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THE PROCESS OF SCHOOL VIOLENCE

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To Dad, Mom, Duncan, Jesse, Dave, and Fran – for support;

To Steve, Kate, and Jens – for guidance;

To Lynne, Linda, and Don – for inspiration;

And to Alli – for everything.

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ABSTRACT

Violence in schools is recognized as a serious public problem, but both its causes and its effects are difficult to study with scientific rigor. Existing research links school violence exposure to various negative outcomes – including socio-emotional problems like depression and suicidality, and academic problems like lower rates of post-secondary attainment – but because of violence's non-random distribution process, it is very difficult to determine whether these constitute causal effects. And theories about crime etiology predict that heterogeneous pathways lead to victimization for different reasons – for example, some students may be victimized because they place themselves at risk by voluntarily participating in violent situations, whereas a different type of students may be victimized because social isolation makes them "easy targets" – but endogeneity and confounding make these pathways difficult to examine. To complicate matters, violence exposure may have different effects on different kinds of students, and different types of students may vary in how much they benefit from schools' protection. In this dissertation, I address these and related problems using data from the National Longitudinal Study of Adolescent Health. Leveraging the school-based longitudinal structure and using techniques to draw causal inferences from observational data, I test several hypotheses about the effects of violence exposure on different types of students and the role of the school in violence prevention. I find that violence increases depression for both males and females; that students who experience violence early in life fare worse than those who do not; that students who experience isolated exposures suffer acute shocks to their well-being; that students who suffer repeat victimizations fare worse overall but are less affected by each specific exposure; that students who perpetrate violence and students with few friends are both more likely to be victimized; and that positive school climate protects socially isolated students from

victimization, but has no such protective effect on students who are perpetrators. Besides confirming predictions about violence derived from life course theory, routine activities theory, and others, this dissertation contributes to a growing body of methodologically rigorous evidence suggesting that violence exposure itself exerts negative causal effects on students' lives.

CHAPTER ONE

Introduction: Why Do We Need a Special Theory of School Violence?

Catastrophic examples of student-committed violence in schools, such as the 1999 shooting at Columbine High School in Colorado and the 2007 shooting at Virginia Tech in Blacksburg, Virginia, have captured public awareness over recent years. Public attention is also increasingly focused on other forms of student aggression, such as the insidious problems of bullying and persistent incivility (Smith & Thompson 1991). Academic scholarship, too, has recognized the severity of the school violence problem. A 2010 special issue of Educational Researcher devoted to school violence revealed that substantial levels of violence, theft, bullying, intimidation, and related behaviors persist in American schools despite a decline in incidence across the 1990s (Mayer & Furlong, 2010); that certain individual characteristics – including male gender, obesity, physical or learning disability, and non-hetero sexual orientation – are associated with an increased risk of violent victimization (Swearer, Espelage, Vaillancourt, & Hymel 2010); and that these problems lead to serious school-, classroom-, and personal-level disruptions such as fear, anxiety, school avoidance, classroom disorder, weakened social bonding, impaired cognitive and academic development, and altered social-emotional trajectories (Cornell & Mayer 2010).

Implicit in all this attention is an understanding of school violence as a distinctive phenomenon, deserving of special and separate attention by virtue of some set of unique characteristics. However, few address this assumption directly. Rarely is it asked: What, if anything, particularly distinguishes school violence? Why should we consider it a singular, separate kind of violence? Do we really need a special theory of school violence, or can we just

understand it as being much like any other kind of violent interaction? To borrow a phrase: "Why is this violence different from all other violence?"

In this introduction I will briefly establish the need for a special theory of school violence by investigating what makes violence in schools unique, distinct from violence in general and even from other types of violence among youth. Some of this is attributable to simple differences in empirical patterns, such as higher rates of theft and nonfatal assault (Robers, Zhang, Truman, & Snyder 2012; Snyder & Sickmund 1995; Whittaker & Bastian 1991) and lower rates of severe violence but higher prevalence of mild to moderate victimization (Benbenishty & Astor 2005; Cook, Gottfredson, & Na 2010) compared to other contexts. But I will call particular attention to the fact that school violence may have uniquely harmful developmental effects (Arseneault, Walsh, Trzesniewski, Newcombe, Caspi, & Moffitt 2006; Copeland, Wolke, Angold, & Costello 2013; Nansel, Overpeck, Pilla, Ruan, Simons-Morton, & Scheidt 2001). I will also point to ways that school violence, unlike other forms of violence, is uniquely structured by the school's special social and institutional context (e.g. Gottfredson 2001, Gottfredson & DiPietro 2011). Doing so, I will explain the particular contributions of this dissertation project in elucidating the distinctive characteristics and properties of school violence.

