

Effects of Adolescent Violent Victimization on Adult Depression: Testing Heterogeneity for Men and Women

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Abstract

Many studies have associated adolescent violence exposure with later mental health outcomes, but because violence cannot be randomly assigned, few have established causality. Additionally, few have directly tested whether violence affects boys' and girls' psychological health differently. The author hypothesizes that (1) violence has a negative causal effect on depression and (2) the effects of violence on depression are worse for women than for men. Using National Longitudinal Study of Adolescent Health data, the author builds multilevel propensity score models with wave 1 predictors in a cross-lagged two-level hierarchical linear model estimating the effects of wave 2 violence on wave 3 outcomes. The results (1) confirm that women have higher depression scores than men net of other factors, (2) show that violence exposure raises average depression scores for both sexes, and (3) uncover no significant sex differences in effect size of violence on depression.

Keywords

depression, gender, trauma, adolescents

Violence exposure, and the associated persistent states of fear (Cornell and Mayer 2010) and helplessness (Peterson and Seligman 1983) that accompany such exposure, represents a serious problem for today's school-age youth. In particular, exposure to violence has been linked causally to a variety of negative outcomes, from poor academic achievement (Sharkey 2010) to later violent perpetration (Bingenheimer, Brennan, and Earls 2005). In these and other studies, mental health states have often been either implicitly or explicitly identified as the culprit for mediating the associations between violence exposure and poor outcomes. Yet any direct connection between violence exposure and poor mental health is not typically examined in a way that is rigorous enough to draw causal inferences. Violence has been conceptually linked to depression and other

poor mental health outcomes (e.g., Biggs et al. 2010; DuRant et al. 2000; Lehman and Repetti 2007; Rigby 1999), but these arguments rarely establish strict causal relationships. What has been missing is a serious attempt to analyze the effects of school violence exposure on mental health, using established causal inference methods to account for both the pretreatment covariates that differentially predict violence exposure for

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individuals and the fact of violence's uneven distribution across social settings.

As well, patterns of violence exposure among school-age youth are highly gendered (Benbenishty and Astor 2005; Popp and Peguero 2011). In particular, boys are far more likely than girls to be exposed to physical violence (Roberts et al. 2012; Rose and Rudolph 2006). However, it has not been established whether the effects of violence differ by gender group. It may be that boys fare worse after violence exposure if their exposure is more severe; it may be that girls fare worse if their exposures are experienced as acute and therefore more traumatic. Research into this question offers mixed findings, with some (e.g., Bond et al. 2001; Lopez and DuBois 2005; Paquette and Underwood 1999) arguing that violence affects the sex groups differently and others (e.g., Biggs et al. 2010; Lehman and Repetti 2007; Nishina and Juvonen 2005; Nishina, Juvonen, and Witkow 2005) finding no such differences.

Here, I test whether the experience of violence is causally linked—not just associated—with depression, a key indicator of mental health. I also investigate the extent to which these effects are gendered: whether violence's effects are worse for one gender group than the other. I aim to show that violence exposure in adolescence has specific effects on later mental health states that are not attributable to endogenous selection or concomitant disadvantage, and to examine whether men and women are differentially vulnerable to these effects.

BACKGROUND

Associations between Adolescent Violence Exposure and Mental Health

Many studies have shown that exposure to violence and victimization during adolescence is linked to key aspects of psychological well-being, including depression and other mental or emotional disruptions. For example, Cornell and Mayer (2010) found that school violence results in significant school- and personal-level disruptions such as fear, anxiety, weakened social bonding, school avoidance, classroom disorder, impaired cognitive and academic development, and altered social-emotional trajectories; Bond et al. (2001) found that past victimization predicts

symptoms of anxiety and depression in late adolescence but that previous emotional problems do not predict later victimization; Lehman and Repetti (2007) found that victimization leads to changes in students' moods and self-esteem that influence perceptions of later interactions with parents and others; and Sharkey et al. (2012) found that recent local violence leads to impaired emotional and impulse regulation in children and acute psychological distress among parents and caregivers. One highly publicized recent addition to this literature is a study by Copeland et al. (2013), published in *JAMA Psychology*, that prospectively tracked a panel of 1,420 students in North Carolina, each interviewed four to six times between the ages of 9 and 16 years. Following this panel into young adulthood (ages 24–26 years) and controlling for childhood psychiatric problems and family hardships, Copeland et al. found victims of violent bullying at highly elevated risk for psychiatric problems such as depression, agoraphobia, panic disorders, generalized anxiety, and suicidal ideation or attempt. The worst effects appeared for those who were both bullies and victims, suggesting that multiple vulnerable conditions increases susceptibility to negative psychological and behavioral outcomes.

However, because of the nature of the treatment—i.e., violence—few studies can address the effects of exposure on mental health in a methodologically rigorous way. Even well-designed prospective cohort studies such as that of Copeland et al. (2013) cannot rule out endogenous selection (i.e., that students are chosen by their peers for victimization on the basis of characteristics that would lead to worse mental health outcomes even in the absence of exposure) or other forms of unobserved confounding. Two notable exceptions that manage to suggest causal effects of violence exposure on emotional and behavioral outcomes are studies by Sharkey et al. (2012), who leveraged variation in the timing of neighborhood homicides relative to assessments conducted for the Chicago School Readiness Project and found that recent local homicides impair children's emotional regulation and cognitive functioning, and Bingenheimer et al. (2005), who, in a strategy similar to this study's, used longitudinal data from the Project on Human Development in Chicago Neighborhoods to show that net of student and school characteristics at wave 1, exposure to firearms violence at wave 2 increased students' likelihood of perpetrating violence at wave 3. The rarity

of such well-done studies underscores the inherent difficulty in determining causal effects of non-randomized treatments. This is perhaps the major challenge facing research into the effects of violence.

Heterogeneity of Effects across Gender Groups

It is widely recognized that the two sexes are differentially susceptible to psychological problems. The difference between the rates at which men and women suffer depression is long established (e.g., Hankin et al. 1998; Nolen-Hoeksema 2001; Piccinelli and Wilkinson 2000): women consistently report more frequent and more severe symptoms. An additional but separate concern is that many studies, including that of Copeland et al. (2013), treat separately the effects of violence exposure on mental health for boys and girls. This often involves distinguishing between emotional and physical health consequences of violence (Brunstein Klomek, Sourander, and Gould 2010; Rigby 1999) or between “internalizing” and “externalizing” symptoms of exposure (Osofsky 1995). Some have suggested that adolescent victimization is associated with emotional distress only among girls (Bond et al. 2001) or more strongly among girls (Due et al. 2005; Lopez and DuBois 2005). It has also been suggested that boys are more vulnerable to the effects of direct victimization, whereas girls are more vulnerable to relational aggression or to witnessing violence (Paquette and Underwood 1999). These differences are typically attributed to gendered coping or adaptation mechanisms (Clampet-Lundquist et al. 2011) or to gendered differences in routine activities, creating gender-specific risks of victimization (Popp and Peguero 2011).

