Published in final edited form as:
Depress Anxiety. 2012 May ; 29(5): 359–385. doi:10.1002/da.21947.

EPIDEMIOLOGIC RESEARCH ON INTERPERSONAL VIOLENCE AND COMMON PSYCHIATRIC DISORDERS: WHERE DO WE GO FROM HERE?

Magdalena Cerdá Dr., P.H., M.P.H.[*,1], **Julia DiGangi, M.S., M.A.**[2], **Dr. Sandro Galea, M.D., P.H.**[1], and **Karestan Koenen, Ph.D.**[1]

[1]Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, New York

[2]Department of Clinical Psychology, DePaul University, Chicago, Illinois

BACKGROUND

Interpersonal violence is one of the major causes of death and disability worldwide.[1] For every death, many more people are exposed to nonfatal violence: for example, in national, population-based surveys, estimates of violence exposure before age 18 range from 14.0% for family violence,[2,3] 49.6–53.1% for physical assault, 6.7–8% for sexual victimization, and 24.8–29.7% for community violence.[4] Several major studies and reviews have identified interpersonal violence as a leading contributor to common forms of psychopathology, including depression, anxiety, and substance abuse.[5–7] In the past 20 years, longitudinal studies on violence and psychopathology have proliferated. However, important methodological issues remain that may affect the validity of inferences that may be drawn from the existing data, including confounding by previolence psychiatric disorders, selection bias, limitations to causal inference inherent to observational studies, and a need to consider the evolution of violence exposures and violence across key life stages.

As we consolidate current evidence and consider how to move forward in research on the mental health implications of violence, it is worthwhile to examine how these methodological issues affect interpretations of the current literature. In this review, we examine current research on the relationship between interpersonal violence and three of the most common psychiatric disorders: depression, anxiety, and substance abuse. We focus on these disorders due to the important public health burden they place on the population: according to the National Comorbidity Survey Replication, 28.8% of the US population had suffered from an anxiety disorder sometime in their life, 20.8% suffered from depression, and 14.6% had a substance use disorder.[8] We only review longitudinal studies because they allow us to establish a temporal relationship between violence exposure and psychopathology. The review is restricted to interpersonal violence, as research on collective forms of violence such as terrorism and war raises a separate set of study design and sampling issues that are beyond the scope of this paper. Prior reviews on the mental health implications of man-made and natural disasters cover key methodological issues that arise in research on collective violence.[9,10]

[*]Correspondence to: Magdalena Cerdá, Department of Epidemiology, Mailman School of Public Health, Columbia University, 722 W168th St., New York, NY 10032. mc3226@columbia.edu.

Drawing on this evidence, we critically evaluate the methodological limitations of the literature. We then propose an epidemiologic framework for future research on violence exposures and psychopathology, discussing how we can build on methodological innovations from the field of epidemiology to improve the study of violence and mental health.

Our review excludes posttraumatic stress disorder (PTSD). The explicit link of PTSD, by definition (criterion A1), with violence-related traumatic events raises a set of methodological issues, in terms of the measurement and link of the violent event with the disorder, the definition of the disorder assessment period, and the definition of the population at risk, that are different from those shared by depression, other types of anxiety, and substance abuse. A separate review is available on prospective studies of PTSD.[11]

METHODS

The review was based on articles found in Psychinfo. Interpersonal violence was defined as: experiences of victimization by physical and sexual abuse, physical and sexual assault, community-based violence, and school-based violence, and witnessing these forms of violence. The inclusion criteria required that participants: (1) experienced at least one of the above-mentioned forms of interpersonal violence; (2) were longitudinally assessed on variables related to depression, anxiety (except if the study focused exclusively on PTSD as an outcome), or substance abuse; (3) used quantitative methods; and (4) were written in English and in peer-reviewed journals. Studies that focused exclusively in forms of collective violence (i.e. war, terrorism) were not included in the review. The search terms included forms of the following terms alone and in combination: abuse, trauma, violence, combat, maltreatment, longitudinal, depression, anxiety, and substance. Studies that examined either psychiatric disorder diagnoses or symptoms were included in the review. To maximize search effectiveness, Boolean methods were used (e.g. depress*). The search initially yielded 499 articles, of which 464 were removed because they did not use a longitudinal study design. The review thus covers 35 studies. Although there was no publication date constraint, no studies published prior to 1996 met the inclusion criteria.

RESULTS

Studies reviewed are in Table 1. Twenty-seven of the studies examined the psychopathological correlates of child victimization,[12–38] five assessed the effects of violence in an adult population[39–43] and three studies included experience of violence as either a child and/or adult.[44–46] The most common categories of violence examined were physical and sexual violence, followed by witnessed- and community-based violence. Twenty-seven of the studies examined depression,[13–17,20,23–29,31–35,37–40,42–46] 17 examined anxiety,[15–17,20,21,25–29,31,33,36,39,41,44,45] and five looked at substance abuse.[12,19,29,38,45]

CHILDHOOD VIOLENCE EXPOSURES

Children who experienced physical or sexual abuse were at greater risk for developing and maintaining depressive and anxious symptoms than children with no abuse history.[15,17,18,23,30,37] Silverman et al.[33] assessed participants at age 15 and 21 and found that depression and anxiety (DAS) were the most common sequelae of child sexual and physical abuse. Likewise, Copeland et al.[17] found that abused children had more than three times the rate of any depressive disorder (i.e. major depressive episode, dysthymia, or depressive disorder not otherwise specified), more than four times the rate of any anxiety disorder (i.e. separation anxiety disorder, generalized anxiety disorder, or social anxiety disorder), and almost double the rate of a substance use disorder by age 16 as those not

abused. Roberts et al.[30] showed women who experienced child sexual abuse were more likely to report being depressed as adult mothers. In addition to anxious and depressive symptomatology, sexual and physical abuse experienced in childhood or adolescence was associated with drug and alcohol abuse in late adolescence and young adulthood [12,19,38]. Child abuse was associated with increased risk for psychiatric comorbidity.[25,27,37]

Most studies that considered the influence of potential confounders on the relationship between child abuse and psychopathology found an independent association between the two factors.[12,19–21,25–29,35,37] For example, Wang et al.[35] found that retrospective accounts of childhood violence, including abuse, predicted higher levels of depression symptoms in police officers at follow-up even after controlling for current depression and PTSD symptoms. Bailey et al.[12] found that girls' childhood sexual abuse was associated with substance use by age 17, even when controlling for co-occurring forms of child abuse, childhood depression and aggression, family income, maternal substance use, and parenting practices. However, the results reported by Boney-McCoy and Finkelhor,[13] authors of the only prospective study with both pre- and postviolence measures of psychopathology, tempered these findings. They found that although there was a link between victimization and subsequent distress, part of this association might be due to confounding by premorbid functioning, which puts children at risk for both victimization and later symptoms.

Similar to many of the child abuse studies, studies that explored the relationship between community violence and mental health outcomes found that violence-exposed children were more likely to report anxious and depressive symptoms than children unexposed to violence.[20–22] Cammack et al.,[16] for example, found that both perceptions of the frequency of violence in the community and direct witnessing of community violence were associated with anxious and depressive symptoms by age 12.

Exposure to multiple forms of violence over time had a particularly strong influence on psychopathology. Margolin et al.[25] found that a one-point increase on a cumulative violence exposure index that summed exposure to parent-to-youth aggression, marital physical aggression, and community violence over time was associated with increased risk of more than 50% for meeting criterion levels of depressive symptoms and anxiety by age 12. Ethier et al. found that chronic maltreatment in early childhood had a stronger relationship with depression in late childhood than transitory maltreatment.[18]

Three studies investigated mediators of the relationship between violence exposure and psychopathology. One study considered PTSD as a potential mediator, and found that it fully mediated the relationship between exposure to community violence in early adolescence and anxiety and depression in middle adolescence in girls.[31] Bailey et al. reported that behavioral undercontrol mediated the relationship between child sexual abuse and substance use by age 17.[12] Schilling found that among females, two-thirds of the effect of sexual abuse on depressive symptoms in young adulthood was mediated by employment status, disruption/conflict, and opportunities, education status, education disruption/conflict, and relationship status and conflict/disruption.[32]

ADULT VIOLENCE

Adult physical and sexual abuse was also associated with later negative mental health outcomes.[39–43] A large population-based study in Finland found that violence was related to anxiety and mental distress 1 year later.[41] Another study that examined the relationship between violence and mental health for impoverished women who were victimized between baseline and follow-up, found that they were two times more likely to be depressed at follow-up than their nonvictimized counterparts.[46]

In cases of child and adult violence, different patterns of psychopathology emerged based on the type of violence experienced. Johnson et al.[20] found that victimization predicted depression whereas witnessed violence predicted both DAS during childhood. Mrug et al.[28] showed that victimization and witnessing violence at home and school (rather than the community) had the strongest effects on internalizing outcomes in early adolescence. In adulthood, a study of participants in an outpatient substance abuse treatment facility[44] indicated that for women, sexual abuse in childhood or adulthood was a greater predictor of adult DAS than physical abuse. For men, sexual abuse was associated only with anxiety while physical abuse was associated with DAS. A female cohort study found that among different types of violence, intimate partner violence (IPV) was the strongest predictor of adult depression.[42]

DISCUSSION

Exposure to physical/sexual abuse, home, school, and community violence in childhood, and IPV in adulthood, is consistently associated with depression, anxiety, and substance abuse. Existing research on the influence of violence on psychopathology has three strengths. First, studies increasingly examine the relative influence of different types of violence exposures, and of acute versus cumulative violence exposures, on different domains of psychopathology. Witnessing and victimization in the home and school environments is particularly associated with psychopathology, relative to community violence; and accumulation of violence across different domains and over time contributes to more psychopathology than acute violent exposures. Second, studies increasingly attempt to address potential confounders of the association between violence exposure and psychopathology, particularly the contribution of previolence psychopathology to the association. Third, studies have examined the long-term influences of violence, particularly investigating the influence that child abuse has on psychopathology by young adulthood, thus providing preliminary evidence of the role that early-life violence plays in shaping the development of psychopathology.

STUDY LIMITATIONS

Despite such advances, important limitations remain in studies investigating the mental health consequences of violence. Notably, we know very little about the relative influence of violence in key life stages (i.e. childhood, adolescence, adulthood) in the onset, escalation, persistence, and desistance of psychiatric disorders. Further, the bulk of research focuses on childhood violence exposures, providing little information on the role that adult violence plays in psychopathology. Most research focuses on affective disorders, providing little information on the relationship between violence exposures and substance abuse. Beyond substantive issues, important methodological limitations affect the types of conclusions we can draw from this research. Issues in study design, sampling, measurement, causal inference, and consideration of developmental trajectories of violence and psychopathology are reviewed below, providing illustrations from our literature review.

Study design—The most important limitation relates to study design. The ideal design, given that it is not possible to randomize violence exposure, is the prospective cohort study, which samples a disorder-free population on the basis of exposure, and follows them into the future to observe the outcome.[47] Our systematic review identified 35 longitudinal studies on the relationship between violence and depression, anxiety, or substance abuse, and of those, only six are actually prospective[13,17,19,26,27,45]—that is, they have pre- and postviolence measures on psychiatric conditions. Postviolence longitudinal studies offer an advantage over cross-sectional designs, since they allow us to assess heterogeneity in the long-term trajectories of psychopathology following violence exposure, as well as the role

that postevent conditions play in recovery. However, postevent longitudinal studies may still be confounded by previolence psychiatric status, which makes it difficult to assess the independent contribution of violence to psychopathology. A history of psychopathology, including depression, anxiety, and substance disorders, predicts increased risk of violence exposure including peer victimization, maltreatment, and sexual victimization,[13,17,48] as well as future instances of psychopathology.

Sampling—Sampling concerns also arise in the study of violence and psychopathology, including issues of sampling bias, selection bias, and lack of an adequate unexposed group. In our review, two types of sampling bias, or systematic exclusion of certain sectors of the intended population, were present: (1) sampling respondents from a group of individuals who were all violence exposed to different degrees[18,21,22] (e.g. respondents were all maltreated children recruited from a program addressing issues of child neglect); and (2) sampling respondents by their psychiatric conditions[15,23,44,45] (e.g. respondents had all been admitted to the hospital with a major depressive episode). Samples defined by violence exposure do not allow us to actually assess the impact of violence on psychopathology, as limited variability exists in the level of violence exposure in such a sample. In a similar way, treatment samples defined by psychiatric problems are a self-selected population that exhibit particularly high levels of psychopathology, and thus only provide information about the impact of violence in the most severe cases of psychopathology who actually seek treatment.