SCHOOL VIOLENCE & GENERAL VIOLENCE

School violence is distinct from other kinds of violence on many grounds. It manifests more frequently and with higher relative prevalence: 2010 National Crime and Victimization Survey (NCVS) data show that more victimizations were committed against students ages 12-18 at school than away from school; that rates of nonfatal violent victimization among school-age students were higher in school than out of school; and that a higher percentage of school-age students report fearing for their safety "at school or on the way to and from school" than "out of

school" during the school year – a difference that is especially exacerbated for urban students, who may be more likely to be in a location other than home or at a supervised activity when not in school (Robers, Zhang, Truman, & Snyder 2012). Also using NCVS data, Cook, Gottfredson, and Na (2010) find in-school and out-of-school victimization rates are roughly equal, but argue that since students spend less than 20% of their time in school, students on average are more likely to be victimized during an hour spent at school than an hour spent elsewhere.

Qualitatively, school violence is unique in its intensity, particularly its repetition over months and years among the same individuals and groups (Olweus 1993, 2013); and in its manifestations, since certain types of violence – including forcible theft, fist-fighting, and nonfatal assault – are disproportionately more likely to occur in schools (Whitaker & Bastian 1991, Gottfredson & DiPietro 2011), although more severe types of violence such as homicide are much less common (Dinkes, Kemp, Baum, & Snyder 2009). School violence is also unique in its spatial concentration: Snyder and Sickmund (1995) found that in 1991 – during a peak in rates of violence nationally, especially for school-age youth – 56% of all juvenile victimizations occurred in or around school; although these differences were largely attributable to thefts rather than violent victimizations, the authors point out that this high level of concentration is unprecedented in any other context, and certainly has no parallel among adults.

SCHOOL VIOLENCE & YOUTH VIOLENCE

Violence in schools differs even from other kinds of violence among youth. Schools provide a different opportunity structure for violence than do other youth settings: greater time spent at risk (Carnegie Council on Adolescent Development 1993, Gottfredson 2001), different sets of sanctions and constraints (McFarland 2001), and a tremendous variation in these factors across schools (Benbenishty & Astor 2005, Gottfredson & Gottfredson 1985). And the social

structure of schools differs from neighborhood and other elective associations in ways that shape opportunities for violent behavior, such as ascribed roles and peer groups (Parsons 1959) and ratios of different age groups and sex groups (Dreeben 1968). Of course, schools are relatively advantaged in some of their structural characteristics – in particular the ratio of children to adults (Dreeben 1968) and the specialized training those adults receive for the task of supervising children, which likely explains the lower rates of serious and fatal victimization.

Although NCVS data show that young people in general are two to three times more likely than older people to be victims of violence (Whitaker and Bastian 1991), there are distinct differences in patterns of violent behavior in school when compared with other settings. Gottfredson (2001) estimates that teens spend about 18% of their time in or around school, but finds in the NCVS that 37% of violent incidents against teens occur in school, as well as 81% of thefts. Beyond this, data from the Youth Risk Behavior Surveillance System (YRBSS) survey suggest that more than half of youths who carry weapons do so on school property, and approximately 40% of youth fights occur on or around school grounds (Centers for Disease Control and Prevention 1995). The upshot of these statistics is that the amount of youth violence tied to the school context is disproportionately high. Rather than being a safe haven, in many cases the school is a place where the risks of particular kinds of violent exposure are elevated (Gottfredson & DiPietro 2011).

As noted, it does appear to be the case that violence committed in school is less likely to escalate – especially to escalate to fatalities – than is violence committed out of school (Soulé, Gottfredson, & Bauer 2008). NCVS data show that, despite the level of concern generated around school rampage shootings (Newman et al 2004), the ratio of murder rates out of school to murder rates in school is approximately 100 to 1 (Cook, Gottfredson, & Na 2010). Those violent

crimes committed against students in schools tend to be simple attacks without weapons which rarely result in worse than superficial physical injuries (Chandler, Chapman, Rand, & Taylor 1998). This is likely due to the increased structure and supervision schools provide; students may chafe against the strictures of school control, but the level of oversight means violence can be prevented or interrupted before it turns deadly. On the flip side of this, the last two decades have seen an increasing reliance on police or security presence to deter in-school behavioral infractions (e.g. Noguera 1995). Devine (1996) argues that high security and "zero-tolerance" policies cause schools to resemble jails – featuring metal detectors or body scans, police supervision, cameras in classrooms, etc. – which leads students to adopt the mentality of prisoners. In this paramilitary environment, the logic of violence becomes incorporated into the everyday actions and praxes of both students and teachers (Devine 1996). Additionally, zero tolerance disciplinary practices in schools have been linked to a drastic increase in students' entry into the criminal justice system – often for relatively minor infractions, which once would have been handled by school administrators – and this trend disproportionately affects poor and minority students in inner cities (Advancement Project and The Civil Rights Project 2000).

UNIQUE STRUCTURES AND CONSEQUENCES

Perhaps what makes school violence most distinctive, though, is the school itself. Schools are required to carry out an uncommonly burdensome task: to take people in the demographic category at the highest risk for violence (Gottfredson 2001), aggregate them in a single building together all day long, and somehow endeavor to keep the peace. This strongly implicates school functioning and school climate in the process of school violence. School violence, perhaps more than other kinds of violence, may vary according to the extent to which its institutional context is well-functioning (Khoury-Kassabri, Benbenishty, & Astor 2005). This suggests that an analysis

of the process of school violence should pay special attention to school structure and culture itself, making this a ripe target for sociological perspective and insight.

