjusted for some of the covariates that do not meet the definition of a confounder – place of victimization (inside home/ outside home/ friend’s home/ commercial place/ parking places/ school/ public places/ other), victim injury during the baseline victimization (yes/ no) and bystander presence (yes/ no/ don’t know).

It may seem odd to adjust for covariates that do not meet the definition of a true confounder. To understand, let us consider the scenario where these factors were not adjusted. Essentially, when we adjust for a covariate we nullify its effect on other factors in the DAG using our statistical model. So if we only adjust for the covariates that are true confounders, the regression model may do something like in Figure 2b to our data. Note, the arrows don’t disappear in reality, but their effect is nullified by controlling for them. By removing the arrows associated with the true confounders, we can explicitly appreciate the remaining non-causal paths.

Upon removing the arrows associated with the true confounders, we see that there are still eight non-causal paths that remain:

1. Police report ← Injury ← Place of victimization → Future victimization,
2. Police report ← Injury ← Weapon → [baseline victimization] ← Gang → Future victimization
3. Police report ← Injury ← Weapon → [baseline victimization] ← Place of victimization → Future victimization

4. Police report ← Bystander → Injury ← Weapon → [baseline victimization] ← Place of victimization → Future victimization
5. Police report ← Injury ← Bystander ← Place of victimization → [baseline victimization] ← Gang → Future victimization
6. Police report ← Bystander → Injury ← Place of victimization → [baseline victimization] ← Gang → Future victimization
7. Police report ← Bystander → Injury ← Weapon → [baseline victimization] ← Gang → Future victimization
8. Police report ← Bystander ← Place of victimization → [baseline victimization] ← Gang → Future victimization

Path 1 above can be controlled by controlling for place of victimization. Paths 2 through 8 may appear to be controlled since baseline victimization on those paths was adjusted for (indicated by square brackets around it). However, on paths 2–8, the variable baseline victimization is something we call a “collider” (where two arrows collide) [29–32]. A collider is a covariate that is affected by two other variables that are otherwise independent of each other [30,32]. In such instances, since the two variables affecting the collider are independent, the path is naturally blocked or closed. However, adjusting (or restricting or stratifying) for the collider will open up the pathway and induce a relationship between two naturally independent predictors of the collider. This resulting bias leads to a form of selection bias, also known as collider stratification/ conditioning bias [29–32].

In our example, one way to not induce the collider conditioning bias would be to not control for baseline victimization; however, baseline victimization is a true confounder (Police report ← type of baseline victimization → Future victimization), hence this path must be controlled. Therefore, a better way to control for these paths is by controlling for other covariates on that path. Note that paths 3–5 will be also get closed once we control for place of victimization for path 1. Also note that weapon and gang variables were not well measured in this dataset, so controlling for those will not solve the problem. Hence, the only remaining option to close path 2 was to control for the injury variable. However, that the injury variable is also a collider on paths 6 and 7. So if we had not controlled for injury, paths 6 and 7 would have been closed naturally, being blocked at the injury variable. However, since we did control for injury to close path 2, we effectively opened paths 6 and 7. So finally, by simply adjusting for bystander presence on paths 6 and 7. Lastly, controlling for place of victimization (for paths 1, 3–5) and/or bystander presence (for paths 6 and 7) would automatically close path 8. Thus, we were able to completely close all the pathways. Hence, in addition to adjusting for the traditional confounders, we also adjusted for the place of victimization, bystander presence and injury, which addressed all measured confounding.

Once a minimally sufficient set of well measured control variables is identified, we can use standard statistical methods (e.g., regression analyses) to control for these covariates and estimate the association between a risk/ protective factor and a violent outcome [32]. In studies with repeated measurements of the exposure/ risk factor (e.g., prison entry) and violent outcome (e.g., homicide death), the confounder (e.g., mental health condition) may

vary with time, thereby causing time-varying confounding [41]. Essentially, time-varying confounding occurs when a subsequent measure of a confounder is affected by prior exposure [41]. Such relationships can be mapped out using a DAG; however, addressing such relationships may require use of advanced statistical techniques like inverse probability weighted marginal structural models [29] or g-formula [42].

Why are traditional methods of identifying confounding control covariates inadequate?