Despite this attention to the intersection of gender and violent victimization, few studies have directly tested whether exposure to violence has different effects on boys’ and girls’ psychological health—that is, whether the effect sizes of violence on mental health outcomes are statistically distinguishable for girls and boys. Many simply assume, either directly or implicitly, that the two sexes must be different. Explicit tests for gender-specific differences in violence’s effect on mental health have provided mixed results. Some detect different effects of victimization across gender groups. Bond et al. (2001) prospectively surveyed

middle school students in the United Kingdom and found that repeated victimization predicts symptoms of anxiety or depression for girls but not boys. Paquette and Underwood (1999) found, in a study of seventh and eighth grade students in the American Pacific Northwest, that girls are more distressed by victimization because they dwell on it more and are more likely to see it as reflective of their self-worth. And both Grills and Ollendick (2002) and Lopez and DuBois (2005) found that peer victimization affects middle school girls’ self-esteem more than boys’, leading to more emotional problems.

Others, on inspection, uncover no such differences. Biggs et al. (2010) tested gender as a moderator of the effect of victimization on emotional well-being; they found no significant differences in the trajectory of victimization and no significant gender moderating of the association between victimization and affect, suggesting that victimization has similar effects on boys’ and girls’ emotional well-being. Lehman and Repetti (2007) found increased anxiety and emotional trouble among fifth graders following victimization but no difference between boys and girls in the size of this effect. Nishina and Juvonen (2005) and Nishina et al. (2005), using multilevel and structural equation modeling on a middle school sample, found an effect of violence exposure on maladjustment but no difference between boys and girls in the process by which violence produces the effect. The existence of gendered differences in school violence’s effects on emotional and mental health appears to remain an open question.

Summary of the Current Study

In this article, I attempt to address both of these apparent lacunae: the dearth of methodologically rigorous examinations of the causal effect of violence on mental health, and the open question of gender differences in effect sizes. To summarize the method, I estimate propensity scores for exposure to violence using a hierarchical binary logistic regression model with cross-lagged predictors, then control for propensity score in regression models predicting depression at a later time; more detail is provided below. I use three waves of longitudinal data from the National Longitudinal Study of Adolescent Health (Add Health). This follows the example of Bingenheimer et al. (2005), one of the few existing studies to apply

propensity score modeling to the effects of individual violence exposure on later outcomes.

On the basis of the summarized literature, I test two main hypotheses: (1) violence exposure will have a negative causal effect on depression, observed as higher scores on the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff 1977), after controlling for propensity score stratum assignment, and (2) the effect of violence on depression will be worse for women than for men, observed as a significant female $\times$ violence interaction effect in regression models controlling for propensity score stratum. I also expect, consistent with the overwhelming consensus in the literature (e.g., Hankin et al. 1998; Nolen-Hoeksema 2001; Piccinelli and Wilkinson 2000), that women will report higher levels of depression in young adulthood than men, but this is more a “reality check” than a hypothesis *per se*.

The empirical findings, described in the following, confirm that women generally fare worse than men on depression score after controlling for all other factors. Propensity score model results are consistent with hypothesis 1: I observe negative effects of violence on depression, which can be interpreted causally. However, I find no support for hypothesis 2, with no significant female $\times$ violence interaction effects in any of the main models. This suggests that school violence exposure is equally detrimental to mental health for both women and men.

METHOD

Analytic Approach

There are three main obstacles to estimating the effects of violence exposure on mental health in later life: (1) nonrandom assignment of treatment to individuals, (2) nonindependent likelihood of treatment assignment across social contexts (here, schools), and (3) ascertaining a correct temporal order of pretreatment covariates, treatment exposure, and outcomes or effects.

The first of these, nonrandom treatment assignment, is probably the most significant complication. Any investigation addressing the outcomes of a nonrandomized exposure must use extra caution to rule out the possibilities of endogenous selection and unobserved confounding (Rubin 1974). The problem is magnified when the treatment is violence and the outcome is mental health. Students are not victimized at random: certain

individual characteristics increase students' risk for violence exposure. These include observable attributes such as male gender (Olweus 1993), nonheterosexual orientation (Swearer, Espelage, Vaillancourt, and Hymel 2010) or perceived insufficient displays of masculinity (Klein 2012), and obesity (Swearer et al. 2010), and also preexisting mental health complications or factors that predict later mental health problems with or without violence exposure, such as disadvantaged social background (Morenoff, Sampson, and Raudenbush 2001; American Academy of Pediatrics 2000), anxiety, insecurity, negative self-concept, internalizing problems, physical weakness, and peer rejection or social unpopularity (Hodges and Perry 1999; Olweus 1993). All this amounts to one problem: it is very difficult to determine if any negative outcomes are actually caused by violence exposure.

The gold standard for determining the causal effects of a treatment or exposure is the controlled randomized trial (CRT). The benefit of a CRT is that by randomly assigning subjects to treatment and control conditions, we break any association between treatment assignment and pretreatment characteristics; ideally, the treated and untreated populations are identical in all ways except that one group received the treatment and the other did not. Under these conditions, known as *strongly ignorable treatment assignment*, any posttreatment differences between the treatment and control groups are attributable to the treatment itself, and adjusting for observable covariates is sufficient to remove bias (Rosenbaum 1984). Unfortunately, or perhaps fortunately, conducting a CRT is neither feasible nor desirable when the treatment is school violence. No ethical scientist would suggest randomly assigning students to violent victimization.

Instead, I use propensity score stratification (Rosenbaum and Rubin 1983, 1984) to estimate causal effects. Propensity score modeling is based on the potential outcomes framework of counterfactual causal inference, developed by Rubin (1974) and others (e.g., Holland 1986; Morgan and Winship 2007). It assumes that exposure is an outcome of both structural and random processes and therefore that every individual has a probability of being exposed to violence: a “propensity score,” π . The analysis proceeds in three stages: (1) Obtain estimates of individuals' propensity scores ($\hat{\pi}$) using a multivariate logistic regression model in which the outcome

is violence exposure and the predictors are as many different relevant observable characteristics as possible. (2) Once propensity scores are estimated, divide individuals into strata with others who have similar values of $\hat{\pi}$. Each stratum contains a mixture of individuals who did and did not receive the treatment, and the treated and untreated groups within each stratum are balanced on all relevant variables. Thus, each stratum contains similar people with comparable chances of receiving the treatment, but some of whom were treated and some of whom were not for reasons we assume are random. (3) Within strata, we have *conditionally ignorable treatment assignment*, with each stratum acting like its own CRT. Rosenbaum and Rubin (1984) showed that conditioning on propensity score can be sufficient to control for variability in both observed and unobserved covariates. That is, thanks to the collinearity of variables, estimated propensity scores capture information even about unmeasured variables, as long as the model is correctly specified. Indeed, because predicted propensity scores adjust for both systematic and random imbalance among covariates, simulations show that predicted propensity scores give even more precise estimates of treatment effects than do "true" propensity scores (Hirano, Imbens, and Ridder 2003; Rubin and Thomas 1996). Thus, given proper modeling, the mean difference in outcomes between treated and untreated within each stratum can be computed (using standard regression or other simple comparisons) to obtain an unbiased estimate of the average treatment effect.