We found instances of selection bias due to sampling respondents by a third selection condition related to convenience of sample access[35,40,43,46] (e.g. respondents were HIV positive women recruited from HIV primary care clinics). Such sampling may create selection bias if individuals are selected based on a condition that is related to violence and to the psychiatric disorder of interest: for example, a study may compare psychopathology among emergency room attendees, by type of trauma they experienced (e.g. violence, falls, motor vehicle injury). In such a case, the risk for presenting to the emergency room with a source of injury-related trauma may be related both to the risk of violence exposure and to the risk of experiencing a psychiatric disorder. Even if violence and the psychiatric disorder are not associated in the general population, by selecting participants on a common effect (i.e. emergency room attendance), a statistical association is induced where a causal one does not exist—this is known as Berkson's bias.[49] In order to assess the relationship between violence exposure and psychopathology without facing issues of selection bias or external validity concerns, the best approach is to use a population-based sample. In our review, 19 studies used population-based samples.

Studies of the mental health consequences of violence exposure sometimes compare those exposed to a separately sampled unexposed control group. Five studies in our review used an exposed–unexposed control group design:[12,20,29,37,39] they obtained abused women or children through shelters, court records, or child protective agency referrals, and nonabused respondents from the community or other venues. There are several issues with the choice of an adequate unexposed control group. As explained by Briere,[50] the risk for victimization runs parallel to the risk for many other factors that may also influence the risk for psychopathology, such as low socioeconomic status, parental psychopathology, and history of psychopathology. Although matching on such potential confounders may address some of these concerns, the very fact of recruiting the unexposed group separately from the cases raises the question of whether they represent the same underlying population, and whether other types of unmeasured confounding may affect the estimated relationship.

Sample size—A related sample concern is small sample size. In this review, samples ranged from 45 to 9,682 (mean = 1,394; median = 603), with 16 out of 38 studies with a sample below 400. To provide a point of comparison, using a two-sided test at 0.05

significance level, and assuming a very simple longitudinal study design with three interviews over 3 years and no covariates, 400 respondents gives us 78% power to detect a moderate effect size of 0.3. Hence, sample sizes below 400 with fewer measurement frequencies raise concerns about study power. Many of these studies incorporate multiple covariates into the models and use complicated regression models with interactions, which raise the power limitations even further.

Measurement—Studies on violence exposure have three main measurement limitations. First, such studies often rely on retrospective reports of violence exposure. Of the studies in this review, 28 relied on such reports. Considerable controversy exists about the accuracy of such recall. For example, Henry et al. compared retrospective reports measured at age 18 about family conflict at ages 7–15 to mothers' reports of family conflict measured at ages 7–15, and correlations between the two ranged from 0.09–0.27, with a mean of 0.19.[51] A large body of literature has documented the phenomenon of mood-dependent memory whereby an individual's recall of experiences is affected by current psychological state.[52] Lewinsohn and Rosenbaum,[53] for example, found that retrospective recall of parental behavior was a function of their current depressive state. This creates a concern of recall bias in studies using retrospective recall of violent events.[54–60]

Measure construction directly relates to the accuracy of retrospective recall. Reliance on a single item to represent the construct, use of general questions about abuse, or use of a short checklist of violent events reduces rates of disclosure.[61–63] Reviewed studies used a variety of measures, ranging from court records, open-ended interviews, structured and detailed questionnaires, and a short and general set of questions. Prescott et al.[62] and Silvern[63] found that accuracy of recall was better for instruments that asked about exposure to specific abusive behaviors in specific contexts, rather than asked general questions about "being abused." Beyond the issue of accuracy of recall, short checklists may obscure important variability in terms of the nature of the violent event, the level of exposure, sources of exposure, and duration of exposure, all of which may influence the impact a violent event has on psychopathology.

A third measurement limitation concerns the way the exposure is actually modeled. Studies traditionally sum reports of different types of violent events to create un-weighted scales that do not account for the severity of different types of events.[64] Further, many such reports fail to account for other factors that may contribute to the impact of the violent exposure, including location and the characteristics of the victim and perpetrator.[65,66]

Causal inference—As with other types of epidemiologic studies, causal inference is an important issue in observational longitudinal studies of the mental health consequences of violence exposure.[67–69] There are some threats to causal inference that, while common to all epidemiologic studies, are particularly worth discussing in reference to the issue of violence and psychopathology—these are illustrated in Figure 1. First, selection by constitutional factors (e.g. temperament), individual history of psychopathology, or environmental factors, may present a particular concern. For example, disturbances in the parent–child relationship may make children symptomatic for depression and also put them at risk for victimization by reducing supervision, protection, or support.[70,71] Second, a cyclic relationship may exist between victimization and psychopathology, given the following conditions: (1) past experience of victimization increases one's risk for subsequent victimization;[72] (2) past experience of psychopathology strongly predicts later psychiatric problems[73–75] such that even early indications of psychopathology in childhood, such as withdrawal, inhibition, and aggression, may be early premorbid indicators of later psychopathology;[76–78] (3) violence exposure will influence the likelihood of future psychiatric problems, but at the same time, psychiatric problems may influence the risk of

victimization.[79,80] Even studies that have information on previolence psychopathology are often unable to capture such cyclic relations due to their reliance on deterministic models that estimate unidirectional relations. Hence, we are not able to state conclusively to what extent violence is a cause and not an effect of psychopathology. Third, time-dependent confounding may exist by factors that influence the likelihood that an individual will be victimized, and that are in turn also outcomes of violence exposure.[81] For example, residence in an economically disadvantaged neighborhood may (given the higher concentration of violence in such a neighborhood) increase the risk of victimization and also increase the risk of psychiatric problems such as depression, anxiety, or substance abuse.[82,83] At the same time, victimization may have an impact on financial well-being and hence on neighborhood location, which will in turn also influence psychopathology. In this case, level of neighborhood disadvantage is both a confounder of the victimization–psychopathology relation, as well as a mediator in the causal pathway between victimization and psychopathology. Thus, studies that attempt to control for a time-dependent confounder such as neighborhood disadvantage may be underestimating the potential impact of victimization on psychopathology, as they block the pathways connecting victimization to psychopathology that may be mediated by this covariate.

Limited consideration of such issues of causal inference is given in current studies on violence and psychopathology. In our review, 26 studies controlled, to varying degrees, for a range of confounders, such as pre-violence and comorbid psychopathology (e.g. prior depression; PTSD), demographic variables, and quality of social support (e.g. relationship with parents). But the issues of unobserved confounding, time-dependent confounding, and cyclical relations remain unaddressed.

Developmental trajectories of violence and psychopathology—Few studies have considered the relative influence of violent events at different developmental stages (e.g. childhood versus adolescence versus adulthood) on psychopathology, or have adequately characterized the different types of *longitudinal trajectories* of psychopathology that develop from childhood to adulthood in the aftermath of victimization. There are four important issues to consider here: (1) the importance of timing of violence exposure—that is, are there sensitive periods for the influence of violence exposure on psychopathology? (2) The relative influence of accumulated versus single violence exposures on psychopathology; (3) are there stage-specific manifestations of psychopathology after violence exposure? That is, individuals may exhibit patterns of "heterotypic continuity,"[84] so that, for example, childhood manifestations of aggression and internalizing symptoms in response to child abuse translate into drug abuse and proneness to revictimization in adolescence and young adulthood, and depression and substance dependence in adulthood; and (4) what range of responses do individuals have to the same type of violent exposure? In this review, some notable examples of a developmental approach include a study on the intergenerational transmission of abuse and its influence on offspring depression,[29] a study examining the relative influence of child sexual abuse and time-varying factors such as substance-using peers and cannabis/cigarette smoking on adolescent/young adult illicit drug use,[19] and a study examining the contribution of child maltreatment before age 11 (relative to other family and individual characteristics in childhood) on young adult depression.[27]

BUILDING ON INNOVATIONS FROM EPIDEMIOLOGY TO IMPROVE THE STUDY OF THE MENTAL HEALTH CONSEQUENCES OF VIOLENCE EXPOSURE

As the study of violence exposure and mental health evolves, advances in epidemiologic methods can provide insights into directions for future research. We discuss how innovations in epidemiologic study design, sampling, statistics, and causal inference can help guide forward the design of future studies on violence and psychopathology.

Study design—As discussed above, a need exists to invest in prospective study designs, with pre- and postviolence data on psychopathology, to be able to establish that violence exposure temporally precedes the onset of psychopathology and to assess the relationship between violence and psychopathology that does not reflect baseline psychiatric problems. Further, with prospective studies, if we observe that changes in violence predict changes in psychopathology, we can rule out concerns of unobserved confounding, provided those unobserved confounders have time-constant effects.[67] A birth cohort followed into adulthood offers two key advantages over other study designs: (1) it starts off with a population that is free of abuse and psychopathology; and (2) it makes it possible to document violence exposure and psychiatric problems at multiple stages of life. In the absence of such an option, a promising approach might be to select a type of cohort study of the age group closest to the type of violence of interest. Before embarking on new data collection efforts, it is worth considering that cohort studies have existed for many years that can provide a lot of the information we may need on the mental health consequences of violence. Notable cohorts include the Dunedin[27] and Christchurch[85] cohorts, the Great Smoky Mountain Study,[17] the Pittsburgh Youth Study,[86] and the Project on Human Development in Chicago Neighborhoods.[87,88]

Sampling—A concerted effort needs to be made, whenever possible, to invest in the selection of community population-based samples that do not suffer the problems of selection bias that treatment- or exposure-based samples exhibit. However, sometimes a community population-based sample will not provide sufficient numbers of people exposed to certain types of violence, such as combat experiences or exposure to a terrorist incident, that have a low prevalence in the general population. In such cases, several alternative options exist. First, “exposed-unexposed control” studies, where a sample of individuals exposed to the type of violence of interest is compared to a separately sampled control group of unexposed individuals, offer a promising alternative. The most important aspect to consider in such a design is the recruitment of the unexposed control group: does it represent the same population as the exposed group? One way to ensure this is to carry out a nested exposed–unexposed control group study: that is, obtaining the “exposed” from an existing cohort sample, and sampling the unexposed controls from the same sample, hence ensuring they come from the same underlying population—one study in our review followed this strategy.[20] A second way to ensure this is matching the exposed and unexposed so that they are comparable in potential confounders: Widom et al., for example, matched children with court-substantiated cases of child abuse with nonvictimized children, on age, sex, race/ethnicity, and approximate social class.[37,89] A second alternative to population-based samples is to sample within the population of interest: for example, to assess the impact of combat-related violence on psychopathology, prior studies have used military registries and compared soldiers with different levels of violence exposure[90]—although such a design poses issues of external validity, the issues of selection bias are avoided, since the comparison groups arise from the same underlying population.

Measurement—Several strategies can improve recall of violent events and reduce measurement bias. First, using multiple sources of information, such as self-reports and court or police records, is critical, as each source has its strengths and weaknesses that may compensate each other. Self-reports of violence exposure may suffer from recall bias given prior psychiatric disorder symptoms or incomplete recall over time due to forgetting, but can be asked of everyone in the population with critical details about place of occurrence, relationship to the perpetrator, and types of violent events. Criminal reports may be more objectives sources of violent exposures, but they undercount actual cases of violence, since violent events are notoriously underreported.[91] Second, “objective” measures of violence exposure may be developed. One approach is to directly observe conflict-laden interactions:

Moffitt et al., for example, ascertained maltreatment at age 3 using behavioral observations of rejecting mother–child interactions.[27] Another is to use archival sources to create spatial measures of violence exposure: to estimate the degree of exposure to community violence, for example, study respondents may be geocoded to their residence, and the average homicide rate in a network buffer (a network buffer reflects the area that a respondent can travel in all directions around his/her house in a given time)[92] can be estimated from archival sources. A combination of archival sources can also be used to construct more "objective" measures. Although we are not aware of any relevant applications in interpersonal violence, Dohrenwend's[93] study of war-related violence illustrates this point. He used a combination of military personnel files with information about the respondent's military occupational specialty, historical records of the monthly killed-in-action rate during the respondent's Vietnam service, and military archival sources on the killed-in-action rate in the respondent's larger military unit, to capture the level of war-zone stress suffered by Vietnam veterans in his study.

Psychometric methods can also be applied to obtain more accurate and precise estimates of violence exposure. Here we just discuss three among many different statistical approaches that could be exploited to improve violence exposure measures. First, item response theory (IRT), originally pioneered as a theory by Frederic Lord,[94] Georg Rasch,[95] and Lazarsfeld[96] in the 1950s and 1960s, offers a tool to estimate measures of violence exposure that are adequately weighted for the severity of each of the acts included in the scale.[97,98] IRT models have two parameters: item difficulty or severity (denoted by how frequently the item is endorsed) and subject ability. A continuous summary measure is obtained by modeling the conditional probabilities of responding yes to each violence question, given the severity of each question and the unobserved violence exposure level of each person. Although we did not find any applications of this technique in our review, they have been used in studies relating violence exposures to PTSD or to aggression.[97,98] In an application of IRT to a measure of children's exposure to community violence, Suglia et al.[98] found that the estimated score was more strongly related to posttraumatic stress symptoms than a simple sum of the scale items, indicating that accounting for differential severity of scale items and incorporating more detailed information on circumstances surrounding discrete violent events may reduce measurement error in the assessment of violence exposure.