As stated earlier, some conventional techniques used to identify potential confounders may depend on *p*-values, *a-priori* change in estimate criteria and bias-variance tradeoff. Although their use is widespread, these methods have several limitations, as described below.

A *p*-value of > 0.05 in the statistical model, for a particular covariate, indicates that the covariate is a predictor of the outcome, but it does not tell us anything about the covariate's relationship with the exposure (risk/protective factor for violence). Such a factor may either have no relationship with the exposure or even be on the causal pathway from the exposure to outcome – for example, the variable for change in behavior in Figure 2. Adjusting for such an intermediate variable may in fact block the causal pathway and induce selection bias due to adjustment on colliders. Note, for example, that change in behavior (e.g., change in work commute route) is a collider on this path: Police report \rightarrow [Change in behavior] \leftarrow unmeasured confounders (e.g., job change) \rightarrow Future victimization. This method of controlling for only strong predictors of the outcome is similar to model fitting approaches generally used in predictive modeling. But, using model fitting approaches to examine the association of a specific risk factor with an outcome is fraught with similar limitations [43]. A model will be more parsimonious (or better fit) if it includes more and the strongest of the predictors of the outcome, which may or may not be related to the exposure and may even be on the causal path. Hence, measures of associations (e.g., risk ratios, rate ratios, odds ratios, hazard ratios, etc.) obtained from a predictive model may not represent the entire relationship between the risk factor and the outcome, and may even be affected by collider conditioning bias. Similar criticism has been appropriated toward interpreting model coefficients for confounders obtained from statistical models [31].

Likewise, an *a-priori* determined change (e.g., 10% or 15%) in the effect estimate does not satisfy all the requirements for a confounder. Specifically, if adjustment for a covariate leads to a substantive change in effect estimate, it indicates that the covariate is on some pathway between the exposure and the outcome. But such change in estimate will also be observed when controlling for an intermediate variable. Thus, change-in-estimate criteria do not distinguish between intermediate and confounder variables and can lead to blocking of the causal effect. Additionally, they may induce selection bias due to collider conditioning. Similar limitations are also observed when selection of adjustment covariates is based on the comparison between the magnitude of bias removed (examined by the % change in estimate) and the variance introduced (change in variance of the effect estimate) in the model.

In contrast, utilizing a DAG explicitly examines all potential pathways through which confounding may arise [4,7,38–40]. This helps address bias not only in the data analyses

phase, but also in the study design phase. As an example, one study examining the association of firearm possession on gun assaults presented “fully adjusted” results controlling for the known predictors of the outcome, and “limited adjusted” results controlling for factors that produce 15% or more change in estimate [28]. The fully adjusted model included factors such as bystander presence and surrounding area at the time of assault, which predict the outcome, but do not temporally precede the risk factor of interest (firearm possession). Hence, they are not traditional confounders of the firearm possession and assault relationship. We may be able to argue that they are part of some non-causal pathway, but we can also equally argue that they may in fact be a part of a causal pathway. Similarly, if a DAG is drawn for such a study (association of firearm possession on gun assaults) before the data is collected, we may be able to see that factors like gang affiliation and gun ownership would be strong confounders, which were not controlled in this study [28]. Because of such limitations of the conventional confounding control methods, we cannot be sure which effect estimate to be certain about, the fully adjusted or the reduced one. Regardless, in this example, it should be noted that, given the large effect estimates in this study and other literature supporting similar results [13,26], the direction of associations noted in this study seems robust [28].

Limitations of DAGs

DAGs offer a pictorial view of a research question at hand, but the picture is only as good as the substantive knowledge of those developing it [44]. The relationships of the known covariates with the exposure and outcome should be determined based on published literature, expert knowledge, and research team consensus. Similarly, a minimally sufficient set of adjustment covariates obtained from a DAG is only as good as the covariate measurement methods [44]. Errors in covariate measurement (misclassification) or large amount of missingness in covariates may lead to further bias.