Another key characteristic of school violence, linked to its institutional setting, is its distinctly harmful consequences for students' health and development. Because school violence affects a younger population, and because it affects them in what is otherwise presumed – rightly or wrongly – to be a safe space, a life course perspective suggests that it has developmental consequences that other kinds of violence do not (Rigby 2003). In particular, there is reason to believe that exposure to violence in school may have an effect on students' mental health – including concerns like depression (DuRant et al 2000, Lehman & Repetti 2007) and suicidal ideation (Copeland et al 2013) – and on their academic outcomes in both the short and long term (Sharkey 2010). Burdick-Will (2013) finds that violence in schools is more disruptive to students' academic performance than is non-school local violence, saying "There seems to be something unique about violence that takes place at school above and beyond what students experience around their homes or in the neighborhood around school" (355), and posits that school violence's disruption of classroom functioning accounts for its outsize influence.

This also raises questions about whether there exist variations in vulnerability to the violence that happens in schools. If school violence is uniquely harmful, some students may be at greater risk of harm – or at risk of greater harm – than others. There is reason to believe that the harmful effects of school violence may vary as a function of students' background characteristics such as sex (e.g. Bond et al 2001) and life experiences (Harding 2009; Wodtke, Harding, & Elwert 2011). This suggests that to understand school violence requires disentangling these various contingent patterns.

OUTLINE OF THE DISSERTATION

This dissertation addresses questions about the kinds of harm done by violence in schools, the kinds of people for whom this violence is most harmful, the kinds of situations in which violence is most likely to occur, the effects of violence exposure, and what schools can do to get a handle on this problem.

Chapter 2 takes on the question of whether violence exerts a harmful effect on students' mental health, and also examines whether this effect varies by sex. Many studies associate adolescent violence exposure with later mental health outcomes; but because violence cannot be randomly assigned, few establish causality. Additionally, few directly test whether violence affects boys' and girls' psychological health differently. I hypothesize that 1. violence has a negative causal effect on depression; and 2. the effects of violence on depression are worse for females than for males. In the chapter, I use Add Health data to build multilevel propensity score models with Wave 1 predictors in a cross-lagged 2-level HLM estimating the effects of Wave 2 violence on Wave 3 outcomes. Results 1. confirm that females have higher depression scores than males net of other factors; 2. show that violence exposure raises average depression scores for both sexes on average; and 3. uncover a moderate but statistically insignificant difference by sex in the size of violence's effect on depression which appears, surprisingly, to be worse for males. The results also reveal one significant factor that overwhelmingly predicts violence exposure at Wave 2: violence exposure at Wave 1.

Given the persistence of violence exposure over time, Chapter 3 draws on cumulative disadvantage and life course theories to determine if violence's effects on students' academic and mental health outcomes vary as a function of whether the students' exposure is isolated or repeated. I test three hypotheses about the effects of time-specific exposure to violence as a

function of previous history of exposure: 1. those who are exposed to violence earlier in life will have lower rates of college enrollment and higher rates of depression and suicidal ideation than those for whom exposure comes later; 2. among those who report no early violence exposure, later isolated exposure to school violence will negatively impact later outcomes (i.e. will cause lower college enrollment rates and higher rates of depression and suicidality); and 3. those who do report previous violence exposure, despite faring worse overall, will to be less susceptible to specific negative effects from any one instance of later exposure to school violence – that is, repeat exposure will not significantly affect their college enrollment or mental health patterns. I test these hypotheses using data from Add Health in three-wave cross-lagged models similar to the previous chapter, but take the additional step of separating those who report Wave 1 violence exposure from those who do not and estimating separate propensity score models for each group. The results support Hypotheses 1 and 3 and partially support Hypothesis 2: 1. those who report Wave 1 violence exposure manifest worse results on all outcomes at Wave 3 than those who were not exposed to Wave 1 violence; 2. Wave 2 violence exposure negatively affects college enrollment and suicidal ideation (but oddly, not depression) on those who were not exposed to violence at Wave 1; and 3. the effects of Wave 2 violence exposure on those who were exposed to violence at Wave 1 are generally insubstantial.

Chapter 4 more directly addresses the school context. Victimization in schools, as established, is a function of students' individual characteristics as well as schools' organizational functioning; certain kinds of students are at higher risk of victimization than others, and more favorable school climate should exert a protective effect. However, there likely exist different configurations of individual-level risk factors that predict victimization, and school efficacy may not affect these different kinds of students in the same way. Drawing on routine activities theory,

this chapter hypothesizes that 1. students who are habitual perpetrators of violence are more likely to be victimized by violence themselves; 2. students who are socially isolated, with few friends, are more likely to be victimized by violence; and 3. more favorable school climate – measured as average subjective sense of safety – lowers the likelihood of victimization among socially isolated students, but not among habitual perpetrators. Using data from the first two waves of Add Health, I construct various 2-level HLMs to test whether victimization varies as a function of level-1 perpetration and social isolation, and whether the size of these associations varies at level 2 with school-wide sense of safety. Results broadly support all three hypotheses: 1. students who perpetrate violence at Wave 1 are much more likely to be victims of violence at Wave 2, 2. students with fewer received friendship nominations at Wave 1 are more likely to be victimized at Wave 2, and 3. positive school climate is a protective factor for socially isolated students (reducing the size of the association between social isolation and later victimization) but not for students who are perpetrators themselves.