The second major obstacle, nonindependent likelihood of treatment assignment, stems from the fact that π (propensity for violence exposure) is a function of both individual and contextual factors. Specifically, because adolescents are sorted into schools, the analysis must account for the fact that characteristics of schools affect the likelihood of violence exposure independent of individual characteristics. This defies the independence-of-units assumption of standard regression modeling (Bursstein 1980; Raudenbush and Bryk 1986), which requires that every subject's error term in a model be independent of every other subject's.

To solve this problem, the values of $\hat{\pi}$ must be estimated using a hierarchical model (Raudenbush and Bryk 2002). In this case, because estimating propensity scores requires using a model with a binary outcome (exposed or unexposed to

violence), I use hierarchical binary logistic regression, a species of hierarchical generalized linear models (HGLMs). Because propensity for treatment assignment is expected to vary both within and across schools, a two-level HGLM with students at level 1 and schools at level 2 allows us to predict the binary treatment exposure (violence) at level 1 while controlling for facts about both the individual subjects (level 1 covariates) and the schools in which they are nested (level 2 covariates). The calculated propensity scores then give students' likelihood of receiving the treatment as a function of both level 1 and level 2 factors.

The third major problem facing this kind of analysis is obtaining the correct temporal ordering of events. Cross-sectional data, on which case-control studies of violence's effects are based (e.g., Due et al. 2005; Williams et al. 1996), raise the concern of whether our conceptual ordering of events actually aligns with their ontological ordering. On the basis of cross-sectional data, it is not strictly possible to ensure that individual-level covariates actually preceded treatment. Rather than predicting exposure, students' behaviors, self-concepts, or social contexts may alter as a result of exposure; this makes controlling pretreatment covariates problematic. Likewise, cross-sectional data cannot establish with certainty that the putative outcomes are actually consequences of the treatment. It may be that what we treat as outcomes are really preexisting characteristics of individuals that predict treatment assignment (Nishina et al. 2005). Even if questions about pretreatment states or behaviors are asked retrospectively, the experience of violence may lead exposed individuals to remember and report covariates differently. For example, to reduce cognitive dissonance, a person suffering depression who has been victimized by violence might report retrospectively that the victimization triggered the depression, even if the latter existed prior to exposure; this poses obvious problems for the validity of any study based on these kinds of data. (In their review, Brunstein Klomek et al. 2010 compared cross-sectional with longitudinal findings for the effects of school victimization on mental health.)

The clear solution to this problem is to use longitudinal data. This is often accomplished in the form of prospective cohort studies, many of which (including Bond et al. 2001 and Copeland et al. 2013) have made major contributions to our

understanding of the processes linking violence exposure and health. However, as noted, even a well-designed prospective cohort study cannot fully control for unobserved confounding; moreover, depending on the frequency of follow-up, it may still not be possible to determine whether violence preceded the outcome(s) or vice versa. Here, following Bingenheimer et al. (2005), I use data drawn from three separate waves of a longitudinal study. I control for pretreatment covariates measured at wave 1, define the treatment specifically as violence experienced at wave 2, and then assess mental health outcomes at wave 3. The cross-lagged structure obviates reverse causality; it is impossible for the pretreatment covariates to be affected by the treatment, because the treatment (at wave 2) is measured after the covariates (at wave 1); likewise, the treatment cannot be caused by the outcomes, because the outcomes are measured several years later (during wave 3).

To summarize the analytic approach: I estimate propensity scores for exposure to violence at wave 2 using a cross-lagged HGLM that controls for pretreatment covariates at wave 1; I then control for the derived propensity score strata in separate cross-lagged models estimating the effects of wave 2 violence exposure on depression at wave 3.

Data

Data used in this study come from the Public Use subsample of Add Health, one of the largest and most comprehensive surveys of adolescents ever undertaken (Harris et al. 2009). Add Health is a longitudinal study that follows a nationally representative sample of adolescents in the United States. The study began during the 1994–1995 school year with respondents in grades 7 to 12 and has continued through three successive waves of follow-up in 1996, 2001–2002, and 2007–2008. A sample of 80 high schools and 52 middle schools from the United States was chosen, with unequal probability of selection. Systematic sampling methods and implicit stratification incorporated into the Add Health study design ensure that this sample is representative of U.S. schools with respect to region of country, urbanicity, school size, school type, and ethnicity. In-school as well as in-home surveys were given to the study subjects; data were also gathered from their parents, siblings, fellow students, school administrators, and romantic partners, and these data were

matched with information about neighborhoods and communities available in extant databases. For this article, as noted, I draw on the first three waves of Add Health data, gathered in 1994–1995, 1996, and 2001–2002. By using pretreatment covariates from wave 1 to predict school violence exposure at wave 2, then propensity scores from wave 2 to predict outcomes at wave 3, I remove the possibility of reverse causality.

I further reduce the sample by several criteria. I retain only students in 11th grade or lower at wave 1, indicating that they would be no further along in school than 12th grade at the time of wave 2. I drop students who report that they are no longer in school as of wave 2 for any other reason. In addition, I retain only those who provide data at all three waves and whose violence exposure status at wave 2 could be determined. This leaves me with an initial sample of 3,766 students, 2,031 female and 1,735 male, in 132 schools, for whom I estimate propensity scores.

Key Variables

The main treatment variable for this investigation is exposure to violence at wave 2. “Exposure” is defined many different ways but typically consists of at least one of three dimensions: witnessing violence, direct victimization by violence, or perpetrating violence (e.g. Acosta et al. 2001; Selner-O’Hagan et al. 1998). I focus only on witnessing and victimization. At wave 2, Add Health asked respondents several questions about their experiences with violence in the past 12 months. Specifically, students were asked the following questions: “During the past 12 months, how often did each of the following things happen? You saw someone shoot or stab another person; Someone pulled a knife or gun on you; Someone shot you; Someone cut or stabbed you; You were jumped.” To treat this as a classic exposure variable, respondents were counted as “exposed” if they answered anything other than “never” to any of these questions.