Second, statistical models can also be used to incorporate information on violence exposure from multiple sources—for example, from parents, teachers, and children in the case of children's violence exposure. Horton et al. propose a series of likelihood-based latent variable models that use data from multiple informants to estimate a child's violence exposure.[99] These models characterize the relationship between informants and violence exposure using a latent variable predictor model, and make varying assumptions about the independence of the informants and the influence of one informant's responses on the other's. This type of approach allows us to consolidate information in the most efficient way, and empirically evaluate assumptions about how to best combine responses from different sources.

Third, we can use a Bayesian framework to improve the accuracy of violence exposure estimates, as it accommodates unobserved or missing variables and explicitly uses information from prior studies in model estimation. Examples exist of using Bayesian approaches to account for reporting bias[100] and differential and nondifferential misclassification on the measure of violence.[101] The issue of misclassification in violence exposure measures is an important problem: for example, victims of violence may underreport their exposure to violence due to fear of the perpetrator finding out about their report. With a Bayesian approach, we can use subjective prior information on at least some

of the parameters, such as the misclassification probabilities, in order to estimate and perform inference on the parameters. For example, Yu et al.[100] used a Bayesian approach to address bias in rates of rape and domestic violence in the National Crime Victimization Survey (NCVS), caused by privacy concerns in telephone interviews and lack of confidentiality of in-person interviews. The Bayesian model estimated probabilities for respondents' bias, taking into account known influential factors: that crime was not reported because it was a telephone interview, that rape was not reported because spouse was present, and that domestic violence was not reported because spouse was present. Rates of rape and domestic violence that were weighted by these probabilities were higher than unweighted rates.

Causal inference—As stated above, current studies on violence and psychopathology suffer from multiple limitations to causal inference, including issues of selection, bidirectional relations between violence exposure and psychopathology, and time-dependent confounding. We have already discussed how cohort studies can be designed to address issues of confounding. However, measures of violence and psychopathology are often rife with problems of reliability and validity, which pose limits on the ability to assess the relationship between change in violence exposure and change in psychopathology. Further, such designs are still subject to time-varying confounding, which cannot be entirely eliminated even if all known confounders have been measured, as there may be unknown confounders. Given that violence can obviously not be ethically randomized, a set of alternative quasiexperimental options exists to address concerns of measured and unmeasured threats to causal inference.

First, we can take advantage of naturally occurring changes in violence that are unrelated to potential confounders of the relationship between violence exposure and psychopathology to conduct a natural experiment. Departing from a potential outcomes framework, which considers the potential outcome an individual would have obtained if, contrary to fact, he/she had not been exposed to violence,[102,103] the validity of a natural experiment relies on a set of assumptions: (1) the exogenous assignment to change in violence is ignorable given a set of covariates (in other words, within strata, the groups are comparable on their potential outcomes); and (2) an individual's outcome does not depend on the intervention assignment to other individuals. Assumptions of ignorability given covariates are not dispensable; effect estimates depend centrally on the assumption that assignment of individuals to violence exposure is probabilistically like a randomized assignment scheme. A war or another natural disaster may serve as a natural experiment: for example, if the probability of evacuation to safer areas was randomly assigned, and hence the likelihood of exposure to war-related violence was random, we could compare change in psychopathology pre- and postevacuation between those who were evacuated and those who remained close to the conflict. Prior studies have used World War II as a natural experiment to study the health effects of trauma.[104] Natural experiments are particularly promising, as they take advantage of an exogenously generated change in violence and thus address problems of omitted variable bias. Although these examples cover topics outside the scope of a review on interpersonal violence, they serve to illustrate the ways exogenous events can be used to address unmeasured confounding.

Instrumental variables (IVs)[105] offer another promising strategy to address issues of selection. An IV is a variable that is thought to mimic the exposure allocation process in a randomized study—if an appropriate and valid instrument is found, the effects of measured and unmeasured confounding may be eliminated. A good instrument is strongly related to the actual exposure of interest, the relationship between the IV and the exposure is not confounded by other variables, and there is no correlation between the IV and other factors explaining the outcome. Mendelian randomization is a particular type of IV that uses a

genetic instrument.[106–109] The basic premise (as applied to our relationship of interest) is that there may be genetic variants that can alter the level of, or imitate the biological effects of, a potentially modifiable environmental exposure such as violence, and that has itself an effect on risk for psychopathology. The advantages of such an approach are that genetically determined differences remain constant if untreated, are not influenced by selection bias and reflect prolonged differences—hence, they are not susceptible to reverse causality, are not influenced by confounding, and are independent of short-term fluctuations. We could, for example, select a gene that influences aggression and use that as the IV for the effect of parental aggression on offspring psychopathology. Some potential issues with this approach include the need to ensure that there is a strong relationship between the genetic variant and aggression (or risk of victimization), that the gene does not act through other pathways connecting it to the outcome, and that no confounding of the genotype by linkage disequilibrium occurs.[106]

Often options such as IV or natural experiments are not available, or do not provide the level of detail on the progression of violence exposure and common psychopathology that is necessary to push the field forward. Even in cases when observational cohort studies remain the best option to examine the question of interest, an effort should be made to use statistical approaches such as propensity score matching and marginal structural models (MSMs) to address measured confounding. Propensity score matching of (in this case) victimized and nonvictimized pairs is a widely used tool to reduce extrapolation and establish a balance in observed time-stable characteristics between comparison groups that is probabilistically comparable to the balance achieved by randomization.[110,111] Propensity scores estimate the conditional probability of falling in the exposed versus the unexposed group given the covariates[110] and match exposed group subjects to unexposed whose estimated propensity scores are similar—within matched sets, baseline group differences average close to zero.[112] MSMs,[81] in contrast, are used to address problems of time-dependent confounding in studies that examine time-varying exposures. As discussed above, including a time-dependent confounder as a covariate in the outcome model is problematic, because since the confounder is also a mediator, one blocks part of the pathway connecting the exposure and the outcome. MSMs are accomplished by fitting a model of interest (e.g. the effect of violence on anxiety) to the observed data, but weighting the responses from each subject by the inverse of the probability of having his/her exposure history (e.g. violence exposure history) as a function of his/her covariate history. Assuming no unmeasured confounders, weighting creates a “pseudopopulation” in which confounders are no longer associated with the exposure (see[81] for a simple example of this approach). Although no studies have yet applied MSMs to the study of violence, applications in areas such as perinatal epidemiology,[113] HIV treatment,[114] and neighborhood effects on health[115] indicate that failure to appropriately account for time-dependent confounding may lead to reversed or at least underestimated reports of the causal relationships of interest. Future studies could use these types of methods to ensure that the comparison between exposed and unexposed individuals is actually conducted between respondents who are comparable on such previolence exposure factors as psychiatric function, family psychopathology, and temperament.

Although advances in study design and in analysis of observational studies have the potential to advance our understanding of the mental health consequences of violence exposure, they have a limited ability to incorporate changing relations between individuals, which may play a crucial role in the risk of violence exposure and the development of psychopathology, to consider the dynamic relations between violence exposure and multiple other causes of psychopathology across multiple levels, or to really account for the bidirectional relations between violence exposure and psychopathology. Hence, current analytical approaches may lead us to incorrectly estimate the “causal effect” of violent

victimization or the impact of changing violence. Complex systems approaches offer a way forward to address such limitations. Complex systems approaches are computational approaches that use computer-based algorithms to model dynamic interactions between individuals and between individuals and their environment.[116,117]

Agent-based models (ABMs), a type of complex system model in which agents with a defined set of features interact with each other and their environment according to initial preset rules, are receiving particular attention in epidemiology.[118] Interactions between individuals, their social networks, and the environments they are embedded in are explicitly modeled. Agents can also adapt their behavior to past experiences, changes in their environment and in their social networks, and can in turn also exert change in the networks and environments where they are embedded. With these features, we can use ABMs to model the reciprocal relations between violence and psychopathology, and the particular influence of social networks on exposure to and reaction to violence. ABMs are simulations based on probabilities estimated from population-based data—as we now have a plethora of studies on violence and psychopathology, we are at the perfect moment to use such data to inform the creation of these innovative complex systems approaches. For example, using ABM techniques, we found that while populations with higher levels of social ties were exposed to more traumas, both directly (reflecting the influence of the experience of trauma by network members) and indirectly (reflecting secondary traumas that occur when learning of traumas to family or friends), cumulative PTSD incidence over the course of the model remained lower in populations with higher levels of social ties, suggesting that the benefits of social ties outweigh the hazards associated with having a large social network.[119]

Developmental trajectories of violence and psychopathology—A life-course framework,[120] that seeks to understand the interplay of violence and psychopathology across the human life span, should guide study design, sampling, measurement, and analytic choices in the study of violence and psychopathology. Several elements constitute this framework. First, changes in violence exposure and in common psychopathology should be considered across long stretches of time, such as childhood to adulthood, rather than at specific periods such as marriage or entry into high school. Within such an approach, particular attention should be placed on timing of violence exposure and the development of disorders. This means focusing not only on the timing of onset, escalation, and desistance of psychopathology, but also on the cumulative, co-occurring or sequential contribution of different types of violence in different ages: that is, examining the links between child abuse, adolescent peer aggression and dating violence, and adult IPV and criminal behavior, and their separate and linked relationship with psychopathology. Second, studies should consider variation in the course of psychopathology after violence exposure. Marked heterogeneity exists in individual responses to the same event. Individuals may exhibit a broad range of responses to violence, including resilience, or successful adaptation after violence,[121–127] "resistance," whereby dysfunction is minimal,[123] "recovery," in which a period of distress and dysfunction lasting several months or longer is followed by a gradual return to previolence levels of functioning,[128] "chronic dysfunction," in which violence-related psychopathology persists for years in affected persons,[129–137] delayed dysfunction after violence,[123,138] and relapse/remission, in which dysfunction displays a cyclical course, perhaps influenced by ongoing stressful circumstances.[123] Third, violence exposure should be considered in relation to the development of multiple comorbid disorders over time, rather than single isolated psychiatric problems. A substantial overlap has been found in depression, anxiety, and substance abuse in the aftermath of violence.[17,38,139–145] Yet, little is understood about how violence modifies the *developmental trajectories* of single and comorbid disorders.

Time-sensitive modeling techniques that are able to incorporate multiple interacting factors across long periods of time are critical in understanding the complexity of the relationship between violence and psychopathology from early development to the end stages of life. First, person-centered statistical techniques such as latent growth analysis (LCA) and growth mixture modeling (GMM) can group people with common experiences based on their responses to observed variables of interest:[16,146–150] such techniques can be used to examine common longitudinal patterns of violence exposure at different time points, as well as common trajectories of psychopathology. For example, Nooner et al.[151] used LCA to identify meaningful groups of at-risk preadolescent youth based on their self-report of physical and sexual abuse. They found four classes, based on the responses to specific items: (1) no physical or sexual abuse; (2) high physical abuse/low sexual abuse; (3) no physical abuse/moderate sexual abuse; and (4) high physical and sexual abuse. Hence, the LCA approach permitted the identification of groups that accounted for the occurrence of multiple types of abuse as well as differing severities associated with each type of abuse. GMMs can also be used to examine heterogeneous mental health trajectories following violence exposure. Although we did not find any studies that examined heterogeneous depressive, anxious, or substance abuse trajectories following violence exposure, a similar example illustrates the method's utility. Cote et al. used GMMs to model the trajectories of DAS in early childhood.[78] They identified three DAS trajectories: high-rising DAS symptoms (14.7%), moderate-rising (55.4%), and low symptoms (29.9%). Difficult temperament at 5 months and maternal lifetime depression distinguished the high-rising group from the other two, while high family dysfunction and low parental self-efficacy distinguished the high-rising group from the low group. One may imagine also using these methods to estimate the different patterns of violence exposure over the life course, distinguishing, for example, between individuals with no experience of violence, those with an isolated period of child abuse, others with exposure solely to IPV in adulthood, and others exposed to multiple sources of violence across their life. After taking an empiric approach to identifying patterns of long-term violence exposure, one may then relate such patterns to different types of psychiatric problems. These methods thus help us identify how different types of violence may shape psychopathology, and how violence, in combination with other factors, may differentiate different psychiatric trajectories from one another.