Chapter 5 concludes that the results of all these analyses tell a consistent story. School violence impacts mental health and academic achievement, and is associated with raised probability of more victimization later. School violence exposure is harmful to both male and female students, and is harmful no matter when it happens; earlier exposures seem to have larger aggregate consequences than later exposures, but may desensitize students to additional harmful effects of later violence by setting them on a more negative trajectory, and later exposure is still harmful in a variety of ways. And students' risk of victimization is a function of heterogeneous personal characteristics – traits that also alter schools' capacity to prevent student victimization. In other words, no matter who you are, school violence is bad for you – but for some of the most vulnerable students, schools can take effective steps to help.

CHAPTER TWO

Effects of Adolescent Violent Victimization on Adult Depression: Testing Heterogeneity for Males and Females[1]

Violence exposure – and the associated persistent states of fear (Cornell & Mayer 2010) and helplessness (Peterson & Seligman 1983) that may 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, & Earls 2005). In these and other studies, mental health is implicitly or explicitly identified as the culprit for mediating the associations between violence exposure and poor outcomes (e.g. Song, Singer, & Anglin 1998). Yet direct connections between violence exposure and poor mental health are not typically examined using research designs strong enough to overcome potential endogenous and confounding factors. Violence has been conceptually linked to depression and other poor mental health outcomes (e.g. Biggs et al 2010, DuRant et al 2000, Lehman & Repetti 2007, Rigby 1999), but these arguments rarely establish credible causal relationships. What's missing has been a serious attempt to analyze the effects of school violence exposure on mental health, using methods rigorous enough to account for both the pre-treatment covariates that differentially predict violence exposure for individuals and the fact of violence's uneven distribution across social settings.

Additionally, patterns of violence exposure among school-age youth are highly gendered (Benbenishty & Astor 2005, Popp & Peguero 2011). In particular, boys are far more likely than girls to be exposed to physical violence (Robers et al 2012, Rose & Rudolph 2006). However, it

[1] An earlier version of this chapter was published as:
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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 & DuBois 2005, Paquette & Underwood 1999) arguing that violence affects the sex groups differently and others (e.g. Biggs et al 2010, Lehman & Repetti 2007, Nishina & Juvonen 2005; Nishina, Juvonen, & Witkow 2005) finding no such differences.

Here, I examine whether the experience of violence can be 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 show 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 & Mayer (2010) find 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 and colleagues (2001) find that past victimization predicts symptoms of anxiety and depression in late adolescence but previous emotional problems do not predict later victimization; Lehman & Repetti (2007) find that victimization

leads to changes in students' mood and self-esteem that influence perceptions of later interactions with parents and others; and Sharkey and colleagues (2012) find 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 and colleagues (2013), published in *JAMA Psychology*, which prospectively tracks a panel of 1420 students in North Carolina, each interviewed 4 to 6 times between the ages of 9 and 16. Following this panel into young adulthood (ages 24-26) and controlling for childhood psychiatric problems and family hardships, Copeland et al find victims of violent bullying at highly elevated risk of psychiatric problems such as depression, agoraphobia, panic disorders, generalized anxiety, and suicidal ideation or attempt (Copeland et al 2013). The worst effects appear for those who were both bullies and victims, suggesting that suffering multiple vulnerable conditions increases vulnerability to negative psychological and behavioral outcomes.

However, because of the nature of the treatment – violence – few studies can address the effects of exposure on mental health in a methodologically rigorous way. Even well-designed prospective cohort studies like 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 – and other forms of unobserved confounding. Two notable exceptions that manage to suggest causal effects of violence exposure on emotional and behavioral outcomes are Sharkey, Tirado-Strayer, Papachristos, and Raver (2012), which leverages variation in the timing of neighborhood homicides relative to assessments conducted for the Chicago School Readiness Project and finds that recent local homicides impair children's emotional regulation and cognitive functioning; and Bingenheimer, Brennan, and Earls (2005), which – in a strategy similar to this paper's – uses

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 increases 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.

Of course, the link between violence exposure and social adjustment is not simple or unidirectional. For example, not all victimized students are passive or helpless; predictions from routine activities theory (Cohen & Felson 1979) and other social-ecology approaches suggest that many students are exposed to violence because they are violent themselves, and therefore tend to spend time in risky situations with other violent peers. This process will be investigated directly in Chapter 4; for the discussion here, we will not differentiate between those who are exposed to violence because they are hapless targets and those who are exposed because they are violent actors themselves.

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 & Wilkinson 2000): women consistently report more frequent and more severe symptoms. An additional but separate concern is that many studies – including 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 (Klomek et al 2010; Rigby 1999), or between "internalizing" and "externalizing" symptoms of exposure (Osofsky 1995). Some suggest 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 & DuBois 2005). It is also 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 & Underwood 1999). These differences are typically attributed to gendered coping or adaptation mechanisms (Clampet-Lundquist, Edin, Kling, & Duncan 2011), or to gendered differences in routine activities creating gender-specific risks of victimization (Popp & Peguero 2011).