Two caveats are necessary for the use of this variable. One is the likelihood of gender differences. As noted, male and female adolescents’ most common exposures differ. In part, using one definition of “exposure” controls some of this variation: we are confident that both sexes refer to the same thing when they indicate exposure. If the exposure does have different sequelae for

the two groups, that difference should be reflected in the interaction effects used to test hypothesis 2. It might be additionally useful to include other gender risk-specific types of exposure, such as victimization with a sexual component, but I am limited in this regard by the questions Add Health provides. The second caveat is the treatment of violence exposure as a single traumatic incident. Increasingly, analyses of violence exposure account for the substantial co-occurrence of different kinds of trauma and the cumulative effects of disadvantage over time (e.g., Arseneault et al. 2006; Sampson, Sharkey, and Raudenbush 2008; Wodtke, Harding, and Elwert 2011). I recognize these issues—indeed, my findings are strongly in line with the account of violence as a persistent stressor—but choose the simpler operationalization to establish a baseline effect.

Depression, the key outcome variable, is measured with a modified version of the CES-D (Radloff 1977). The standard CES-D contains 20 self-report items forming a scale that measures symptoms of a number of different aspects of depression, including negative affect, positive affect, interpersonal relations, and somatic complaints (Radloff 1977). It was developed in 1976 for general use in populations aged 18 years or older, but research has shown that the CES-D measures are valid for multiple categories of adolescents in the United States, including across race-ethnicity, gender, immigrant generation, and cultural categories (Perreira et al 2005). Although the CES-D is not a clinical diagnostic tool, it is widely used in research for identifying populations at risk for developing depression or anxiety disorders. Add Health includes a modified version of the CES-D in all of its survey waves; at wave 3, the survey instrument contains 10 questions from the different categories of the CES-D. These are summed together (reverse-coding the positive-affect items as necessary) to create a single depression-score index, with values potentially ranging from 0 to 30, where higher numbers indicate a higher intensity and/or greater number of depressive symptoms.

In the wave 1 data, I identified approximately 125 pretreatment covariates in several categories: home environment, demographic traits, mental health, physical health, temperament and behaviors, school environment, peer influences, social support, academic characteristics, and violence exposure. Aggregating student data to the school level, I created variables measuring schools'

average levels of student self-reported feelings of school connectedness, safety at school, closeness to people at school, happiness at school, teachers' fairness to students, and other students' prejudice. These were used in estimating the propensity scores, described below.

Propensity Score Estimation and Stratification

I used a maximum likelihood hierarchical logistic regression model to predict propensity scores $\hat{\pi}$ for all members of the data set who met the criteria outlined previously. As noted, it is best to use a wide variety of predictor variables when constructing propensity score models to maximize within-stratum balance on both observable and unobservable traits. For this reason, I selected variables for the propensity score model using an iterative stepwise regression procedure. With each iteration, the procedure either added to the model the covariate most strongly associated with wave 2 violence exposure conditional on the other covariates in the model, as long as that association was significant at the $\alpha = .15$ level, or removed from the model any covariates that were no longer associated with wave 2 violence exposure at that significance level. I ran this procedure for the level 1 covariates using Stata and then used the selected variables in estimating a two-level model with HLM software, at which point I selected among the level 2 covariates which were significantly associated with wave 2 violence exposure net of the effects of all the other covariates. In the final model, I reintroduced measurements for conditions such as temperament, age, and home condition, which were not significant at $\alpha = .15$ in the stepwise model but which extant research identifies as relevant for violence exposure. This resulted in a final model that estimated each subject's $\hat{\pi}$ using 39 level 1 covariates and a single level 2 covariate. (Details of the final model are available from the author on request.)

Having calculated $\hat{\pi}$ values for each subject in the sample, the second stage of propensity score modeling involves stratifying subjects into groups containing both treated and untreated subjects, but within which the two exposure status groups do not significantly differ in their estimated propensity score or (ideally) any other covariates. This requires further restricting the sample to the region of "common support": the range of values of $\hat{\pi}$

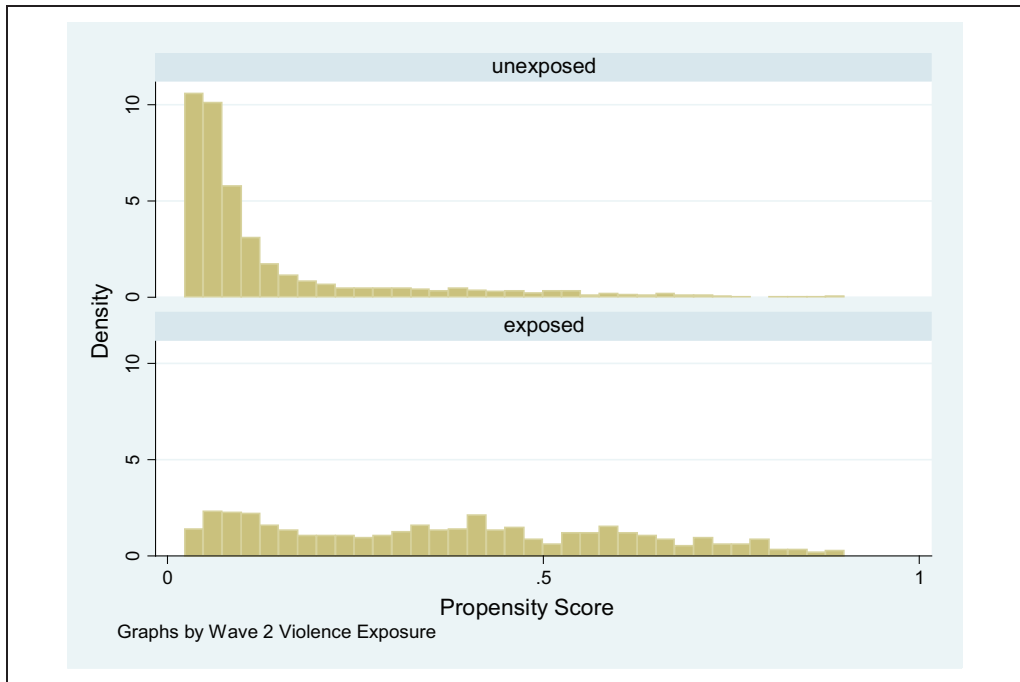


Figure 1. Probability Density Distribution of $\hat{\pi}$ for Treated and Untreated Groups.
Source: National Longitudinal Study of Adolescent Health.

that includes both treated and untreated observations. Figure 1 shows the probability density distribution of $\hat{\pi}$ for the treated and untreated groups, revealing some stark differences. In particular, the distribution of $\hat{\pi}$ among the untreated is sharply skewed to the right, with a mean of 0.123 and more than half (52.82%) of the observations in the region of $\hat{\pi} < 0.075$. By contrast, the distribution of $\hat{\pi}$ for the treated is much more uniform, with a mean of 0.377 and median of 0.364. Restricting to the region of common support resulted in the exclusion of subjects with values of $\hat{\pi}$ less than 0.0227 (all of whom were unexposed) or greater than 0.9004 (all of whom were exposed). This leaves a final sample of 3,354, of whom 2,783 were unexposed and 571 were exposed at wave 2.