The life-course approach advocates identifying chains of risk to better understand the etiology of disease. This approach seems highly relevant to the study of violence and psychopathology, since risk factors such as violence, association with deviant peers, and parenting styles are highly correlated and may be associated in a causal manner themselves. Structural equation models (SEMs) can highlight direct and indirect associations between multiple variables and offer a promising tool to examine the bidirectional pathways connecting violence and psychopathology, as well as the dynamics between violence and other types of exposures that contribute to psychopathology. A few studies in our review used SEM to examine the indirect relations between violence and psychopathology.[12,13,30,31,41] Ruchkin et al.,[31] for example, used SEM to examine the mediating role of post-traumatic stress in the relationship between violence exposure and psychopathology, using data from a sample of 1,358 urban adolescents. Investing in techniques such as SEM that allow us to examine the mechanisms through which violence exposure shapes psychopathology, will be key to advancing our understanding of the causal nature of the relationship between violence and psychopathology.

PUTTING IT ALL TOGETHER: WHERE DO WE GO FROM HERE

Although everyone would love to have access to a birth cohort with rich multisource data on violence exposures and psychopathology at multiple time points, such options are rarely available. Hence, we have proposed alternative designs, such as postviolence longitudinal

designs, exposed–unexposed control studies, and natural experiments. Table 2 attempts to tie together the recommendations just discussed, by presenting each of the proposed study designs and sampling approaches, the types of measures of violence exposure that can be used in each case, statistical approaches to address limitations to each measurement choice, issues of causal inference inherent to each design, and analytic options to address limitations to causal inference and to examine the violence exposure and psychopathology relationship.

The first option is to carry out a prospective cohort study, with pre- and postviolence measures of psychopathology. Such a design offers several options for measurement of violence exposure, including (1) retrospective self-reports of violence, which we can use to create an IRT score of violence exposures, (2) a combination of self-report and official records of abuse, which we can empirically combine with latent variable models; (3) geospatial measures of exposure in the case of community violence; and (4) direct observation of violent situations. These studies suffer from a number of limitations to causal inference, including unobserved and observed confounding, time-dependent confounding, bidirectional relations between violence and psychopathology, and the need to assess heterogeneity of responses due to modifiers. Propensity score matching can address issues of observed confounding. Researchers should check to see if there is time-dependent confounding, and in that case, using MSMs to weight the outcome models by the inverse probability of violence exposure will address this issue. Analytic models to estimate the violence exposure–psychopathology relation depend on the question of interest: (1) if the interest lies in assessing the bidirectional relations between violence and psychopathology exhibited by individuals in nested systems of influence, complex systems approaches, using probabilities estimated from the cohort data to inform the simulations, are a promising approach; (2) if the interest is in examining mediation, SEMs may be used; and (3) if the interest lies in examining the heterogeneity of pathways of response to exposure to violence, latent variable and growth mixture models are a promising option.

A second, less ideal option is to use a postviolence longitudinal study. The same measurement choices exist here, except that direct observation of potentially violent situations is obviously not possible. An additional concern about confounding by previolence psychopathology exists. This can be partly addressed by using retrospective approaches to assess date of onset of the psychiatric disorders to track any previolence psychopathology. Propensity score matching can be used to address fixed confounding, but the absence of time-varying measures of violence exposure makes it impossible to address issues of time-dependent confounding. Further, the absence of previolence measures makes it necessary to rely on retrospective measures of confounders, which adds to the concern about recall bias and the consequent limitations to causal inference.

A third option if the first two are not possible is to carry out an exposed–unexposed control group study. Here there are two choices: selecting the exposed and unexposed groups from the same population, which is preferable, or using matching to select an adequate control group. With both of these approaches, a mix of official and self-report information is usually available and actually used to select the exposed group members. The nested exposed-unexposed control study selected the unexposed from the same population as the exposed and is comparable to a post violence longitudinal study. In contrast, the matched exposed-unexposed group studies have their own particular issues. In such studies, there is an additional concern about measured and unmeasured differences between the comparison groups. These can be partly addressed through approaches like propensity score matching, but that only addresses retrospective, measured differences between the groups.

A fourth option that offers many advantages is the natural experiment. With this design it is more plausible to assume, given the exogenous source of violence exposure, that there are

no unmeasured differences between the exposed and unexposed. Although its non-random design raises concerns about residual confounding, we can test whether the degree of balance between the comparison groups is probabilistically comparable to that achieved under randomization. In the case that nonrandom imbalance remains, propensity score matching can be used to match exposed to unexposed subjects. Difference-in-difference models, which compare the change in psychopathology in the exposed to violence to the change in psychopathology in the unexposed, further address unobserved, time-constant sources of confounding.

IVs offer a final option that also addresses concerns about observed and unobserved confounding. The challenge is to actually find an instrument that is strongly associated with the violence exposure and only associated with psychopathology through the violence exposure—something that is not easy to achieve. Tests such as *F*-tests and *r*-squared coefficients can be used to assess the strength of the association between the instrument and the violence exposure. Specific analytic methods are used with IV studies, such as two-stage least squares, which first estimate the violence exposure as predicted by the instrument, and then estimate the relationship between the predicted score for the violence exposure (obtained from the first model) and the psychopathology outcome. The direct effect of violence exposure is a ratio of the coefficient denoting the association between the IV and the outcome, and the coefficient denoting the association between the IV and the actual violence exposure.

CONCLUSION

We have reviewed existing longitudinal studies examining the relationship between exposure to violence and common psychopathology, including depression, anxiety, and substance abuse. A consistent relationship has been found between violence exposure, particularly taking place in the home and school, and these forms of psychopathology. Given the magnitude of the literature on violence and psychopathology (at our count, 499 studies), a surprisingly small number (35) have used a longitudinal study design, and even fewer have used a population-based sample. A few investigators outside our review have attempted to address violence exposure measurement issues with item response modeling and Bayesian techniques, and have applied sophisticated approaches to examine developmental trajectories of psychopathology following violence exposure. Important limitations remain in study design, sampling, measurement, causal inference, and in the use of a developmental framework to examine this relationship. We propose a framework for future research, drawing on recent methodological innovations in epidemiology.

REFERENCES

1. Krug, E. World Report on Violence and Health. World Health Organization; Geneva, Switzerland: 2002.
2. McLaughlin KA, Green JG, Gruber MJ, et al. Childhood adversities and adult psychiatric disorders in the national comorbidity survey replication II: associations with persistence of DSM-IV disorders. Arch Gen Psychiatry. 2010; 67(2):124–132. [PubMed: 20124112]
3. Green JG, McLaughlin KA, Berglund PA, et al. Childhood adversities and adult psychiatric disorders in the national comorbidity survey replication I: associations with first onset of DSM-IV disorders. Arch Gen Psychiatry. 2010; 67(2):113–123. [PubMed: 20124111]
4. Finkelhor D, Turner H, Ormrod R, Hamby SL. Trends in childhood violence and abuse exposure: evidence from 2 national surveys. Arch Pediatr Adolesc Med. 2010; 164(3):238–242. [PubMed: 20194256]
5. Howard LM, Trevillion K, Agnew-Davies R. Domestic violence and mental health. Int Rev Psychiatry. 2010; 22(5):525–534. [PubMed: 21047164]

6. O'Donnell ML, Creamer M, Pattison P, Atkin C. Psychiatric morbidity following injury. Am J Psychiatry. 2004; 161(3):507–514. [PubMed: 14992977]
7. Perepletchikova F, Kaufman J. Emotional and behavioral sequelae of childhood maltreatment. Curr Opin Pediatr. 2010; 22:610–615. [PubMed: 20736837]
8. Kessler RC, Berglund P, Demler O, et al. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Arch Gen Psychiatry. 2005; 62:593–602. [PubMed: 15939837]
9. Galea S, Maxwell AR, Norris F. Sampling and design challenges in studying the mental health consequences of disasters. Int J Methods Psychiatr Res. 2008; 17(Suppl 2):S21–S28. [PubMed: 19035439]
10. Kessler RC, Keane TM, Ursano RJ, et al. Sample and design considerations in post-disaster mental health needs assessment tracking surveys. Int J Methods Psychiatr Res. 2008; 17(Suppl 2):S6–S20. [PubMed: 19035440]
11. DiGangi J, Koenen KC, Jason L, Keys C. Prospective longitudinal studies of posttraumatic stress disorder: a systematic review of the literature. Compr Psychiatry. 2011; 52:e4–e5.
12. Bailey J, McCloskey L. Pathways to adolescent substance use among sexually abused girls. J Abnorm Child Psychol. 2005; 33(1):39–53. [PubMed: 15759590]
13. Boney-McCoy S, Finkelhor D. Is youth victimization related to trauma symptoms and depression after controlling for prior symptoms and family relationships? A longitudinal, prospective study. J Consult Clin Psychol. 1996; 64(6):1406–1416. [PubMed: 8991327]
14. Brown J, Cohen P, Johnson JG, Smailes EM. Childhood abuse and neglect: specificity of effects on adolescent and young adult depression and suicidality. J Am Acad Child Adolesc Psychiatry. 1999; 38(12):1490–1496. [PubMed: 10596248]
15. Buist A, Janson H. Childhood sexual abuse, parenting and postpartum depression—a 3-year follow-up study. Child Abuse Negl. 2001; 25(7):909–921. [PubMed: 11523868]
16. Cammack N, Lambert S, Ialongo N. Discrepancies between community violence exposure and perceived neighborhood violence. J Community Psychol. 2010; 39(1):106–120.
17. Copeland WE, Keeler G, Angold A, Costello EJ. Traumatic events and posttraumatic stress in childhood. Arch Gen Psychiatry. 2007; 64(5):577–584. [PubMed: 17485609]
18. Ethier LS, Lemelin JP, Lacharite C. A longitudinal study of the effects of chronic maltreatment on children's behavioral and emotional problems. Child Abuse Negl. 2004; 28:1265–1278. [PubMed: 15607769]
19. Fergusson D, Boden J, Horwood L. The developmental antecedents of illicit drug use: evidence from a 25-year longitudinal study. Drug Alcohol Depend. 2008; 96:165–177. [PubMed: 18423900]
20. Johnson RM, Kotch JB, Catellier DJ, et al. Adverse behavioral and emotional outcomes from child abuse and witnessed violence. Child Maltreat. 2002; 7(3):179–186. [PubMed: 12139186]
21. Kennedy AC, Bybee D, Sullivan CM, Greeson M. The effects of community and family violence exposure on anxiety trajectories during middle childhood: the role of family social support as a moderator. J Clin Child Adolesc Psychol. 2009; 38:365–379. [PubMed: 19437297]
22. Kennedy AC, Bybee D, Sullivan CM, Greeson M. The impact of family and community violence on children's depression trajectories: examining the interactions of violence exposure, family social support, and gender. J Fam Psychol. 2010; 24(2):197–207. [PubMed: 20438195]
23. Klein DN, Shankman SA, Rose S. Dysthymic disorder and double depression: prediction of 10-year course trajectories and outcomes. J Psychiatr Res. 2008; 42(5):408–415. [PubMed: 17466334]
24. Latzman R, Swisher R. The interactive relationship among adolescent violence, street violence and depression. J Community Psychol. 2005; 33:355–371.
25. Margolin G, Vickerman KA, Oliver PH, Gordis EB. Violence exposure in multiple interpersonal domains: cumulative and differential effects. J Adolesc Health. 2010; 47:198–205. [PubMed: 20638013]
26. Mason WA, Kosterman R, Hawkins JD, et al. Predicting depression, social phobia, and violence in early adulthood from childhood behavior problems. J Am Acad Child Adolesc Psychiatry. 2004; 43(3):307–315. [PubMed: 15076264]