Despite this attention to the intersection of gender and violent victimization, few studies directly test 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 provide mixed results. Some detect different effects of victimization across gender groups. Bond and colleagues (2001) prospectively survey middle school students in the UK and find that repeated victimization predicts symptoms of anxiety or depression for girls but not boys. Paquette and Underwood (1999) find, 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) find 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 and colleagues (2010) test gender as a moderator for the effect of victimization on emotional well-being; they find no significant differences in the trajectory of victimization and no significant gender moderating of the association between victimization and affect, suggesting victimization has similar effects on

boys' and girls' emotional well-being. Lehman and Repetti (2007) find 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, Juvonen, and Witkow (2005), using multilevel and structural equation modeling on a middle school sample, find 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 chapter I will address both of these apparent lacunae: the lack 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 employ three waves of longitudinal data from the National Longitudinal Study of Adolescent Health. This follows the example of Bingenheimer, Brennan, and Earls (2005), one of the few existing studies to apply propensity score modeling to the effects of individual violence exposure on later outcomes.

Based on the summarized literature, I test two main hypotheses in this paper: 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 females than for males, observed as a significant female × 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 & Wilkinson 2000), that females will report higher levels of depression in young adulthood than males; but this is more a "reality-check" than a hypothesis per se.

The empirical findings, described in the following, confirm that females generally fare worse than males 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; the female × violence interaction effect is nonsignificant in all models, and direct comparison of average treatment effects suggests that if anything the effect is worse for males. This indicates that school violence exposure is detrimental to mental health for both females and males.

METHOD

Analytic Approach

There are three main obstacles to estimating the effects of violence exposure on mental health in later life: 1. Non-random assignment of treatment to individuals; 2. Non-independent likelihood of treatment assignment across social contexts (here, schools); and 3. Ascertaining a correct temporal order of pre-treatment covariates, treatment exposure, and outcomes or effects.

The first of these – non-random 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 of violence exposure. These include observable attributes such as male gender (Olweus 1993), non-hetero sexual orientation (Swearer, Espelage, Vaillancourt, & Hymel 2010)

or perceived insufficient displays of masculinity (Klein 2012), and obesity (Swearer et al 2010); but also include preexisting mental health complications or factors that predict later mental health problems with or without violence exposure, such as disadvantaged social background (Morenoff, Sampson, & Raudenbush 2001; American Academy of Pediatrics 2000), anxiety, insecurity, negative self-concept, internalizing problems, physical weakness, and peer rejection or social unpopularity (Hodges & 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 post-treatment 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 & 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 & 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 where 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) show 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, & Ridder 2003; Rubin & Thomas 1996). Thus, given proper modeling, the mean difference in outcomes between the treated and untreated groups 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 – non-independent likelihood of treatment assignment – stems from the fact that propensity for violence exposure (π) 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 (Burstein 1980, Raudenbush & Bryk 1986), which requires that every subject's error term in a model is independent of every other subject's.

To solve this problem, the $\hat{\pi}$s must be estimated using a hierarchical model (Raudenbush & Bryk 2002). In this case, because estimating propensity scores requires using a model with a binary outcome – exposed or unexposed – I employ hierarchical binary logistic regression, a species of hierarchical generalized linear models (HGLM). Because propensity for treatment assignment is expected to vary both within and across schools, a 2-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. Using cross-sectional data, on which case-control studies of violence's effects are based (e.g. Due et al 2005, Williams et al 1996), raises the concern of whether our conceptual ordering of events actually aligns with their ontological ordering. Based on 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 pre-treatment 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 pre-treatment 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 this kind of data. (Klomek et al's 2010 review compares cross-sectional to 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) – make 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, Brennan, and Earls (2005), I use data drawn from three separate waves of a longitudinal study. I control for pre-treatment 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, since 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 model which controls for pre-treatment 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 the National Longitudinal Study of Adolescent Health (Add Health), one of the largest and most comprehensive surveys of adolescents ever undertaken. Add Health is a longitudinal study that follows a nationally representative sample of adolescents in the United States. The study began during the 1994-95 school year with respondents in grades 7-12, and has continued through three successive waves of follow-up in 1996, 2001-02, and 2007-08. A sample of 80 high schools and 52 middle schools from the US 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 US 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 paper, as noted, I draw on the first three waves of Add Health data, gathered in 1994-95, 1996, and 2001-02. By using pre-treatment 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 11^{th} grade or lower at Wave 1, indicating that they would be no further along in school than 12^{th} 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 3766 students – 2031 female and 1735 male – in 132 schools, for whom I estimate propensity scores.

To account for bias resulting from differential likelihood of censoring, I estimate weights based on inverse probability of retention across Waves 2 and 3 and apply them to the estimates below.

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 experience with violence in the last 12 months. Specifically, students were given 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, males' and females' 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, & Raudenbush 2008; Wodtke, Harding, & 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 in order to establish a baseline effect.