In reality, it is often difficult to accurately identify all confounders or confounding mechanisms in an observational study. Hence, there is often assumed to be some degree of unmeasured confounding in observational studies. In such cases, the best that a DAG could do is to simply acknowledge that fact. The best tool we have to address all potential confounding (known or unknown) is conducting a randomized controlled trial (RCT). There is no DAG needed for an RCT because randomization ensures that the intervention and control arms are balanced with respect to all potential confounders, thereby removing all arrows that go into the assigned intervention. But an RCT may not be ethically feasible for all violence-related research questions. In the absence of a randomized design, it is incumbent on the investigator to use statistical/ epidemiological tools to increase the robustness of our inferences in the face of unmeasured or unknown confounding. Such tools include sensitivity analyses and quantitative bias analyses.

Addressing unmeasured confounding

Unmeasured confounding can be thought of as 1) confounding pathways that we know exist, but do not have data on, e.g., confounding due to gang affiliation and firearm ownership in studies examining the association of firearm possession on firearm assaults [28]; or 2)

confounding pathways that we do not know about, potentially because of lack of research in that area, but may exist and bias the relationship between the risk/ protective factor and the outcome under study [45]. Acknowledging the presence of these types of confounding is essentially acknowledging the limits of our understanding of the phenomenon that take place in nature. Once acknowledged, one of the best way to address unmeasured and unknown confounding is to conduct sensitivity analyses.

The goal of such sensitivity analyses may vary depending on how much information is available to the investigators on the number and strength of plausible unmeasured confounders. For example, consider a study of the association between firearm possession and assaults in which the research team lacks data on gang affiliation and is concerned that it may confound the results. The research team may be able to establish (perhaps from prior research) how gang affiliation affects firearm possession and how it may affect assaults. They could then simulate these associations in their data and examine how the observed effect estimate (of the relationship between the risk/protective factor and the outcome) would change if they hypothetically were able to adjust for gang affiliation. Such analyses can be readily conducted using quantitative bias analyses methods [46,47]. In one study the authors used a simple sensitivity analyses to develop and adjust for a history of crime variable by combining known information from the data while examining association of hospitalization due to a firearm injury and subsequent violent outcomes [48].

In places where no information may be available about a confounder, an alternative way could be to examine the magnitude of confounding it would take to shift the observed effect estimate completely to null [45]. This is, in essence, one form of “worst-case scenario”, in which the observed association is entirely due to unmeasured confounding. Such a measure has been termed the “E-value”, defined as “the minimum strength of association that an unmeasured confounder would need to have with both treatment and the outcome to fully explain away a specific treatment-outcome association” [49]. If for, example, a very large E-value is needed, then it is seems plausible that such a strong phenomenon would have been already studied and characterized. If there is no research that documents such a strong phenomenon regarding potential unmeasured confounders, we can safely assume that the observed effect estimate for the relationship between the risk/protective factor and the outcome of interest is less likely to be subject to confounding. Methods to calculate the E-value are similar to conducting quantitative bias analyses and should be a standard practice for researchers using observational data [49].

Conclusions

Epidemiologic studies of violence prevention have been helpful in informing interventions and policies that have the potential to shape our society for the better. Utilization of modern epidemiologic methods like DAGs and analytic techniques like quantitative bias analyses will strengthen those efforts by producing a robust evidence-base of risk and protective factors of violence.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

References:

1. Rothman KJ, Greenland S, Lash TJ. Validity in epidemiologic studies In: Rothman KJ, Greenland S, Lash TJ, eds. *Modern Epidemiology* (pp.128–147). Philadelphia, PA: Lippincott Williams & Wilkins; 2008.
2. Florence C, Shepherd J, Brennan I, Simon T. Effectiveness of anonymised information sharing and use in health service, police, and local government partnership for preventing violence related injury: experimental study and time series analysis. *Brit Med J*. 2011; 342:d3313. doi: 10.1136/bmj.d3313 [PubMed: 21680632]
3. Jackson V, Chou S, Browne K. Protective Factors Against Child Victimization in the School and Community: An Exploratory Systematic Review of Longitudinal Predictors and Interacting Variables. *Trauma Violence Abuse*. 2017 7;18(3):303–321. [PubMed: 26492892]
4. Ranapurwala SI, Berg MT, Casteel C. Reporting crime victimizations to the police and the incidence of future victimizations: A longitudinal study. *Plos One*. 2016; 11(7):e0160072. [PubMed: 27466811] •• Study utilizes DAGs in violence research.
5. Yakubovich AR, Stöckl H, Murray J, Melendez-Torres GJ, Steinert JI, Glavin CEY, Humphreys DK. Risk and Protective Factors for Intimate Partner Violence Against Women: Systematic Review and Meta-analyses of Prospective-Longitudinal Studies. *Am J Public Health*. 2018 7;108(7):e1–e11. doi: 10.2105/AJPH.2018.304428. Epub 2018 May 17. •Study identifies systematic errors in violence research.
6. Zeoli AM, Malinski R, Turchan B. Risks and Targeted Interventions: Firearms in Intimate Partner Violence. *Epidemiol Rev*. 2016;38(1):125–39. [PubMed: 26739680] •Study identifies systematic errors in violence research.
7. Sivaraman J, Ranapurwala SI, Moracco KE, Marshall SW. Impact of firearm regulatory strictness on intimate partner homicide and homicide-suicide. *Am J Preventive Med*. 2018 In press. •• Study utilizes DAGs in violence research.
8. Bushman BJ, Newman K, Calvert SL, Downey G, Dredze M, Gottfredson M, Jablonski NG, Masten AS, Morrill C, Neill DB, Romer D, Webster DW. Youth violence: What we know and what we need to know. *Am Psychol*. 2016 1;71(1):17–39. [PubMed: 26766763] •Study identifies systematic errors in violence research.
9. Rothman EF, McNaughton Reyes L, Johnson RM, LaValley M. Does the alcohol make them do it? Dating violence perpetration and drinking among youth. *Epidemiol Rev*. 2012;34:103–19. [PubMed: 22128086] •Study identifies systematic errors in violence research.
10. Choi KW, Sikkema KJ. Childhood Maltreatment and Perinatal Mood and Anxiety Disorders: A Systematic Review. *Trauma Violence Abuse*. 2016 12;17(5):427–453. [PubMed: 25985988] ••Study identifies systematic errors in violence research.
11. Levey EJ, Gelaye B, Bain P, Rondon MB, Borba CP, Henderson DC, Williams MA. A systematic review of randomized controlled trials of interventions designed to decrease child abuse in high-risk families. *Child Abuse Negl*. 2017 3;65:48–57. [PubMed: 28110205] •Study identifies systematic errors in violence research.
12. Feltner C, Wallace I, Berkman N, Kistler CE, Middleton JC, Barclay C, Higginbotham L, Green JT, Jonas DE. Screening for Intimate Partner Violence, Elder Abuse, and Abuse of Vulnerable Adults: Evidence Report and Systematic Review for the US Preventive Services Task Force. *JAMA*. 2018 10 23;320(16):1688–1701. [PubMed: 30357304] •Study identifies systematic errors in violence research.
13. Webster DW, Cerdá M, Wintemute GJ, Cook PJ. Epidemiologic Evidence to Guide the Understanding and Prevention of Gun Violence. *Epidemiol Rev*. 2016;38(1):1–4. [PubMed: 26905892] •Study identifies systematic errors in violence research.
14. Chen D, Wu LT. Association Between Substance Use and Gun-Related Behaviors. *Epidemiol Rev*. 2016;38(1):46–61. [PubMed: 26769722] •Study identifies systematic errors in violence research.