27. Moffitt TE, Caspi A, Harrington H, et al. Generalized anxiety disorder and depression: childhood risk factors in a birth cohort followed to age 32. Psychol Med. 2007; 37:441–452. [PubMed: 17201999]
28. Mrug S, Windle M. Prospective effects of violence exposure across multiple contexts on early adolescents' internalizing and externalizing problems. J Child Psychol Psychiatry. 2010; 51(8): 953–961. [PubMed: 20331489]
29. Noll J, Trickett P, Harris W, Putnam F. The cumulative burden borne by offspring whose mothers were sexually abused as children: descriptive results from a multigenerational study. J Interpers Violence. 2009; 24(3):424–449. [PubMed: 18451100]
30. Roberts R, O'Connor T, Dunn J, Golding J. The effects of child sexual abuse in later family life; mental health, parenting and adjustment of offspring. Child Abuse Negl. 2004; 28(5):525–545. [PubMed: 15159068]
31. Ruchkin V, Henrich C, Jones S, et al. Violence exposure and psychopathology in urban youth: the mediating role of posttraumatic stress. J Abnorm Child Psychol. 2007; 35(4):578–593. [PubMed: 17333360]
32. Schilling EA, Aseltine RH, Gore S. Adverse childhood experiences and mental health in young adults: a longitudinal survey. BMC Public Health. 2007; 7:10. [PubMed: 17241463]
33. Silverman AB, Reinherz HZ, Giaconia RM. The long-term sequelae of child and adolescent abuse: a longitudinal community study. Child Abuse Negl. 1996; 20(8):709–723. [PubMed: 8866117]
34. Vaske J, Makarios M, Boisvert D, et al. The interaction of DRD2 and violent victimization on depression: an analysis by gender and race. J Affect Disord. 2009; 112(1–3):120–125. [PubMed: 18501970]
35. Wang Z, Inslicht SS, Metzler TJ, et al. A prospective study of predictors of depression symptoms in police. Psychiatry Res. 2010; 175(3):211–216. [PubMed: 20044144]
36. White K, Bruce S, Farrell A, Kliewer W. Impact of exposure to community violence on anxiety: a longitudinal study of family social support as a protective factor for urban children. J Child Fam Stud. 1998; 7(2):187–203.
37. Widom CS, DuMont K, Czaja SJ. A prospective investigation of major depressive disorder and comorbidity in abused and neglected children grown up. Arch Gen Psychiatry. 2007; 64(1):49–56. [PubMed: 17199054]
38. Wu P, Bird HR, Liu XH, et al. Childhood depressive symptoms and early onset of alcohol use. Pediatrics. 2006; 118(5):1907–1915. [PubMed: 17079561]
39. Blasco-Rios C, Sanchez-Lorente S, Martinez M. Recovery from depressive symptoms, state anxiety and post-traumatic stress disorder in women exposed to physical and psychological, but not to psychological intimate partner violence alone: a longitudinal study. BMC Psychiatry. 2010; 10(98):1–12. [PubMed: 20055988]
40. Hogben M, Gange S, Watts D, et al. The effect of sexual and physical violence on risky sexual behavior and STDs among a cohort of HIV seropositive women. AIDS Behav. 2001; 5(4):353–361.
41. Kivimaki M, Vahtera J, Elovainio M, et al. Death or illness of a family member, violence, interpersonal conflict, and financial difficulties as predictors of sickness absence: longitudinal cohort study on psychological and behavioral links. Psychosom Med. 2002; 64(5):817–825. [PubMed: 12271113]
42. Taft AJ, Watson LF. Depression and termination of pregnancy (induced abortion) in a national cohort of young Australian women: the confounding effect of women's experience of violence. BMC Public Health. 2008; 8:75. [PubMed: 18302736]
43. von Eye A, Bogat G. Mental health in women experiencing intimate partner violence as the efficiency goal of social welfare functions. Int J Soc Welf. 2006; 15(1):531–540.
44. Gil-Rivas V, Fiorentine R, Anglin MD, Taylor E. Sexual and physical abuse: do they compromise drug treatment outcomes? J Subst Abuse Treat. 1997; 14(4):351–358. [PubMed: 9368211]
45. Gil-Rivas V, Prause J, Grella CE. Substance use after residential treatment among individuals with co-occurring disorders: the role of anxiety/depressive symptoms and trauma exposure. Psychol Addict Behav. 2009; 23(2):303–314. [PubMed: 19586147]

46. Rayburn NR, Wenzel SL, Elliott MN, et al. Trauma, depression, coping, and mental health service seeking among impoverished women. J Consult Clin Psychol. 2005; 73(4):667–677. [PubMed: 16173854]
47. Rothman, K.; Greenland, S.; Poole, C.; Lash, T. Causation and causal inference.. In: Rothman, K.; Greenland, S.; Lash, T., editors. Modern Epidemiology. Lippincott Williams & Wilkins; Philadelphia, PA: 2008.
48. Turner HA, Finkelhor D, Ormrod R. Child mental health problems as risk factors for victimization. Child Maltreat. 2010; 15(2):132–143. [PubMed: 19812391]
49. Berkson J. Limitations of the application of fourfold table analysis to hospital data. Biometrics. 1946; 2(3):47–53. [PubMed: 21001024]
50. Briere J. Methodological issues in the study of sexual abuse effects. J Consult Clin Psychol. 1992; 60(2):196–203. [PubMed: 1592948]
51. Henry B, Moffitt T, Caspi A, et al. On the “Remembrance of Things Past”: a longitudinal evaluation of the retrospective method. Psychol Assess. 1994; 6(2):92–101.
52. Cohen L, Towbes L, Flocco R. Effects of induced mood on self-reported life events and perceived and received social support. J Pers Soc Psychol. 1988; 55:669–674. [PubMed: 3193354]
53. Lewinsohn PM, Rosenbaum M. Recall of parental behavior by acute depressives, remitted depressives, and nondepressives. J Pers Soc Psychol. 1987; 52(3):611–619. [PubMed: 3572729]
54. Wessely S, Unwin C, Hotopf M, et al. Stability of recall of military hazards over time. Evidence from the Persian Gulf War of 1991. Br J Psychiatry. 2003; 183:314–322. [PubMed: 14519609]
55. Roemer L, Litz BT, Orsillo SM, et al. Increases in retrospective accounts of war-zone exposure over time: the role of PTSD symptom severity. J Trauma Stress. 1998; 11(3):597–605. [PubMed: 9690197]
56. David AC, Akerib V, Gaston L, Brunet A. Consistency of retrospective reports of peritraumatic responses and their relation to PTSD diagnostic status. J Trauma Stress. 23(5):599–605. [PubMed: 20839309]
57. King DW, King LA, Erickson DJ, et al. Posttraumatic stress disorder and retrospectively reported stressor exposure: a longitudinal prediction model. J Abnorm Psychol. 2000; 109(4):624–633. [PubMed: 11195986]
58. Southwick SM, Morgan CA 3rd, Nicolaou AL, Charney DS. Consistency of memory for combat-related traumatic events in veterans of Operation Desert Storm. Am J Psychiatry. 1997; 154(2): 173–177. [PubMed: 9016264]
59. Schwarz ED, Kowalski JM, McNally RJ. Malignant memories - posttraumatic changes in memory in adults after a school shooting. J Trauma Stress. 1993; 6(4):545–553.
60. White HR, Widom CS, Chen PH. Congruence between adolescents’ self-reports and their adult retrospective reports regarding parental discipline practices during their adolescence. Psychol Rep. 2007; 101(3):1079–1094. [PubMed: 18361122]
61. Bolen RM, Scannapieco M. Prevalence of child sexual abuse: a corrective metanalysis. Soc Serv Rev. 1999; 73(3):281–313.
62. Prescott A, Bank L, Reid JB. The veridicality of punitive childhood experiences reported by adolescents and young adults. Child Abuse Negl. 2000; 24(3):411–423. [PubMed: 10739084]
63. Silvern L, Waelde LC, Baughan BM, et al. Two formats for eliciting retrospective reports of child sexual and physical abuse: effects on apparent prevalence and relationships to adjustment. Child Maltreat. 2000; 5(3):236–250. [PubMed: 11232270]
64. Suglia SF, Ryan L, Wright RJ. Creation of a community violence exposure scale: accounting for what, who, where, and how often. J Trauma Stress. 2008; 21(5):479–486. [PubMed: 18956446]
65. Lynch M. Consequences of children's exposure to community violence. Clin Child Fam Psychol Rev. 2003; 6(4):265–274. [PubMed: 14719638]
66. Ward CL, Flisher AJ, Zissis C, et al. Exposure to violence and its relationship to psychopathology in adolescents. Inj Prev. 2001; 7(4):297–301. [PubMed: 11770655]
67. Glymour B, Glymour C, Glymour M. Watching social science—the debate about the effects of exposure to televised violence on aggressive behavior. Am Behav Sci. 2008; 51(8):1231–1259.

68. Rubin D. Estimating causal effects of treatments in randomized and nonrandomized studies. J Educ Psychol. 1974; 66(5):688–701.
69. Rubin D. Bayesian-inference for causal effects—role of randomization. Ann Stat. 1978; 6(1):34–58.
70. Mullen PE, Martin JL, Anderson JC, et al. Childhood sexual abuse and mental health in adult life. Br J Psychiatry. 1993; 163:721–732. [PubMed: 8306113]
71. Paradise JE, Rose L, Sleeper LA, Nathanson M. Behavior, family function, school performance, and predictors of persistent disturbance in sexually-abused children. Pediatrics. 1994; 93(3):452–459. [PubMed: 8115205]
72. Breslau N, Davis GC, Andreski P. Risk factors for PTSD-related traumatic events: a prospective analysis. Am J Psychiatry. 1995; 152:529–535. [PubMed: 7694900]
73. Breslau N, Peterson EL, Schultz LR. A second look at prior trauma and the posttraumatic stress disorder effects of subsequent trauma. Arch Gen Psychiatry. 2008; 65(4):431–437. [PubMed: 18391131]
74. Brewin C, Andrews B, Valentine J. Meta-anaysis of risk factors for post-traumatic stress disorder in trauma-exposed adults. J Consult Clin Psychol. 2000; 68:748–766. [PubMed: 11068961]
75. Norris FH, Friedman MJ, Watson PJ, et al. 60000 disaster victims speak: part I. An empirical review of the empirical literature, 1981–2001. Psychiatry. 2002; 65(3):207–239. [PubMed: 12405079]
76. Caspi A, Moffitt TE, Newman DL, Silva PA. Behavioral observations at age 3 years predict adult psychiatric disorders—longitudinal evidence from a birth cohort. Arch Gen Psychiatry. 1996; 53(11):1033–1039. [PubMed: 8911226]
77. Cote S, Zoccolillo M, Tremblay RE, et al. Predicting girls' conduct disorder in adolescence from childhood trajectories of disruptive behaviors. J Am Acad Child Adolesc Psychiatry. 2001; 40(6): 678–684. [PubMed: 11392346]
78. Cote SM, Boivin M, Liu XC, et al. Depression and anxiety symptoms: onset, developmental course and risk factors during early childhood. J Child Psychol Psychiatry. 2009; 50(10):1201–1208. [PubMed: 19519755]
79. Breslau N, Davis GC, Peterson EL, Schultz L. Psychiatric sequelae of posttraumatic stress disorder in women. Arch Gen Psychiatry. 1997; 54(1):81–87. [PubMed: 9006404]
80. Orcutt HK, Erickson DJ, Wolfe J. A prospective analysis of trauma exposure: the mediating role of PTSD symptomatology. J Trauma Stress. 2002; 15(3):259–267. [PubMed: 12092919]
81. Robins JM, Hernán MA, Brumback B. Marginal structural models and causal inference in epidemiology. Epidemiology. 2000; 11(5):550–560. [PubMed: 10955408]
82. Browning S, Erickson P. Neighborhood disadvantage, alcohol use, and violent victimization. Youth Violence Juv Justice. 2009; 7(4):331–349.
83. Kim J. Neighborhood disadvantage and mental health: the role of neighborhood disorder and social relationships. Soc Sci Res. 2010; 39(2):260–271.
84. Reef J, Diamantopoulou S, van Meurs I, et al. Child to adult continuities of psychopathology: a 24-year follow-up. Acta Psychiatr Scand. 2009; 120(3):230–238. [PubMed: 19522882]
85. Fergusson D, Horwood L. The Christchurch Health and Development Study: review of findings on child and adolescent mental health. Aust N Z J Psychiatry. 2001; 35:285–296.
86. Hoeve M, Blokland A, Dubas JS, et al. Trajectories of delinquency and parenting styles. J Abnorm Child Psychol. 2008; 36(2):223–235. [PubMed: 17786548]
87. Earls, F.; Buka, SL. Project on Human Development in Chicago Neighborhoods. National Institute of Justice; Washington, DC: 1997.
88. Molnar B, Buka S, Brennan R, et al. A multilevel study of neighborhoods and parent-to-child physical aggression: results from the Project on Human Development in Chicago Neighborhoods. Child Maltreat. 2003; 8(2):84–97. [PubMed: 12735711]
89. Widom, C. Understanding the consequences of childhood victimization.. In: Reece, R., editor. Treatment of Child Abuse. Johns Hopkins University Press; Baltimore: 2000.

90. Koenen KC, Stellman JM, Stellman SD, Sommer JF. Risk factors for course of posttraumatic stress disorder among Vietnam veterans: a 14-year follow-up of American Legionnaires. J Consult Clin Psychol. 2003; 71(6):980–986. [PubMed: 14622073]

91. Maxfield MG, Weiler BL, Widom CS. Comparing self-reports and official records of arrests. J Quant Criminol. 2000; 16(1):87–110.

92. Frank LD, Schmid TL, Sallis JF, et al. Linking objectively measured physical activity with objectively measured urban form—findings from SMARTRAQ. Am J Prev Med. 2005; 28(2): 117–125. [PubMed: 15694519]

93. Dohrenwend BP, Turner JB, Turse NA, et al. The psychological risks of Vietnam for US veterans: a revisit with new data and methods. Science. 2006; 313(5789):979–982. [PubMed: 16917066]

94. Lord, F. Applications of Item Response Theory to Practical Testing Problems. Routledge: 1980.

95. Rasch, G. Probabilistic Models for Some Intelligence and Attainment Tests. Danish Institute for Educational Research; Copenhagen: 1960.