Depression, the key outcome variable, is measured with a modified version of the Center for Epidemiologic Studies Depression (CES-D) scale (Radloff 1977). The standard CES-D instrument 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 or older, but research shows that the CES-D measures are valid for multiple categories of adolescents in the US – 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 of developing depression or anxiety disorders. Add Health includes a modified version of the CES-D scale in all of its survey waves; at Wave 3, the survey instrument contains 10 questions from the different categories of the CES-D scale. 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 pre-treatment 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.

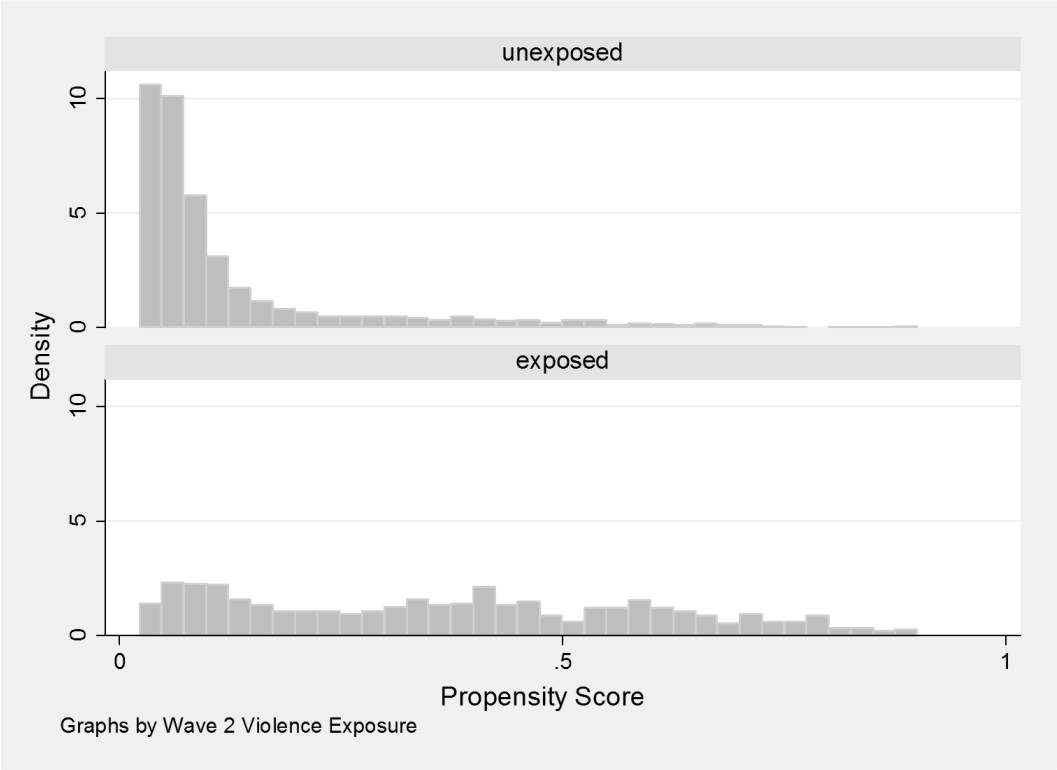
Propensity Score Estimation and Stratification

I employed a maximum-likelihood hierarchical logistic regression model to predict propensity scores $\hat{\pi}$ for all members of the dataset who met the criteria outlined above. As noted, it is best to use a wide variety of predictor variables when constructing propensity score models in order 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 $\alpha = 0.15$ 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, then employed the selected variables in estimating a 2-level model with the 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 that were not significant at $\alpha = 0.15$ in the stepwise model but that extant research identifies as relevant for violence exposure. This resulted in a final model which estimated each subject's $\hat{\pi}$ using 39 level-1 covariates and a single level-2 covariate. (Details of the final model are available upon request.)

Having calculated $\hat{\pi}$s 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 $\hat{\pi}$ which includes both treated and untreated observations. Figure 2.1 shows the probability density distribution of $\hat{\pi}$ for the treated and untreated groups, and reveals 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 us with a final sample of 3354, of whom 2783 were unexposed and 571 were exposed at Wave 2.

FIGURE 2.1: Probability Density Distribution of $\hat{\pi}$ for Treated and Untreated Groups



Source: National Longitudinal Study of Adolescent Health

I divided the 3354 subjects into 10 strata based on 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. Table 2.1 gives stratum cut points and frequency counts for the treated and untreated groups within strata.

TABLE 2.1: Frequency Counts per Propensity Score Stratum

Stratum	Propensity score (range)	Unexposed (N)	Exposed (N)	Total (N)
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		2783	571	3354

At $\alpha = 0.05$, 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 pre-treatment covariates. Table 2.2 displays the weighted average propensity score within each stratum for both the treated and the untreated groups, as well as t-tests for difference, showing that the propensity score is balanced across all 10 strata.