15. Wyatt LC, Ung T, Park R, Kwon SC, Trinh-Shevrin C. Risk Factors of Suicide and Depression among Asian American, Native Hawaiian, and Pacific Islander Youth: A Systematic Literature Review. *J Health Care Poor Underserved*. 2015 5;26(2 Suppl):191–237. [PubMed: 25981098] •Study identifies systematic errors in violence research.
16. Kivisto A, Ray B, Phalen P. Firearm Legislation and Fatal Police Shootings in the United States. *AJPH Policy*. 2017; 107(7): 1068–1075.
17. Kondo MC, South EC, Branas CC, Richmond TS, Wiebe DJ. The Association Between Urban Tree Cover and Gun Assault: A Case-Control and Case-Crossover Study. *Am J Epidemiol*. 2017 8 1;186(3):289–296. [PubMed: 28481962]
18. Wiebe DJ, Richmond TS, Guo W, Allison PD, Hollander JE, Nance ML, Branas CC. Mapping Activity Patterns to Quantify Risk of Violent Assault in Urban Environments. *Epidemiology*. 2016 1;27(1):32–41. [PubMed: 26414941]
19. Miller E, Breslau J, Chung WJ, Green JG, McLaughlin KA, Kessler RC. Adverse childhood experiences and risk of physical violence in adolescent dating relationships. *J Epidemiol Community Health*. 2011 11;65(11):1006–13. [PubMed: 21321063]
20. Snider CE, Brownell M, Dufault B, Barrett N, Prior H, Cochrane C. A multilevel analysis of risk and protective factors for Canadian youth injured or killed by interpersonal violence. *Inj Prev*. 2018 6;24(3):199–204. [PubMed: 28739778]
21. Duke NN, Pettingell SL, McMorris BJ, Borowsky IW. Adolescent violence perpetration: associations with multiple types of adverse childhood experiences. *Pediatrics*. 2010 4;125(4):e778–86. [PubMed: 20231180]
22. Roberts AL, McLaughlin KA, Conron KJ, Koenen KC. Adulthood stressors, history of childhood adversity, and risk of perpetration of intimate partner violence. *Am J Prev Med*. 2011 2;40(2):128–38. [PubMed: 21238860]
23. Mills BM, Nurius PS, Matsueda RL, Rivara FP, Rowhani-Rahbar A. Prior Arrest, Substance Use, Mental Disorder, and Intent-Specific Firearm Injury. *Am J Prev Med*. 2018 9;55(3):298–307. [PubMed: 30122213]
24. Culyba AJ, Abebe KZ, Albert SM, Jones KA, Paglisotti T, Zimmerman MA, Miller E. Association of Future Orientation With Violence Perpetration Among Male Youths in Low-Resource Neighborhoods. *JAMA Pediatr*. 2018 9 1;172(9):877–879. [PubMed: 29971322]
25. Simckes MS, Simonetti JA, Moreno MA, Rivara FP, Oudekerk BA, Rowhani-Rahbar A. Access to a Loaded Gun Without Adult Permission and School-Based Bullying. *J Adolesc Health*. 2017 9;61(3):329–334. [PubMed: 28652055]
26. Kellermann AL1, Rivara FP, Rushforth NB, Banton JG, Reay DT, Francisco JT, Locci AB, Prodzinski J, Hackman BB, Somes G. Gun ownership as a risk factor for homicide in the home. *N Engl J Med*. 1993 10 7;329(15):1084–91. [PubMed: 8371731]
27. Wiebe DJ. Homicide and suicide risks associated with firearms in the home: a national case-control study. *Ann Emerg Med*. 2003 6;41(6):771–82. [PubMed: 12764330]
28. Branas CC, Richmond TS, Culhane DP, Ten Have TR, Wiebe DJ. Investigating the link between gun possession and gun assault. *Am J Public Health*. 2009 11;99(11):2034–40. [PubMed: 19762675]
29. Cole SR, Hernan MA. Constructing inverse probability weights for marginal structural models. *Am J Epidemiol* 2008; 168(6):656–664. [PubMed: 18682488]
30. Greenland S. Quantifying biases in causal models: classical confounding vs collider-stratification bias. *Epidemiology*. 2003;14(3):300–306. [PubMed: 12859030]
31. Westreich D, Greenland S. The table 2 fallacy: Presenting and interpreting confounder and modifier coefficients. *Am J Epidemiol*. 2013; 177(4):292–8. [PubMed: 23371353]
32. Greenland S, Pearl J, Robins JM. Causal diagrams for epidemiologic research. *Epidemiology* 1999; 10(1):37–48. [PubMed: 9888278]
33. Field S, Onah M, van Heyningen T, Honikman S. Domestic and intimate partner violence among pregnant women in a low resource setting in South Africa: a facility-based, mixed methods study. *BMC Womens Health*. 2018 7 4;18(1):119. doi: 10.1186/s12905-018-0612-2. [PubMed: 29973182]