96. Lazarsfeld, P.; Henry, N. Latent Structure Analysis. Houghton Mifflin; Boston: 1968.

97. Brennan RT, Molnar BE, Earls F. Refining the measurement of exposure to violence (ETV) in urban youth. J Community Psychol. 2007; 35:603–618.

98. Suglia SF, Ryan L, Wright RJ. Creation of a community violence exposure scale: accounting for what, who, where, and how often. J Trauma Stress. 2008; 21(5):479–486. [PubMed: 18956446]

99. Horton NJ, Roberts K, Ryan L, et al. A maximum likelihood latent variable regression model for multiple informants. Stat Med. 2008; 27(24):4992–5004. [PubMed: 18613227]

100. Yu QZ, Stasny EA, Li B. Bayesian models to adjust for response bias in survey data for estimating rape and domestic violence rates from the NCVS. Ann Appl Stat. 2008; 2(2):665–686.

101. Gerlach R, Stamey J. Bayesian model selection for logistic regression with misclassified outcomes. Stat Model. 2007; 7(3):255–273.

102. Rubin DB. Estimating causal effects of treatments in randomized and nonrandomized studies. J Educ Psychol. 1974; 66(5):688–701.

103. Rubin DB. Bayesian-inference for causal effects—role of randomization. Ann Stat. 1978; 6(1): 34–58.

104. Pesonen AK, Raikkonen K, Feldt K, et al. Childhood separation experience predicts HPA axis hormonal responses in late adulthood: a natural experiment of World War II. Psychoneuroendocrino. 2010; 35(5):758–767.

105. Bennett DA. An introduction to instrumental variables analysis: part 1. Neuroepidemiology. 2010; 35(3):237–240. [PubMed: 20861655]

106. Bennett DA. An introduction to instrumental variables—part 2: Mendelian randomisation. Neuroepidemiology. 2010; 35(4):307–310. [PubMed: 21042034]

107. Lawlor DA, Harbord RM, Sterne JAC, et al. Mendelian randomization: using genes as instruments for making causal inferences in epidemiology. Stat Med. 2008; 27(8):1133–1163. [PubMed: 17886233]

108. Davey Smith G, Ebrahim S. ‘Mendelian randomization’: can genetic epidemiology contribute to understanding environmental determinants of disease? Int J Epidemiol. 2003; 32(1):1–22. [PubMed: 12689998]

109. Davey Smith G, Leary S, Ness A, Lawlor DA. Challenges and novel approaches in the epidemiological study of early life influences on later disease. Adv Exp Med Biol. 2009; 646:1–14. [PubMed: 19536658]

110. Rosenbaum PR, Rubin DB. Reducing bias in observational studies using subclassification on the propensity score. J Am Stat Assoc. 1984; 79(387):516–524.

111. Stuart EA. Matching methods for causal inference: a review and a look forward. Stat Sci. 2010; 25(1):1–21. [PubMed: 20871802]

112. Rosenbaum, P. Observational Studies. 2nd ed.. Springer Verlag; 2002.

113. Bodnar L, Davidian M, Siega-Riz A, Tsiatis A. Marginal structural models for analyzing causal effects of time-dependent treatments: an application in perinatal epidemiology. Am J Epidemiol. 2004; 159(10):926–934. [PubMed: 15128604]

114. Hernán MA, Brumback B, Robins JM. Marginal structural models to estimate the causal effect of zidovudine on the survival of HIV-positive men. Epidemiology. 2000; 11(5):561–570. [PubMed: 10955409]
115. Cerda M, Diez-Roux AV, Tchetgen ET, et al. The relationship between neighborhood poverty and alcohol use: estimation by marginal structural models. Epidemiology. 2010; 21(4):482–489. [PubMed: 20498603]
116. Galea S, Hall C, Kaplan GA. Social epidemiology and complex system dynamic modelling as applied to health behaviour and drug use research. Int J Drug Policy. 2009; 20(3):209–216. [PubMed: 18930649]
117. Galea S, Riddle M, Kaplan GA. Causal thinking and complex system approaches in epidemiology. Int J Epidemiol. 2010; 39(1):97–106. [PubMed: 19820105]
118. Bonabeau E. Agent-based modeling: methods and techniques for simulating human systems. Proc Natl Acad Sci USA. 2002; 99(Suppl 3):7280–7287. [PubMed: 12011407]
119. Tracy, M.; Cerdá, M.; Galea, S. Causal thinking and complex systems approaches for understanding the consequences of trauma.. In: Widom, CS., editor. Trauma, Psychopathology and Violence: Causes, Consequences, or Correlates?. Oxford University Press; New York, NY: 2011.
120. Elder, G. Human lives in changing societies: life course and developmental insights.. In: Cairns, R.; Elder, G.; Glen, H., Jr., editors. Developmental Science. Cambridge University Press; New York: 1996.
121. Layne, CM.; Warren, JS.; Watson, PJ.; Shalev, AY. Risk, vulnerability, resistance, and resilience: toward an integrative conceptualization of post-traumatic adaptation.. In: Friedman, M.; Keane, T.; Resick, P., editors. Handbook of PTSD: Science and Practice. Guilford Press; New York: 2007. p. 497-520.
122. Norris FH, Stevens SP, Pfefferbaum B, et al. Community resilience as a metaphor, theory, set of capacities, and strategy for disaster readiness. Am J Community Psychol. 2008; 41(1–2):127–150. [PubMed: 18157631]
123. Norris FH, Tracy M, Galea S. Looking for resilience: understanding the longitudinal trajectories of responses to stress. Soc Sci Med. 2009; 68(12):2190–2198. [PubMed: 19403217]
124. Butler, L.; Morland, L.; Leskin, G. Psychological resilience in the face of terrorism.. In: Bongar, B.; Brown, L.; Beutler, L., et al., editors. Psychology of Terrorism. Oxford University Press; NY: 2007. p. 400-417.
125. Egeland B, Carlson E, Sroufe L. Resilience as process. Dev Psychopathol. 1993; 5:517–528.
126. Masten A, Best K, Garmezy N. Resilience and development: contributions from the study of children who overcome adversity. Dev Psychopathol. 1990; 2:425–444.
127. Werner, EE.; Smith, RS. Vulnerable but Invincible: A Longitudinal Study of Resilient Children and Youth. McGraw-Hill; New York: 1982.
128. Bonnano G. Loss, trauma, and human resilience: have we underestimated the human capacity to thrive after extremely aversive events? Am Psychol. 2004; 59:20–28. [PubMed: 14736317]
129. Kessler RC, Sonnega A, Bromet E, et al. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry. 1995; 52(12):1048–1060. [PubMed: 7492257]
130. Blanchard EB, Hickling EJ, Forneris CA, et al. Prediction of remission of acute posttraumatic stress disorder in motor vehicle accident victims. J Trauma Stress. 1997; 10(2):215–234. [PubMed: 9136089]
131. Green BL, Grace MC, Lindy JD, et al. Risk factors for PTSD and other diagnoses in a general sample of Vietnam veterans. Am J Psychiatry. 1990; 147(6):729–733. [PubMed: 2343915]
132. Havenaar JM, Rumyantzeva GM, van den Brink W, et al. Long-term mental health effects of the Chernobyl disaster: an epidemiologic survey in two former Soviet regions. Am J Psychiatry. 1997; 154(11):1605–1607. [PubMed: 9356574]
133. McFarlane AC, Papay P. Multiple diagnoses in posttraumatic stress disorder in the victims of a natural disaster. J Nerv Ment Dis. 1992; 180(8):498–504. [PubMed: 1500931]
134. North CS. The course of post-traumatic stress disorder after the Oklahoma City bombing. Mil Med. 2001; 166(12 Suppl):51–52. [PubMed: 11778434]

135. North CS, Tivis L, McMillen JC, et al. Psychiatric disorders in rescue workers after the Oklahoma City bombing. Am J Psychiatry. 2002; 159(5):857–859. [PubMed: 11986143]

136. Rothbaum B, Foa E, Riggs D, et al. A prospective examination of post-traumatic stress disorder in rape victims. J Trauma Stress. 1992; 5:455–475.

137. Shalev AY, Freedman S, Peri T, et al. Prospective study of post-traumatic stress disorder and depression following trauma. Am J Psychiatry. 1998; 155(5):630–637. [PubMed: 9585714]

138. Andrews B, Brewin CR, Philpott R, Stewart L. Delayed-onset posttraumatic stress disorder: a systematic review of the evidence. Am J Psychiatry. 2007; 164(9):1319–1326. [PubMed: 17728415]

139. de Graaf R, Bijl RV, ten Have M, et al. Pathways to comorbidity: the transition of pure mood, anxiety and substance use disorders into comorbid conditions in a longitudinal population-based study. J Affect Disord. 2004; 82(3):461–467. [PubMed: 15555699]

140. Hayatbakhsh MR, Najman JM, Jamrozik K, et al. Cannabis and anxiety and depression in young adults: a large prospective study. J Am Acad Child Adolesc Psychiatry. 2007; 46(3):408–417. [PubMed: 17314727]

141. de Graaf R, Bijl RV, ten Have M, et al. Rapid onset of comorbidity of common mental disorders: findings from the Netherlands Mental Health Survey and Incidence Study (NEMESIS). Acta Psychiatr Scand. 2004; 109(1):55–63. [PubMed: 14674959]

142. Windle M, Davies PT. Depression and heavy alcohol use among adolescents: concurrent and prospective relations. Dev Psychopathol. 1999; 11(4):823–844. [PubMed: 10624728]

143. Fergusson DM, Goodwin RD, Horwood LJ. Major depression and cigarette smoking: results of a 21-year longitudinal study. Psychol Med. 2003; 33(8):1357–1367. [PubMed: 14672244]

144. Hettema JM, Kuhn JW, Prescott CA, Kendler KS. The impact of generalized anxiety disorder and stressful life events on risk for major depressive episodes. Psychol Med. 2006; 36(6):789–795. [PubMed: 16515735]

145. Murray J, Farrington DP. Parental imprisonment: long-lasting effects on boys' internalizing problems through the life course. Dev Psychopathol. 2008; 20(1):273–290. [PubMed: 18211738]

146. Muthen B. Integrating categorical and dimensional approaches to substance use disorders. Alcohol Clin Exp Res. 2006; 30(6):271 A–271 A.

147. Muthén, B. Latent variable modeling with longitudinal and multilevel data.. In: Rafter, A., editor. Sociological Methodology. Blackwell Publishers; Boston, MA: 1997. p. 453-480.

148. Muthén, B.; Asparouhov, T. Beyond multilevel regression modeling: multilevel analysis in a general latent variable framework.. In: Hox, J.; Roberts, J., editors. The Handbook of Advanced Multilevel Analysis. Routledge Academic; 2010.

149. Muthen B, Muthen LK. Integrating person-centered and variable-centered analyses: growth mixture modeling with latent trajectory classes. Alcohol Clin Exp Res. 2000; 24(6):882–891. [PubMed: 10888079]

150. Nagin, DS. Group-Based Modeling of Development. Harvard University Press; Cambridge, MA: 2005.

151. Nooner KB, Litrownik AJ, Thompson R, et al. Youth self-report of physical and sexual abuse: a latent class analysis. Child Abuse Negl. 2010; 34(3):146–154. [PubMed: 20207000]

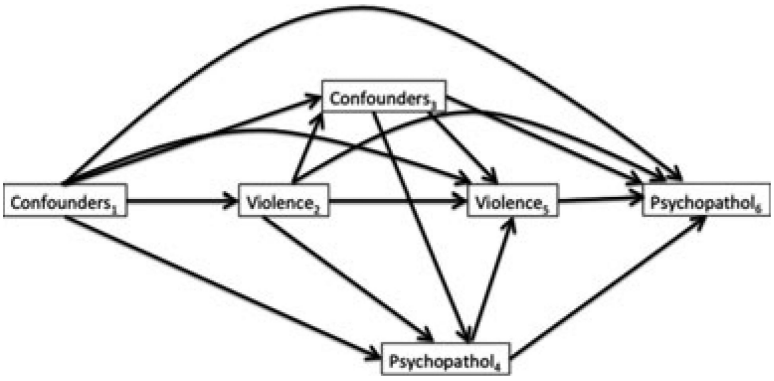

Figure 1.