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 OLS regression models. (Alternate specifications of functional form, such as Poisson, produced substantively

TABLE 2.2: Mean [SE] of Propensity Scores within Strata, by Treatment Assignment

Strat	Range	Z = 0	Z = 1	Diff (t, p)
1	[0.0-0.05)	0.039 [0.001]	0.038 [.0002]	0.001 (0.57, 0.567)
2	[0.05-0.075)	0.063 [0.001]	0.062 [.0003]	0.001 (0.93, 0.350)
3	[0.075-0.1)	0.088 [0.001]	0.087 [.0004]	0.001 (1.36, 0.175)
4	[0.1-0.15)	0.122 [0.002]	0.120 [.001]	0.001 (0.71, 0.475)
5	[0.15-0.25)	0.195 [0.004]	0.189 [.002]	0.006 (1.30, 0.194)
6	[0.25-0.35)	0.304 [0.004]	0.299 [.003]	0.005 (1.01, 0.314)
7	[0.35-0.5)	0.421 [0.004]	0.419 [.004]	0.002 (0.37, 0.714)
8	[0.5-0.55)	0.531 [0.003]	0.526 [.002]	0.005 (1.27, 0.207)
9	[0.55-0.75)	0.613 [0.004]	0.628 [.007]	-0.015 (-1.90, 0.060)
10	[0.75-1.0]	0.779 [0.008]	0.771 [.016]	0.008 (0.43, 0.672)

similar findings.) In these models I employ 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 to 20, 21 to 22, and 23 to 25). To address one of this paper's central questions – 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 × female"). Since there is a well-established association between mental health and educational attainment (Mirowsky & Ross 2003), I also control for educational attainment at Wave 3 as a 4-category ordinal variable based on 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 et al 2007), I control for respondents' Wave 1 indication of whether or not they think they are likely to go to college. More details are presented in the results section below.

RESULTS

Descriptive Findings

It is interesting at the outset to compare the treated and untreated groups on some of the 125 identified pre-treatment covariates. Table 2.3 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 different from those who do. Demographically, compared to 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, as well as poorer mental and physical health. In temperament, the unexposed are much less likely to engage in 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. 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 & 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 2.3: Comparison of Treated and Untreated Groups on Selected Wave 1 Covariates

Pretreatment Covariate	Unexposed Mean	Exposed Mean	Difference	T-Test Statistic	p
Demographic Traits:					
Proportion female	0.5764	0.3739	0.2025	9.76	< 0.001
Proportion white	0.7049	0.5764	0.1285	6.57	< 0.001
Proportion black	0.2179	0.3173	-0.0994	-5.57	< 0.001
Age at Wave 1	14.95	15.17	-0.22	-3.17	0.002
Home Environment:					
Good relationship with mother	0.857	0.813	-0.044	-2.89	0.004
House kept in good condition	0.891	0.783	0.108	7.68	< 0.001
Feel unsafe in neighborhood	0.037	0.072	-0.035	-4.06	< 0.001
School Environment:					
Feel close to people at school*	2.22	2.41	-0.19	-4.63	< 0.001
Feel happy at school*	2.23	2.55	-0.32	-6.67	< 0.001
Feel teachers treat students fairly*	2.13	2.46	-0.33	-7.60	< 0.001
Mental Health:					
Frequently feel like a failure	0.027	0.067	-0.040	-5.25	< 0.001
Frequently feel unhappy	0.186	0.271	-0.085	-5.05	< 0.001
Seriously contemplated suicide	0.120	0.165	-0.045	-3.16	0.002
Physical Health					
Frequently feel sick	0.035	0.067	-0.032	-3.80	< 0.001
Frequently have poor appetite	0.121	0.170	-0.049	-3.47	< 0.001
Frequently have trouble sleeping	0.223	0.283	-0.060	-3.38	< 0.001
Temperament & Behaviors					
Describe self as impulsive	0.356	0.477	-0.121	-5.96	< 0.001
Sexually active	0.278	0.515	-0.237	-12.27	< 0.001
Regular smoker	0.157	0.250	-0.093	-5.79	< 0.001
Binge drink at least once a month	0.069	0.188	-0.119	-9.90	< 0.001
Ever use any illegal drug	0.222	0.441	-0.218	-11.88	< 0.001
Academic Characteristics					
Ever held back a grade	0.175	0.312	-0.137	-8.22	< 0.001
Ever been expelled from school	0.027	0.103	-0.076	-9.16	< 0.001
Frequent trouble with homework	0.242	0.427	-0.185	-9.84	< 0.001
Feel likely to go to college*	4.28	3.79	0.49	10.49	< 0.001

Table 2.3 continues below

Table 2.3 continued

Peer Influences					
Have close friend who smokes	0.380	0.566	-0.186	-8.94	< 0.001
Have close friend who drinks	0.489	0.682	-0.193	-9.20	< 0.001
Social Support					
Feel people dislike you	0.047	0.118	-0.071	-7.16	< 0.001
Feel lonely	0.065	0.115	-0.050	-4.45	< 0.001
Frequent trouble w/ other students	0.145	0.253	-0.108	-6.87	< 0.001
Violence Exposure					
Witnessing or Victimization	0.141	0.623	-0.482	-30.26	< 0.001