I divided the 3,354 subjects into 10 strata on the basis of their estimated propensity scores. Cut points were determined empirically, such that the treated and untreated groups within each stratum are statistically indistinguishable in their mean propensity score and all relevant pretreatment covariates. Table 1 lists stratum cut points and frequency counts for the treated and untreated

groups within strata. At $\alpha = .05$, I found no significant differences between treated and untreated subjects within each propensity score stratum on any wave 1 covariates. This strongly suggests that the propensity score model adequately controls for selection on pretreatment covariates.

Estimating the Effect of Violence on Mental Health

Having estimated propensity scores for wave 2 violence exposure and stratified subjects into groups on that basis, I estimate the effects of wave 2 violence on wave 3 depression by controlling for propensity score stratum assignment using maximum likelihood ordinary least squares regression models. (Alternative specifications of functional form, such as Poisson, produced substantively similar findings.) In these models, I use additional covariates from waves 1 and 3 to adjust for gender (male or female), self-identified race (white, black, or other), and three age categories at wave 3 (18–20, 21–22, and 23–25 years). To address one of this study's central questions—

Table 1. Frequency Counts per Propensity Score Stratum.

Stratum	Propensity Score (Range)	Unexposed (<i>n</i>)	Exposed (<i>n</i>)	Total (<i>n</i>)
1	[0.0–0.05)	831	18	849
2	[0.05–0.075)	639	34	673
3	[0.075–0.1)	393	32	425
4	[0.1–0.15)	310	55	365
5	[0.15–0.25)	213	64	277
6	[0.25–0.35)	132	68	200
7	[0.35–0.5)	142	121	263
8	[0.5–0.55)	45	26	71
9	[0.55–0.75)	56	94	150
10	[0.75–1.0]	22	59	81
Total		2,783	571	3,354

whether violence's effects on mental health are heterogeneous by gender—I also introduce an interaction term between gender and wave 2 violence exposure (hereafter “violence $\times$ female”). Because there is a well-established association between mental health and educational attainment (Mirowsky and Ross 2003), I also control for educational attainment at wave 3 as a four-category ordinal variable on the basis of self-reported highest educational level completed: less than high school, high school but no college, some college, and college graduate or higher. I control for violence exposure status at wave 1 and self-reported depression and suicidal ideation at wave 1 to adjust for the possibility that depression is chronic for some students. And because violence exposure may itself be a chronic problem, I control for violence exposure status at wave 1 (measured with the same questions as at wave 2). Finally, because depression may be linked to a subjective sense of pessimistic future outlook or negative aspirations (Hirsch, Conner, and Duberstein 2007), I control for respondents' wave 1 indications of whether they thought they were likely to go to college. More details are presented in the following section.

RESULTS

Descriptive Findings

It is interesting at the outset to compare the treated and untreated groups on some of the 125 identified pretreatment covariates. Table 2 presents mean values of some covariates for the treated and

untreated groups, as well as mean differences and test statistics with *p* values for the differences. Immediately apparent is that those who do not experience violence at wave 2 were, at wave 1, already quite different from those who do. Demographically, compared with those who will be unexposed at wave 2, those who will be exposed at wave 2 are much more likely to be male, much less likely to be white, and slightly (though significantly) older at wave 1. Exposed subjects report less supportive home environments and lower levels of school connectedness and social support than the unexposed. Exposed subjects also report poorer mental and physical health. In temperament, the unexposed are much less likely to engage in a number of risky behaviors, such as using substances; they are also much less likely to have friends who engage in these kinds of behaviors. The unexposed have better academic backgrounds as well: they are less likely to have been held back a grade or expelled or to report frequent trouble with homework and more likely to report college aspirations. The starkest differences are for previous violence exposure. Among those not exposed at wave 2, only 14.1% report witnessing or victimization at wave 1—lower than the rates estimated by many prevalence studies of school violence exposure (e.g., Benbenishty and Astor 2005)—whereas among those exposed at wave 2, a full 62.3% report either witnessing or victimization at wave 1. These staggeringly different rates of pretreatment exposure suggest that for many, wave 2 violence exposure is part of an ongoing pattern of exposure that begins much earlier in life and persists through the adolescent years.

Table 2. Comparison of Exposed and Unexposed Groups on Selected Wave 1 Covariates.

Pretreatment Covariate	Unexposed Mean	Exposed Mean	Difference	t	p
Demographic Traits:					
Proportion female	0.5764	0.3739	0.2025	9.76	<.001
Proportion white	0.7049	0.5764	0.1285	6.57	<.001
Proportion black	0.2179	0.3173	-0.0994	-5.57	<.001
Age at wave 1 (years)	14.95	15.17	-0.22	-3.17	.002
Home environment:					
Good relationship with mother	0.857	0.813	-0.044	-2.89	.004
House kept in good condition	0.891	0.783	0.108	7.68	<.001
Feel unsafe in neighborhood	0.037	0.072	-0.035	-4.06	<.001
School environment:					
Feel close to people at school ^a	2.22	2.41	-0.19	-4.63	<.001
Feel happy at school ^a	2.23	2.55	-0.32	-6.67	<.001
Feel teachers treat students fairly ^a	2.13	2.46	-0.33	-7.60	<.001
Mental health					
Frequently feel like a failure	0.027	0.067	-0.040	-5.25	<.001
Frequently feel unhappy	0.186	0.271	-0.085	-5.05	<.001
Seriously contemplated suicide	0.120	0.165	-0.045	-3.16	.002
Physical health					
Frequently feel sick	0.035	0.067	-0.032	-3.80	<.001
Frequently have poor appetite	0.121	0.170	-0.049	-3.47	<.001
Frequently have trouble sleeping	0.223	0.283	-0.060	-3.38	<.001
Temperament and behaviors					
Describe self as impulsive	0.356	0.477	-0.121	-5.96	<.001
Sexually active	0.278	0.515	-0.237	-12.27	<.001
Regular smoker	0.157	0.250	-0.093	-5.79	<.001
Binge drink at least once a month	0.069	0.188	-0.119	-9.90	<.001
Ever use any illegal drug	0.222	0.441	-0.218	-11.88	<.001
Academic characteristics					
Ever held back a grade	0.175	0.312	-0.137	-8.22	<.001
Ever been expelled from school	0.027	0.103	-0.076	-9.16	<.001
Frequent trouble with homework	0.242	0.427	-0.185	-9.84	<.001
Feel likely to go to college ^a	4.28	3.79	0.49	10.49	<.001
Peer influences					
Have close friend who smokes	0.380	0.566	-0.186	-8.94	<.001
Have close friend who drinks	0.489	0.682	-0.193	-9.20	<.001
Social support					
Feel people dislike you	0.047	0.118	-0.071	-7.16	<.001
Feel lonely	0.065	0.115	-0.050	-4.45	<.001
Frequent trouble with other students	0.145	0.253	-0.108	-6.87	<.001
Violence exposure					
Witnessing or victimization	0.141	0.623	-0.482	-30.26	<.001