Causal diagram of the hypothesized effects of violence on psychopathology at different times of life, where “confounders” denote potential individual (e.g. temperament, personality, IQ, genetic factors), family (e.g. adverse parent–child relations, low socioeconomic status), peer (e.g. association with deviant peers), and neighborhood (e.g. concentration of poverty, level of social cohesion, and control) characteristics that may operate as confounders of the association between early violence and psychopathology, mediators of the association between early violence and later psychopathology, and confounders of the association between later violence and psychopathology. “Modifiers” denote factors that predict heterogeneity of response to violence exposures and affect the likelihood that violence exposures will lead to psychopathology. Number subscripts are solely meant to denote ordering in time between time-varying confounders, violence exposures, and psychopathology.

TABLE 1

Longitudinal studies examining the relationship between violence exposures and depression, anxiety, or substance abuse, by age of violence exposure

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
Childhood exposure to violence							
Child abuse	Depression	Boney-McCoy and Finkelhor (1996)	1,433 children from the National Youth Victimization Prevention Study; aged: 10–16	Baseline: National random sample telephone survey, Tl: 15 months after telephone survey.	Diagnostic Interview Schedule (DIS)-III	PTSD Symptom Scale—Self-Report (PSSSR)	(1) Victimization between assessments was associated with depression measured at time 2, even after controlling for these symptoms and the quality of the parent–child relationship at baseline. (2) Some of the association found in cross-sectional studies between victimization and psychopathology may be due to prior psychopathology
Child abuse	Depression	Brown et al. (1999)	639 randomly selected cohort of children studied from 5 years to adulthood	Baseline: 1975, Tl: 1983; T2: 1986; T3: 1992.	DIS-C	Maltreatment data from the NY Central Registry for Child Abuse and Neglect	(1) Participants with a history of childhood maltreatment were three times more likely to become depressed compared with individuals without such a history. (2) Child sexual abuse had the strongest relationship with depression of all considered risk factors
Child abuse	Anxiety and depression	Copeland et al. (2007)	1,420 randomly selected cohort from the Great Smoky Mountains Study	Birth cohort study in which children aged 9, 11, and 13 were followed annually through 16.	Child and Adolescent Psychiatric Assessment (CAPA)	Child and Adolescent Psychiatric Assessment (CAPA)	(1)Children who were violence exposed had almost double the rates of psychiatric disorders by age 16 of those not exposed. (2)Higher levels of violence exposure were

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
							particularly related to anxiety and depressive disorders. (3) Past depression best predicted first violence exposure.
Child abuse	Anxiety and depression	Ethier et al. (2004)	49 maltreated preschoolers and school-aged children and their mothers, recruited from Child Protection Services; children's age: 50.6 months.	Baseline, Tl: 3 years after baseline, T2: 3 years after Tl.	Child Behavior Checklist (CBCL)	Child Abuse Potential Inventory	(1)Chronic maltreatment was significandy more related to anxiety and depression than transitory maltreatment. (2)Chronically maltreated children behaved more aggressively and socially withdrawn than the transitory group.
Child abuse	Depression and dysthymia	Klein et al. (2008)	87 outpatients from a naturalistic, prospective, longitudinal study with early onset dysthymic disorder; age 32.1 years	Participants assessed five times at 30-month intervals for 10 years.	Longitudinal Interval Follow-up Evaluation (LIFE); Hamilton Depression Rating Scale (HAM-D)	SCID; Early Home Environment Interview (EHEI)	Predictors of depression severity and functional impairment at 10-year outcome included a history of childhood sexual abuse.
Child abuse	Anxiety and depression	Moffitt et al. (2007)	1,037 children from the Dunedin Multidisciplinary Health and Development Study (New Zealand)	Birth cohort various assessments carried out at ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, and 32.	DIS	Behavioral observations of mother–child interactions at age 3, parental reports of harsh discipline at ages 7 and 9, 2+ changes in primary caregiver before age 11, and retrospective reports by the study members at age 26 of exposure to injurious physical abuse or unwanted sexual contact before age 11.	GAD, whether comorbid or pure, was associated with maltreatment (among other risk factors) whereas MDD in the absence of comorbid GAD was not strongly associated with risk factors during childhood.
Child abuse	Anxiety, depression	Silverman et al. (1996)	375 young adults from representative community sample; age: 21 years	Baseline: 1977 [age 5], Tl: 1987 [age 15], T2: 1993 [age 21].	Child Depression Inventory (CDI); Piers-Harris Self-Concept Scale; Youth Self-Report (YSR); Young Adult Self-Report (YASR)	Interview	(1) Compared to nonabused counterparts, abused individuals demonstrated significant impairments in functioning both at ages 15 and at 21,

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
							including more depressive symptoms and anxiety. (2) Females were almost three times more likely than males to experience any type of abuse and more than 11 times more likely than males to report sexual abuse.
Child abuse	Depression	Wang et al. (2010)	119 police recruits; age: 26.7 (4.1)	Baseline: Academy training, Tl: 12 months later.	Beck Depression Inventory (BDI-II)	Violence assessed within a set of traumatic events. PTSD Checklist (PCL-C); Critical Incident History Questionnaire (CIHQ); Life Experiences Survey (LES); Work Environment Inventory (WEI)	(1) Greater childhood trauma exposure predicted higher levels of depression symptoms at follow-up even after controlling for depression symptom levels during academy training and PTSD symptoms at 12-month follow-up. (2)Exposure to childhood trauma increased vulnerability to routine and traumatic stressors of police service.
Child abuse	Depression	Widom et al. (2007)	1,196 children abused (n = 520) and matched (on sex, race/ ethnicity, age, and socioeconomic status) nonabused children (n = 676); age: 28.7	Baseline: prior to age 11, Tl: average age 28.7	DIS	Court records	(1) Child abuse and neglect were associated with an increased risk for current MDD in young adulthood. (2) Physical abuse or multiple types of abuse increased risk of lifetime MDD, whereas neglect increased risk for current MDD. (3) Child sexual abuse (CSA) was not associated with elevated risk of MDD. (4) Earlier onset of MDD for abused and

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
							neglected children compared with controls. Among those with MDD, comorbidity was higher for abused and neglected individuals than for controls.
Child abuse—sexual	Anxiety and depression	Buist and Janson (2001)	45 women all of whom were admitted to an inpatient specialized psychiatric unit with postpartum depression.	Baseline, Tl: 3 years later.	HAM-D; BDI; Speilberger State/Trait Anxiety Scale (STAI)	Otago Women's Health Survey	(1) Women with a history of sexual abuse had higher depression and anxiety scores and had greater life stresses. (2) Over time, this sexually abused group's symptomatology failed to improve as much as the nonabused group.
Child abuse—sexual	Anxiety, depression and substance abuse	Noll et al. (2009)	84 sexually abused females referred by child protective services in DC matched to 82 controls recruited via ads in community venues; age: 24.89 (3.51)	Baseline, T1, T2, T3.	CDI; BDI; DSM-IV criteria also used to assess obsessive compulsive disorder, generalized anxiety disorder, panic disorder, or social phobia; SCID	Comprehensive Trauma Interview; Domestic Conflict Inventory; DSM-III PTSD criteria inventory	(1) Abused females were more likely than comparison females to have experienced at least one physical victimization, reached clinical cutoff for adult depression; be diagnosed with at least one psychiatric disorder; report substance dependence; report alcohol dependence. (2) Offspring born to abused mothers were more likely than offspring born to comparison mothers to have been born to a teen mother; have been born premature, and have been involved in child protective services.

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
Child abuse—sexual	Anxiety and depression	Roberts et al. (2004)	8,292 families from the Avon Longitudinal Study of Parents and Children an ongoing study of women and their families in Avon, England.	Baseline, Tl: 33 postnatal.	Edinburgh Post-Natal Depression Scale; Crown-Crisp Experiential Index	Self-report on sexual assault	Women who reported a history of CSA at baseline reported higher levels of depressive and anxious symptomatology 33 months postnatal.
Child abuse—sexual	Depression	Schilling et al. (2007)	1,093 students public school students in Boston; ages 16-20+ at baseline.	Baseline: 1998, Tl: 2000.	Center for Epidemiologic Studies Depression Scale (CES-D)	Interview	(1) Sexually abused women and men were more depressed during their senior year of high school, and this difference was more pronounced 2 years later. (2) Among females, two-thirds of the effect of abuse on depressive symptoms was mediated by experiences and choices in the domains of work, education, and intimate relationships.
Community violence	Aggressive behavior, anxiety, and depression	Cammack et al. (2011)	456 African Americans from community sample of middle school students; age: 11.77	Baseline: first grade, Tl: sixth grade	The Baltimore How I Feel-Adolescent Version	Children's Report of Violence Exposure; Neighborhood Environment Scale	(1) Both experienced (CVE) and perceived community violence (PNV) were associated with anxious, depressive, and aggressive symptoms. (2) After controlling for prior behaviors, youth with low CVE and high PNV reported more anxious and depressive symptoms and had higher teacher-reported aggressive behaviors than youth with low CVE and low PNV.

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
Community violence	Depression	Latzman and Swisher (2005)	8,939 youth from the National Longitudinal Study of Adolescent Health; age: 11–21	Baseline, Tl: 1 year.	Depression measured by an eight-item scale about the presence of positive and negative affect during the last week	Self-report on perpetration of six violent acts and exposure to four violent acts in past year	(1) Perpetration and victimization/ witnessing of street violence were associated with more adolescent depression; (2) the association between exposure to street violence and lower positive affect was lower for those reporting perpetration of violence; (3) these moderating effects were most pronounced among males and older adolescents.
Community violence	Depression and anxiety	Ruchkin et al. (2007)	1,488 adolescents from the Social and Health Assessment (SAHA); age: 12.4	Baseline, Tl: 2 years	Behavioral Assessment System for Children (BASC)	Survey of exposure to community violence: parent report version; two subsets of items, one assessing direct victimization and the other witnessing of community violence	(1) Posttraumatic stress fully mediated the relationships between victimization and depression and anxiety in girls, and partially so in boys. (2) Posttraumatic stress partially mediated the relationships between violence exposure and commission of violence in boys.
Community violence	Anxiety	White et al. (1998)	385 urban public school students; age: 12.3	Baseline: May 1996, Tl: November 1996.	Revised Children's Manifest Anxiety Scale (RCMAS)	Things I Have Seen and Heard	(1)After controlling for gender, exposure to violence at baseline did not significandy predict changes in anxiety. (2)Girls who reported higher initial violence exposure reported greater increases in subsequent concentration anxiety than boys.

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
Various (i.e. interparental violence; physical child abuse; CSA)	Substance abuse	Bailey and McCloskey (2005)	150 girls recruited along with their mothers from battered women's shelters or the community. Age: 9.2	Baseline: 1990–1991, Tl: 1996–1997, T3: 1998–1999.	Parent Report of CBCL; Rosenberg Self-Esteem Scale (RSES); CES-D	Open-ended questions about the child's sexual victimization	(1) Girls' childhood sexual abuse was associated prospectively with their later substance use. (2) Behavioral undercontrol mediated the relationship between childhood sexual abuse and later substance use, but depressive self-concept did not.
Various (i.e. CSA, physical abuse, interparental violence)	Substance abuse	Ferguson et al. (2008)	1,003 adults from Christchurch Health and Development Study (CHDS); age: 25.	Birth cohort, includes measures at ages 7–25	Self-reported frequency of drinking alcohol ranging from "never" to "almost every day"	Measures of parental adjustment and substance use.	(1) Child abuse was associated with illicit drug use and abuse/dependence from ages 16 to 25. (2) Pathways to illicit drug use and abuse/dependence were mediated via cannabis use, affiliation with substance-using peers, and alcohol use during ages 16–25.
Various (i.e. violence experienced or witnessed by the child in the home and community)	Depression and anxiety	Johnson et al. (2002)	70 children identified as under suspicion of maltreatment by social services were randomly selected from the Stress, Social Support and Abuse & Neglect in High Risk Infants Study and matched with two controls by age, socioeconomic status, race, and sex. Age: 6–8.	Baseline: age 6, Tl: age 8.	CBCL; Trauma Symptom Checklist for Children (TSCC)	Central Registry data prior to the age 8 interview; caregiver responses to the Conflict Tactics Scales (CTS); Things I've Seen and Heard; Child Life Events survey	(1) Victimization was a significant predictor of child depression and aggression. (2) Witnessed violence was found to be a significant predictor of aggression, depression, anxiety, and anger.
Various (i.e. intimate partner violence (IPV), community and school violence)	Anxiety	Kennedy et al. (2009)	100 children and their families recruited from a social services agency that	Baseline: 2 or 3 weeks after recruitment, Tl: 4 months after baseline,	RCMAS	4-item Child Report of Witnessing IPV scale; modified version of Things I Have Seen and Heard scale.	(1) Initial community and school violence exposure and witnessing IPV

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
			supported battered women, a domestic violence shelter, and county prosecutor's office; age: 9.90	T2: 4 months after Tl, T3: 4 months after T2, T4: 6 months after T3, T5: 6 months.			were both positively associated with initial levels of anxiety. Change in both community and school violence exposure and witnessing IPV positively covaried with anxiety. (2) For boys with initially high support, decline in support + increase in community violence was associated with higher anxiety; increase in support + increase in community violence was not associated with anxiety
Various (i.e. IPV, community and school violence)	Depression	Kennedy et al. (2010)	100 children and their families recruited from a social services agency that supported battered women, a domestic violence shelter, and county prosecutor's office; age: 9.90	Baseline: 2–3 weeks after recruitment, Tl: 4 months after baseline, T2: 4 months after Tl, T3: 4 months after T2, T4: 6 months after T3, T5: 6 months.	CDI	4-item Child Report of Witnessing IPV scale; modified version of Things I Have Seen and Heard scale to measure community and school violence (CSVE)	(1) Change in witnessing IPV and CSVE was positively associated with depression over time; change in support was negatively associated with depression. (2) Among children with lower baseline support, the effect of reduction in witnessing IPV on depression was significandy stronger for girls. (3) Among children who initially witnessed lower levels of IPV, reduction of witnessing IPV was associated with significandy lower levels of depression.