34. Hernán MA, Robins JM (2018). Causal Inference. Boca Raton: Chapman & Hall/CRC, forthcoming.
35. Rothman K. Epidemiologic methods in clinical trials. *Cancer* 1977;39:1771–75. [PubMed: 322841]
36. Sjölander A1, Greenland S. Ignoring the matching variables in cohort studies - when is it valid and why? *Stat Med.* 2013 11 30;32(27):4696–708. [PubMed: 23761197]
37. Howards PP, Schisterman EF, Poole C, Kaufman JS, Weinberg CR. “Toward a clearer definition of confounding” revisited with directed acyclic graphs. *Am J Epidemiol.* 2012;176(6):506–511. [PubMed: 22904203]
38. Gurka KK, Marshall SW, Casteel C, Runyan CW, Loomis DP, Richardson DB. An examination of strategies for preventing workplace homicides committed by perpetrators that have a prior relationship with the workplace or its employees. *J Occ Environ Med.* 2012 12;54(12):1533–8. •• Study utilizes DAGs in violence research.
39. Hatzenbuehler ML, Schwab-Reese L, Ranapurwala SI, Hertz M, Ramirez MR. Anti-Bullying Policies Reduce the Risk of Bullying Victimization: A State-Level Analysis. *JAMA Pediatrics,* 2015; 169(10):e152411 10.1001/jamapediatrics.2015.2411. [PubMed: 26437015] •• Study utilizes DAGs in violence research.
40. Ranapurwala SI, Peek-Asa C, Casteel C. Volunteering in Adolescence and Adult Delinquency: A Longitudinal Analysis from the Add Health Study. *Inj Epidemiol.* 2016 12;3(1):26. [PubMed: 27807807] •• Study utilizes DAGs in violence research.
41. Robins J, Hernán M. Estimation of the causal effects of time-varying exposures In: Fitzmaurice G, Davidian M, Verbeke G, Molenberghs G, eds. *Advances in Longitudinal Data Analysis.* Boca Raton, FL: Chapman & Hall; 2009:553–599.
42. Naimi AI, Cole SR, Kennedy EH. An introduction to g methods. *Int J Epidemiol.* 2017;46(2):756–762. [PubMed: 28039382]
43. Stoddard SA, Zimmerman MA, Bauermeister JA. Thinking about the future as a way to succeed in the present: a longitudinal study of future orientation and violent behaviors among African American youth. *Am J Community Psychol.* 2011 12;48(3–4):238–46. [PubMed: 21104432]
44. Glymour MM, Greenland S. Causal diagrams In: Rothman KJ, Greenland S, Lash TJ, eds. *Modern Epidemiology* (pp.183–209). Philadelphia, PA: Lippincott Williams & Wilkins; 2008.
45. Fewell Z, Davey Smith G, Sterne JAC. The impact of residual and unmeasured confounding in epidemiologic studies: A simulation study. *Am J Epidemiol.* 2007; 166(6):646–55. [PubMed: 17615092]
46. Lash TL, Fox MP, MacLehose RF, Maldonado G, McCandless LC, Greenland S. Good practices for quantitative bias analysis. *Int J Epidemiol.* 2014;43(6):1969–1985. [PubMed: 25080530]
47. Last TL, Fox MP, Fink AK. *Applying quantitative bias analysis to epidemiologic data.* London, New York: Springer; 2009.
48. Rowhani-Rahbar A, Zatzick D, Wang J, Mills BM, Simonetti JA, Fan MD, Rivara FP. Firearm-related hospitalization and risk for subsequent violent injury, death, or crime perpetration: a cohort study. *Ann Intern Med.* 2015 4 7;162(7):492–500. [PubMed: 25706337] •• Study utilizes sensitivity analyses in violence research.
49. VanderWeele TJ, Ding P. Sensitivity Analysis in Observational Research: Introducing the E-Value. *Ann Intern Med.* 2017 8 15;167(4):268–274. [PubMed: 28693043]