a. Measured on a Likert-type scale ranging from 1 = *strongly agree* to 5 = *strongly disagree*.

In contrast, Table 3 provides the means of the treatment (wave 2 violence exposure) and outcome (wave 3 CES-D score) variables, and selected wave 3 controls (described earlier), over propensity score strata. Here, too, interesting patterns emerge. Because the propensity score is the predicted risk for exposure to violence, the trend toward higher rates of violence exposure in the higher propensity score strata is expected, but it is worth noting that the observed rate of violence exposure in the eighth stratum is lower than predicted, leading to a large apparent jump in average propensity score between strata 8 and 9. Average CES-D scores within strata show an upward trend as well, indicating that those more likely to be exposed to violence are also more depressed on average. The distribution of most control variables is in line with expectations: female and white respondents are overrepresented in the lower risk strata and underrepresented in higher risk strata; those in higher risk strata attain less education by wave 3, and those in lower risk strata attain more; and wave 1 depression scores generally increase from low-risk to high-risk strata, and wave 1 self-reported college aspirations generally decline. The distribution of wave 1 violence exposure stands out in particular: none of the 1,947 subjects in strata 1 through 3 experienced any wave 1 violence, and very few of those in stratum 4 were exposed either. However, over 80% of the subjects in stratum 6 were exposed at wave 1, as well as over 90% of those in strata 7 through 9 and all 81 members of stratum 10. The rate of wave 1 violence exposure in stratum 5 closely mirrors the rate in the sample as a whole.

Overall, this suggests serious distinctions between those who are likely to be victimized and those who are not. Even before the administration of Add Health wave 2, those who go on to be exposed to violence are markedly different on a wide variety of key variables, including family and social support, risky behaviors, school connectedness, peer influences, and mental health and emotional functioning. This underscores the serious problem of accounting for the endogenous nature of treatment assignment net of these factors: those likeliest to be exposed to violence are, at baseline, also likely at greater risk for later life depression whether or not they experience violence. There are also stark patterns of pretreatment variables' association with propensity for wave 2 violence exposure; the differences in the rates of violence exposure at wave 1 stand out in particular,

as well as the clustering of risk for violence by race and gender. Results from the propensity score models, described below, clarify whether wave 2 violence exerts a causal effect on wave 3 depression given these complicating considerations.

Effects of Violence on Depression

Table 4 shows results from the ordinary least squares regression models predicting the effect of wave 2 violence exposure on wave 3 CES-D score, controlling for propensity stratum and adjusting for other covariates. Model 1, the "naive" model, controls only for propensity score stratum in estimating the effect of violence on depression. The results suggest a clear harmful effect of violence: the coefficient for the violence "treatment" variable shows that across each of the 10 propensity score strata, CES-D scores for students who were exposed to violence were an average of 0.672 of a point higher than scores for students who were not exposed to violence. This corresponds to about 0.154 of a standard deviation, a modest but significant effect, with a p value less than 0.01. Separate tests for stratum-specific effects (not shown) produced no significant results, suggesting that the effect of violence on depression score is approximately the same across all propensity score strata.

Model 2 introduces a control for wave 1 CES-D score. Unsurprisingly, those with higher depression scores at an earlier time tend also to have higher scores later, reflected in the positive effect size; each point difference on the wave 1 CES-D corresponds to approximately 0.278 of a point change in predicted CES-D score at wave 3, and this result is highly statistically significant ($p < .001$). However, the estimated effect of wave 2 violence on Wave 3 CES-D score remains virtually unchanged, at 0.633 of a point. This finding is both substantively meaningful and statistically significant: even after controlling for preexisting depressive symptomatology, exposure to violence still engenders a negative effect on later depression status. Once again, tests of stratum-specific effects produced no significant results.

Model 3 additionally controls for gender, again with unsurprising results. Consistent with expectations, female respondents show an expected average CES-D score approximately 1.65 points higher than male respondents, a highly significant result that is in line with existing literature on

Table 3. Means of Treatment, Outcome, and Control Variables by Propensity Score Stratum.

	Stratum									
	1	2	3	4	5	6	7	8	9	10
Propensity Score (Range)	[0.0–0.05]	[0.05–0.075]	[0.075–0.1]	[0.1–0.15]	[0.15–0.25]	[0.25–0.35]	[0.35–0.5]	[0.5–0.55]	[0.55–0.75]	[0.75–1.0]
Treatment										
Violence at wave 2	0.026	0.053	0.076	0.156	0.242	0.370	0.423	0.380	0.647	0.849
Outcome										
CES-D score at wave 3	4.294	5.324	5.547	5.283	5.733	5.645	5.650	6.000	7.293	6.588
Controls										
Female	0.804	0.605	0.482	0.337	0.401	0.490	0.331	0.268	0.253	0.210
White	0.816	0.733	0.659	0.674	0.542	0.590	0.578	0.648	0.487	0.444
Black	0.139	0.195	0.254	0.244	0.332	0.330	0.289	0.310	0.387	0.407
Age 18–20 years	0.386	0.352	0.362	0.342	0.282	0.280	0.308	0.310	0.207	0.259
Age 21–22 years	0.356	0.421	0.409	0.422	0.437	0.420	0.407	0.394	0.453	0.432
Age 23–25	0.258	0.227	0.228	0.236	0.282	0.300	0.285	0.296	0.340	0.309
Less than HS	0.049	0.060	0.120	0.186	0.191	0.185	0.183	0.282	0.287	0.420
HS graduate	0.206	0.278	0.354	0.370	0.422	0.325	0.399	0.465	0.493	0.358
Some college	0.531	0.513	0.427	0.356	0.318	0.405	0.373	0.225	0.173	0.210
College graduate	0.213	0.149	0.099	0.088	0.069	0.085	0.046	0.028	0.047	0.012
Violence at wave 1	0.000	0.000	0.000	0.027	0.278	0.810	0.943	0.972	0.993	1.000
CES-D score at wave 1	2.498	3.420	3.967	4.521	5.522	4.820	5.490	5.437	6.920	7.100
College aspiration ^a	4.674	4.419	4.162	3.942	3.870	4.195	3.939	3.493	3.353	2.975
<i>n</i>	849	673	425	365	277	200	263	71	150	81

Note: CES-D = Center for Epidemiologic Studies Depression Scale; HS = high school.

a. Measured on a Likert-type scale ranging from 1 = *highly unlikely to go to college* to 5 = *highly likely to go to college*.