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
Various (i.e. physical abuse, interparental violence, community violence)	Anxiety and depression	Margolin et al. (2010)	103 community-based families; children's age: 12.4	Baseline, Tl: approx 1 year after baseline, T2: approx 2 years after baseline.	CDI; trait Scale of the State-Trait Anxiety Inventory	Conflict Tactics Scale—Parent/Child; Domestic Conflict Inventory; Survey of Children's Exposure to Community Violence	(1) Marital aggression contributed uniquely to anxiety. (2) With a one-point increase on violence exposure index, risk of meeting criteria for depression increased by >50%. (3) Significant curvilinear effects showed high cumulative violence increased risk of comorbid symptoms; 76% of youth with higher cumulative violence met thresholds on adverse outcomes, compared to 36 and 7% for youth with moderate and low violence exposure.
Various forms of violence (e.g. picking fights, assault, robbery, rape, threatening serious violence)	Anxiety and depression	Mason et al. (2004)	765 children from Seattle Social Development Project; age: 21.3	Baseline: fifth grade (10–11 years old), Tl: 21 years old	DIS	Self-report on violence	(1) 21% of participants reported two or more violent acts in the past year at age 21 years. (2) Strongest positive predictor of young adult depressive episode was self-reported perpetration of violence (or conduct problems) in grade 5
Various (i.e. school, home, community)	Anxiety, depression	Mrug and Windle (2010)	603 adolescent from Birmingham Youth Violence Study; age: 13.2 (SD = 0.9)	Baseline: 2003, Tl: 16 months later	Major Depressive Disorder scale of the Diagnostic Interview Schedule for Children Predictive Scales (DPS)	Birmingham Youth Violence Study Violence Exposure	(1) Violence exposure at home and school had the strongest effects on internalizing and externalizing outcomes. (2)Witnessing community violence attenuated the effects of witnessing home

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
							violence on anxiety and externalizing problems. (3) No comparable attenuation effects were observed for victimization across contexts.
Various (e.g. stressful life events, including witnessed violence)	Depression	Vaske et al. (2009)	2,023 adolescents from National Longitudinal Study of Adolescent Health (Add Health); age: 18–26 at final wave	Baseline: 1995–1996, Tl: 2001–2002	CES-D	Self-report on violence	(1) DRD2 polymorphism interacted with witnessed violence to predict higher levels of depressive symptoms among African–American females. (2) DRD2 polymorphism did not moderate the effects of witnessed violence on depressive symptoms for other groups. (3) 5HTTLPR did not moderate the effects of witnessed violence on depression for any of the gender–race subgroups.
Various (i.e. parental abuse; community violence)	Substance abuse and depressive symptoms	Wu et al. (2006)	1,119 children/ adolescents from longitudinal study of psychopathology among Puerto Rican children and early adolescents; age: 11.5.	Assessed over three waves between 2000 and 2004.	Questions on lifetime and past-year alcohol use; the alcohol abuse section of the DISC-IV.	Data from parent interview covering various forms of punishment; Things I Have Seen and Heard	(1) Depressive symptoms and alcohol use shared some significant risk factors, including child exposure to violence. (2) Child lifetime physical abuse was positively associated with alcohol use in children. (3) Depressive symptoms also predicted alcohol use; however, when exposure to violence was included in the model, it reduced some of the

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
							association between depression and alcohol use.
Adult exposure to violence							
IPV	Anxiety, depression, and suicide	Blasco-Ros et al. (2010)	56 women originally recruited through the Centers for Helping Women in Spain; a control group of 35 women recruited through women's clubs	Baseline: 2000–2002, Tl: 3 years later	BDI; STAI; suicide assessed by asking women about their lifetime incidence of thoughts and attempts of suicide.	Interview	(1) While at baseline, victims of physical and psychological IPV both had more depressive symptoms than the nonabused group, at the follow-up, only the psychologically abused group continued to have higher levels than the nonabused group.
IPV	Depression	von Eye and Bogat (2006)	188 mothers from Southern Michigan recruited in the third trimester of pregnancy to study health effects of welfare. Age: 25	Baseline, Tl: 1 year, T2: 1 year, T3: 1 year, T4: 1 year	BDI	Self-report	(1) Only IPV—and not welfare variables—predicted the development and level of depression over time. (2) Violence experienced at both more distal and more proximal periods affected level of depression.
Physical and sexual violence	Depression	Hogben et al. (2001)	1,109 HIV+ women recruited from HIV primary care clinics, hospital-based programs; age: 36 years	Baseline; Tl: 6 months after baseline; T2: 1 year after baseline	CES-D	Sexual and physical violence questions	Women's experience of sexual and physical violence predicted depression and risky sexual behavior.
Physical and sexual abuse (e.g. IPV)	Depression	Taft and Watson (2008)	9,683 women from Australian Longitudinal Study on Women's Health;	Baseline: 1996, Tl: 2000	CES-D	Interview	(1) Any form of violence but especially that of partner violence was significantly associated with depression. (2) Violence had a fourfold greater effect on women's depression than pregnancy loss.

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
Various stressful life events (e.g. physical, psychological, or sexual violence)	Anxiety	Kivimaki et al. (2002)	2,991 adults from a community-based longitudinal study of employees in Finland; age: 45.7 years	Baseline: 1995, Tl: 1997; T2: 1998	STAI; General Health Questionnaire; alcohol use assessed with 4-item measure	Measure of stressful life events that included physical, psychological, or sexual violence	(1) Violence was positively related to anxiety. (2) Psychological problems were associated with heightened alcohol consumption. (3) Increased risk for sickness absence associated with life events was only observed for men.
Childhood and adult exposures to violence							
Physical and sexual abuse	Anxiety, depression, and sexual abuse	Gil-Rivas et al. (1997)	330 participants from 26 outpatient drug treatment facilities; age: 35.	Baseline: during treatment, Tl: posttreatment	Hopelessness Scale; RSES; Impact of Event Scale (IES).	Client-Needs-Services-Outcomes Questionnaire	(1) For women, sexual abuse was associated with higher levels of depression and anxiety, while physical abuse was associated with fewer psychological disturbances. (2) For men, sexual abuse was associated only with anxiety; physical abuse was associated with depression and anxiety. (3) Abuse was not necessarily associated with lower levels of drug use at follow-up.
Various (i.e. assault; physical abuse; sexual abuse; accidents; disasters).	Anxiety, depression, and substance abuse disorder	Gil-Rivas et al. (2009)	250 recruits from 11 residential substance abuse programs; age: 36.2. Only 7.1% did not have some form of IPV)	Baseline: minimum of 48 hrs postentry into residential substance abuse program, Tl: 6 months, T2: 12 months	Measure of substance use created to reflect the use during the 30 days preceding the follow-up interviews.	Lifetime and recent violence as measured by: The Life Stressor Checklist—Revised (LSC- R)	(1) Lifetime violence exposure and a diagnosis of PTSD at treatment entry were not associated with substance use over the follow-up. (2) Violence exposure over the follow-up was associated with an increased risk of substance use.
Various (e.g. any major physical violence, any	Depression	Rayburn et al. (2005)	810 women sampled as part of a larger study	Baseline, Tl: approximately 6 months later	Hopkins Symptom Checklist	Interview	Women who experienced major physical violence

Violence type	Psychiatric disorder	Authors	Sample	Study design	Outcome measures	Measure(s) of violence	Article conclusions
physical violence, sexual violence, childhood physical and sexual abuse)			examining experiences of drug use, violence, and HIV risk among impoverished women; age: 34 years				(e.g. hit, choked, threatened, or assaulted with gun) during the time interval between baseline and follow-up were two times more likely to have diagnosis of depression at follow-up than those women without these experiences

TABLE 2

Study designs and sampling, measurement and analytic choices to address limitations in measurement and causal inference

Study design	Sampling	Measurement	Statistical strategies to address limitations of each measure	Limitation to causal inference	Solutions to causal inference issues	Analytic strategies
Prospective cohort study	Population-based	Retrospective self-report with specific questions on types of abusive behaviors and context of abuse	IRT models accounting for severity of violent acts; Bayesian models to reduce misclassification	Unobserved and observed common causes of violence and psychopathology	Propensity-score matching; violence change-on-psychopathology change models	
		Self-report and official court records	Latent variable models to incorporate multiple sources	Bidirectional relation between violence and psychopathology		Complex systems approaches
		Objective geospatial measure of exposure		Time-dependent confounding and mediation	Marginal structural models	Structural equation models
		Direct observation of violence				Latent variable and growth mixture models
Postviolence cohort study	Population-based	Retrospective self-report with specific questions on types of abusive behaviors and context of abuse	IRT models accounting for severity of violent acts; Bayesian models to reduce misclassification	Confounding by previolence psychopathology	Propensity-score matching	
				Unobserved and observed common causes of violence and psychopathology		
		Self-report and official court records	Latent variable models to incorporate multiple sources	Bidirectional relation between violence and psychopathology		Complex systems approaches
		Objective geospatial measure of exposure		Time-dependent confounding and mediation		Structural equation models
		Direct observation of conflict		Heterogeneity of responses to violence due to moderators		Latent variable and growth mixture models
Exposed–unexposed control study	Matched exposed and unexposed groups	Self-report and official court records	Latent variable models to incorporate multiple sources	Unmeasured differences between exposed and unexposed		

Study design	Sampling	Measurement	Statistical strategies to address limitations of each measure	Limitation to causal inference	Solutions to causal inference issues	Analytic strategies
				Unobserved and observed common causes of violence and psychopathology	Propensity-score matching	
				Bidirectional relation between violence and psychopathology		Complex systems approaches
				Time-dependent confounding and mediation		Structural equation models
				Heterogeneity of responses to violence due to moderators		Latent variable and growth mixture models
Exposed–unexposed control study	Exposed and unexposed defined by varying degrees of exposure in same population (e.g. military or population surrounding a mass disaster)	Self-report and official court records	Latent variable models to incorporate multiple sources	Unobserved and observed common causes of violence and psychopathology	Propensity-score matching	
				Bidirectional relation between violence and psychopathology		Complex systems approaches
		Objective geospatial measure of exposure		Time-dependent confounding and mediation		Structural equation models
				Heterogeneity of responses to violence due to moderators		Latent variable and growth mixture models
Natural experiment	Population may be defined by exposure to the natural experiment + matched unexposed control group	Objective measures of exposure to the exogenous source of exposure to violence		Unobserved and observed confounding due to nonrandom selection of exposed versus unexposed	Propensity-score matching	Difference-in-difference models
		Self-reports of exposure to violence	IRT models accounting for severity of violent acts; Bayesian models to reduce misclassification	Heterogeneity of responses to violence due to moderators		Latent variable and growth mixture models

Study design	Sampling	Measurement	Statistical strategies to address limitations of each measure	Limitation to causal inference	Solutions to causal inference issues	Analytic strategies
		Self-report and official court records	Latent variable models to incorporate multiple sources			
		Objective geospatial measures of exposure				
Instrumental variable approach	Population-based	Objective measures of instrument		Low correspondence between instrument and violence exposure	*F*-test to assess strength of instrument; partial *r*-squared between IV and exposure; sensitivity analyses	Two-stage least-squares models: (1) estimating relation between instrument and exposure; and (2) estimating relation between predicted value of the treatment and the outcome
	Exposed and unexposed respondents from same population	Self-reports of exposure to violence	IRT models accounting for severity of violent acts; Bayesian models to reduce misclassification	The instrument influences the outcome through other pathways		
		Self-report and official court records	Latent variable models to incorporate multiple sources	The instrument actually reflects another exposure that contributes to psychopathology		
		Objective geospatial measures of exposure				