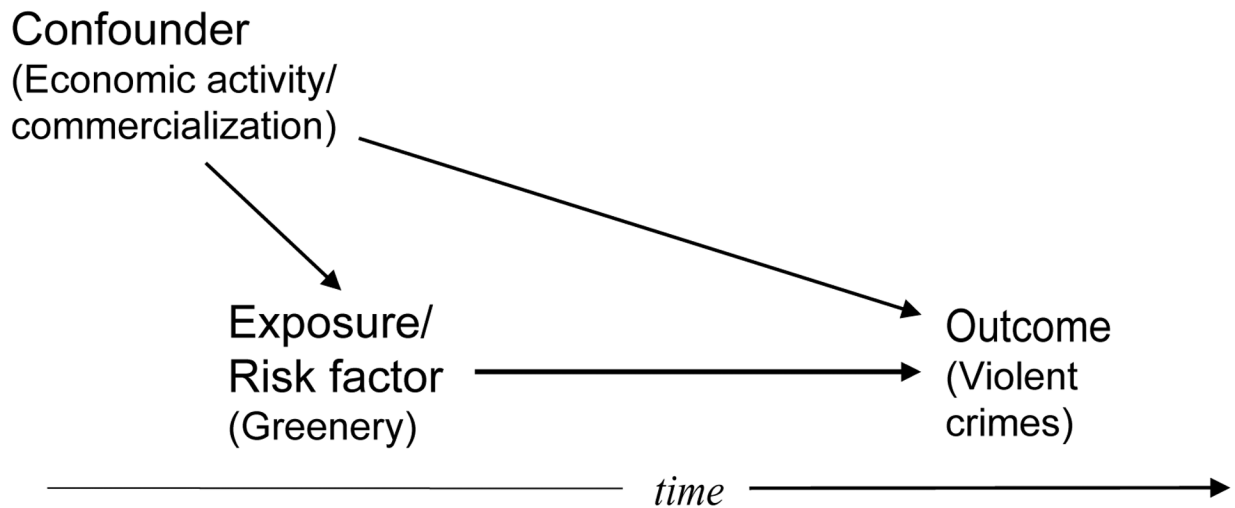


Figure 1.
Relationship of a confounder with exposure and outcome

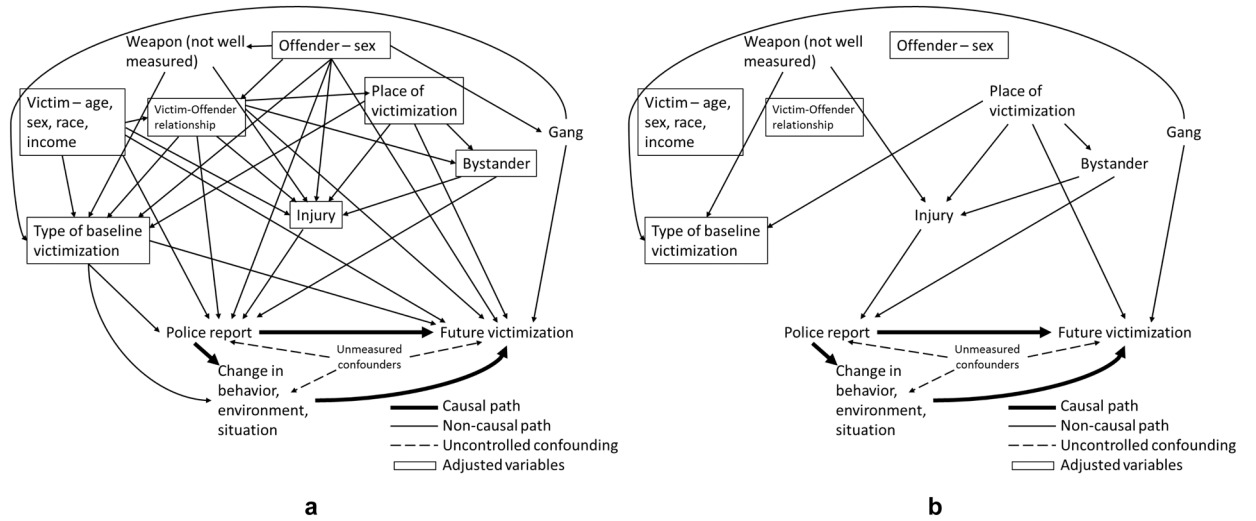


Figure 2. Association of police reporting with incidence of future victimization;
 a) minimal sufficient set of well measured covariates (boxed variables) needed to control for all observed confounding; b) adjustment for only the traditional confounders leads to incomplete confounding control