Table 4. Ordinary Least Squares Coefficients and Standard Errors (in Parentheses) for Effects of Wave 2 Violence Exposure on Wave 3 CES-D Score.

Variable	Model 1	Model 2	Model 3	Model 4	Model 5
Wave 2 violence	0.672** (0.230)	0.633** (0.219)	0.617** (0.215)	0.593* (0.265)	0.639* (0.263)
Wave 1 depression	—	0.278*** (0.015)	0.240*** (0.015)	0.240*** (0.015)	0.207*** (0.016)
Female	—	—	1.65*** (0.156)	1.64*** (0.170)	1.50*** (0.173)
Wave 2 violence × female	—	—	—	0.059 (0.386)	−0.024 (0.383)
Propensity score					
Stratum 1	—	—	—	—	—
Stratum 2	0.380 [†] (0.224)	0.132 (0.213)	0.499* (0.212)	0.496* (0.213)	0.377 [†] (0.214)
Stratum 3	0.586* (0.257)	0.177 (0.245)	0.764** (0.248)	0.760** (0.248)	0.509* (0.254)
Stratum 4	0.272 (0.273)	−0.280 (0.261)	0.571* (0.269)	0.568* (0.270)	0.180 (0.279)
Stratum 5	0.667* (0.305)	−0.207 (0.293)	0.576 [†] (0.297)	0.573 [†] (0.298)	0.281 (0.330)
Stratum 6	0.507 (0.347)	−0.127 (0.332)	0.485 (0.331)	0.480 (0.333)	0.336 (0.447)
Stratum 7	0.431 (0.321)	−0.384 (0.308)	0.517 (0.315)	0.514 (0.315)	0.273 (0.478)
Stratum 8	0.844 (0.540)	0.040 (0.514)	1.04* (0.514)	1.04* (0.515)	0.673 (0.638)
Stratum 9	1.96*** (0.407)	0.765 [†] (0.392)	1.84*** (0.399)	1.84*** (0.399)	1.56** (0.555)
Stratum 10	1.19* (0.531)	−0.049 (0.510)	1.11* (0.514)	1.11* (0.514)	0.438 (0.662)
Racial group					
White	—	—	—	—	—
Black	—	—	—	—	−0.017 (0.174)
Other	—	—	—	—	0.591* (0.244)
Age group (years)					
18–20	—	—	—	—	—
21–22	—	—	—	—	−0.276 [†] (0.167)
≥23	—	—	—	—	−0.599** (0.193)
Education					
Less than HS	—	—	—	—	0.828*** (0.241)
High school graduate	—	—	—	—	0.469** (0.172)
Some college (modal category)	—	—	—	—	—
College graduate	—	—	—	—	0.044 (0.237)
Wave 1 violence	—	—	—	—	−0.340 (0.358)
Wave 1 suicide ideation	—	—	—	—	1.27*** (0.220)
Wave 1 college aspiration	—	—	—	—	−0.236*** (0.069)
Constant	4.91	2.99	3.14	2.99	3.86
R ²	0.02	0.11	0.14	0.14	0.16

[†] $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

depression and gender (Hankin et al. 1998; Nolen-Hoeksema 2001; Piccinelli and Wilkinson 2000). Likewise, wave 1 CES-D score still predicts higher wave 3 CES-D score net of gender, violence exposure, and propensity score assignment: the coefficient estimate is about 0.240 of a point on the CES-D at wave 3 per point at wave 1, very close to the estimate from the previous model. Crucially, the effect of wave 2 violence exposure on wave 3 CES-D score also holds rather stable at 0.617 of a point, a finding that remains significant at the $p < .01$ level. Again, tests for stratum-specific effects gave no significant results. Controls for gender and previous depression score seem to modulate the effect of violence exposure on later depression score only slightly if at all and do not appear to substantially reduce the estimated significance of the effect.

In model 4, I introduce the violence $\times$ female interaction term, with surprising results. In this model, the regression coefficients for gender and previous depression score remain virtually unchanged, at 1.64 and 0.240, respectively; each remains highly significant. The estimate for the effect of wave 2 violence exposure on wave 3 CES-D score decreases slightly to 0.593 of a point, which represents 0.136 of a standard deviation, still a substantively meaningful effect and still statistically significant at the $p < .05$ level. However, the coefficient for the violence $\times$ female term (which can be interpreted as the difference between male and female respondents in how much wave 2 violence is expected to affect their wave 3 CES-D scores) is neither substantively large nor statistically significant. At an estimated value of 0.059 and a propensity score well above any conventional significance level, this result suggests that the effect of violence exposure on depression does not vary by gender; that is, male and female students exposed to violence manifest equally poor mental health responses several years after the fact. Tests for stratum-specific effects showed that neither the main effect of violence nor the interaction effect varied across propensity score strata.

Finally, in model 5, I introduce a set of control variables to adjust for the effects of other individual characteristics on depression score net of propensity score stratum. Race group shows some association with depression: whites and blacks appear to have similar average CES-D scores, but members of other races score more than half a point higher on average. As expected, depression

seems to decline with age, as 21- and 22-year-olds have a marginally significant ($p < .10$) 0.276-point lower average CES-D score than 18- to 21-year-olds, and 23- to 25-year-olds have a highly significant ($p < .001$) 0.599-point lower average CES-D score than 18- to 21-year-olds. Education shows a much clearer trend: those who never finished high school and those who finished high school but went no further have much higher average CES-D scores (respectively, 0.828 and 0.469; $p < .01$ for both) than those with college experience. Interestingly, completing college does not seem to affect depression net of the other variables; those with some college experience but no degrees are statistically indistinguishable from those who graduated college. This may be because the subjects are still young: many who report "some college" may still be in the process of completing their degrees, making them similar to those who finished college in all ways but age, whereas those who did not finish high school or who finished but did not go to college are more likely to have completed their education. Wave 1 suicidal ideation and wave 1 college aspirations both show significant and substantive associations with depression in the predicted directions. A surprising result is that net of propensity score for wave 2 violence and the other covariates, wave 1 violence exposure has no significant association with wave 3 CES-D score, suggesting either that recent violence is more important to mental health than previous violence or that most of the people who were exposed at wave 2 experienced a chronic pattern of exposure that remained in effect at wave 1. The most important finding in model 5, however, is that the estimated coefficients for our key predictor variables remain remarkably stable even after adjusting for the covariates: wave 2 violence exposure still seems to affect wave 3 CES-D score by about 0.639 of a point ($p < .05$), wave 1 CES-D score is still strongly associated with wave 3 CES-D score ($b = 0.207$, $p < .001$), women still have CES-D scores an average of 1.50 points higher than men ($p < .001$), and the statistically insignificant violence $\times$ female term still suggests that the effect of violence on male and female respondents is essentially identical.

DISCUSSION AND CONCLUSION

Summarizing these findings with respect to the study's key hypotheses reveals some substantively

interesting mixed conclusions. The clearest and perhaps most important finding regards the first hypothesis: that violence exposure during the school years exerts a negative causal effect on depression score for both men and women. The results presented here strongly uphold this prediction. Across all model specifications, I observe a distinct harmful effect of violence on depression score: between 0.672 and 0.593 of a point on the CES-D, representing between 0.154 and 0.136 of a standard deviation. This remarkably stable observed effect indicates that violence exposure exerts a corrosive influence on at least one generalized indicator of mental health and well-being for both men and women.

Also upheld was the prediction, based on extensive previous findings, that women in general would have higher levels of depression than men. Both overall and within all but the riskiest propensity score stratum, women show significantly higher scores on the CES-D than men do. Across all specifications of the propensity score models, the gap between men and women in predicted CES-D score both persists and remains relatively stable.

Last, and possibly most interesting, are the results regarding the third hypothesis. Surprisingly, the violence $\times$ female interaction effect never approaches statistical (or substantive) significance in any of the estimated propensity score models for depression score. This implies that violence has a negative causal effect on depression score and that women have a higher level of depression overall, but the effect of violence exposure is equally bad for both sexes. This finding is consistent with Nishina et al. (2005) and some others (Biggs et al. 2010; Lehman and Repetti 2007; Nishina and Juvonen 2005) who found that the pathways between peer victimization and negative mental health outcomes are the same for boys and girls.

These findings, although preliminary, contribute to the ongoing debate about whether effect sizes of violence on mental health for the two sex groups are different. My results imply that violence exposure affects depression among both men and women but that the size of the effect is the same for both. I suggest the possibility that many earlier findings of sex group differences may be detecting noncausal associations, due to the use of cross-sectional (e.g., DuRant et al. 2000; Lopez and DuBois 2005) or prospective cohort (e.g., Bond et al. 2001; Copeland et al.

2013) data designs and an attendant methodological inability to adequately address endogenous selection for violence exposure on the basis of students' preexisting mental health status or other relevant variables. In this study, I make the additional contribution of using rigorous and widely accepted methods for drawing causal inferences from observational data. Therefore, the null findings in this context may speak to the nature of the sex differences observed in previous studies. The results obtained here suggest that there exist no differences in the size of any true causal effect, defined in counterfactual terms.

It is necessary to stress the limitations of a study such as this. For one, this methodological design isolates exposure to violence at wave 2 of Add Health as its treatment. The descriptive results paint a clear picture, though, that exposure at wave 2 may not be an isolated incident but rather may be part of a long-term pattern of chronic or repeated exposure that begins at or before wave 1 and lasts over many years. Future studies ought to separate out the effects of chronic and isolated exposure to violence; there is reason to suspect that these different forms of exposure will have different sequelae for young people's mental health. Another limitation is necessarily imposed by the propensity score model's restriction to the region of common support. The exclusion of subjects from both the least risky and most risky parts of the propensity score distribution means that the subsample analyzed may not be representative of the population as a whole. However, because I detected no stratum-specific effects, it is possible that violence's effects on health may be similar across the spectrum of propensity for exposure. If anything, because many more observations were dropped from the bottom than from the top of the distribution, these effects estimates may be somewhat conservative: the effects of violence exposure on those least likely to experience it may be even worse than on those who fall within the common support restriction. Last, it is worth noting that the definition of the treatment may have implications for the null gender-difference finding. Although it is possible that men and women react similarly to the kinds of severe violence measured here, it is still plausible that other forms of victimization—including intimate partner violence, sexual assault, and other forms of interpersonal aggression—would differentially affect women.

A number of pragmatic implications stem from these findings, particularly with respect to possible

interventions. The first implication, obvious though it may sound, is that violence really is bad for young people's health. It is not just the case that those who are most at risk for mental and emotional health problems thanks to other circumstances are also likely to be victimized, although that does seem to be the case as well. Rather, it appears that the experience of violence itself exerts a harmful effect on depression score above and beyond the effects of violence's other correlates. This suggests that targeting students for immediate intervention after violence exposure might serve to mitigate some of the detrimental effects on depression and suicidal ideation that follow in the etiology.

Second, if it is the case, as the results suggest, that the effects of violence exposure are equally detrimental to men and women—and if we take seriously findings such as those of Nishina et al. (2005) that the process leading from victimization to poor emotional adjustment is the same for boys and girls—then it follows that some of the same posttraumatic mental health interventions may be effective for both gender groups. Rather than focusing on externalizing problems for boys and internalizing problems for girls (e.g., Osofsky 1995), these results imply that members of both gender groups may be at risk for depression as a consequence of violence exposure and that the same kinds of counseling and therapy may be useful for both. In short, the understanding that violence is bad for anyone may afford us opportunities to provide better help and recovery to everyone. This strikes this researcher as a valuable goal and as worthy of further investigation.

ACKNOWLEDGMENTS

I acknowledge the invaluable assistance of Steve Raudenbush and Kate Cagney in this article's early development, as well as helpful comments from the editors of *Society and Mental Health* and three anonymous reviewers, all of which helped clarify and strengthen the argument substantially. This research was supported in part by a grant from the American Educational Research Association, which receives funds for its AERA Grants Program from the National Science Foundation under grant DRL-0941014. The opinions reflect those of the author and do not necessarily reflect those of the granting agencies. This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill and funded by grant

P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Special acknowledgment is due to Ronald R. Rindfuss and Barbara Entwisle for assistance in the original design. Information on how to obtain the data files is available on the Add Health Web site (<http://www.cpc.unc.edu/addhealth>). No direct support was received from grant P01-HD31921 for this analysis.

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