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

By contrast, Table 2.4 shows 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. Since the propensity score is the predicted risk for exposure to violence, the trend towards 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 8th 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 upwards 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: females and whites are over-represented in the lower-risk strata and under-represented in higher-risk strata; those in higher-risk strata attain less education by Wave 3, and those in lower-risk strata attain more; 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 1947 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

TABLE 2.4: Means of Treatment, Outcome, and Control Variables (Measured at Wave 1) by Propensity Score Stratum

	Stratum									
p-score range:	**1 [0.0 - 0.05)**	**2 [0.05- 0.075)**	**3 [0.075- 0.1)**	**4 [0.1- 0.15)**	**5 [0.15- 0.25)**	**6 [0.25- 0.35)**	**7 [0.35- 0.5)**	**8 [0.5- 0.55)**	**9 [0.55- 0.75)**	**10 [0.75- 1.0]**
Treatment										
Viol2	0.026	0.053	0.076	0.156	0.242	0.370	0.423	0.380	0.647	0.849
Outcome										
CES-D3	4.924	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
18-20	0.386	0.352	0.362	0.342	0.282	0.280	0.308	0.310	0.207	0.259
21-22	0.356	0.421	0.409	0.422	0.437	0.420	0.407	0.394	0.453	0.432
23-25	0.258	0.227	0.228	0.236	0.282	0.300	0.285	0.296	0.340	0.309
lt HS	0.049	0.060	0.120	0.186	0.191	0.185	0.183	0.282	0.287	0.420
HS grad	0.206	0.278	0.354	0.370	0.422	0.325	0.399	0.465	0.493	0.358
some coll	0.531	0.513	0.427	0.356	0.318	0.405	0.373	0.225	0.173	0.210
coll grad	0.213	0.149	0.099	0.088	0.069	0.085	0.046	0.028	0.047	0.012
Viol1	0.000	0.000	0.000	0.027	0.278	0.810	0.943	0.972	0.993	1.000
CES-D1	2.498	3.420	3.967	4.521	5.522	4.820	5.490	5.437	6.920	7.100
coll aspir*	4.674	4.419	4.162	3.942	3.870	4.195	3.939	3.493	3.353	2.975
N	849	673	425	365	277	200	263	71	150	81

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

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. We will return to this finding in the conclusion.

Overall, this suggests serious distinctions between those who are likely to be victimized and those who are not. Even before Add Health Wave 2, those who would 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 endogenous treatment assignment net of these factors: those likeliest to be exposed to violence are, at baseline, also at greater risk of later problems whether or not they experience violence. We also see stark patterns

of pretreatment variables' association with Wave 2 violence exposure propensity – 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. Again, this is consistent with the concept that the exposed are victims of circumstance as well as the possibility that they are students who "opt in" to violent situations by being violent themselves; in either case, those exposed at Wave 2 come from more troubled backgrounds than the unexposed. The starkest differences are for previous violence exposure. 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 2.5 shows results from the OLS regression models predicting the effect of Wave 2 violence exposure on Wave 3 CES-D score after controlling for propensity stratum, weighting for inverse probability of retention at follow-up, and adjusting for other covariates. Model 1, the "naïve" 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 .701 of a point higher than scores for students who were not exposed to violence ($p < 0.01$). This corresponds to about .146 of a standard deviation – a modest but substantively important effect. Separate tests for stratum-specific effects (not shown) produced no significant results, suggesting that the effects of violence on depression score are not significantly different across 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 scale corresponds to approximately .278

TABLE 2.5: OLS Coefficients and [Standard Errors] for Effects of Wave 2 Violence Exposure on Wave 3 CES-D Score († p<0.1, *p<0.05, **p<0.01, ***p<0.001)

		Model 1	Model 2	Model 3	Model 4	Model 5
Wave 2 Violence		.701** [.260]	.664** [.252]	.587* [.243]	.655* [.283]	.650* [.279]
Wave 1 Depression		—	.278*** [.019]	.238*** [.019]	.238*** [.019]	.208*** [.020]
Female		—	—	1.67*** [.174]	1.71*** [.184]	1.57*** [.191]
Wave 2 Violence × Female		—	—	—	-.210 [.446]	-.227 [.442]
Propensity Score:	Stratum 1	—	—	—	—	—
	Stratum 2	.377 [.234]	.131 [.224]	.532* [.227]	.542* [.228]	.4567* [.229]
	Stratum 3	.505† [.265]	.140 [.254]	.767** [.260]	.780** [.261]	.534* [.265]
	Stratum 4	.186 [.274]	-.354 [.264]	.534† [.280]	.549* [.281]	.222 [.294]
	Stratum 5	.649† [.336]	-.149 [.318]	.687* [.327]	.700* [.329]	.391 [.367]
	Stratum 6	.600 [.412]	.021 [.395]	.660† [.398]	.677† [.399]	.523 [.575]
	Stratum 7	.336 [.355]	-.396 [.330]	.515 [.343]	.531 [.342]	.343 [.588]
	Stratum 8	.376 [.575]	-.395 [.589]	.657 [.580]	.670 [.580]	.367 [.781]
	Stratum 9	2.17*** [.498]	.1.01* [.473]	2.09*** [.486]	2.10*** [.486]	1.86* [.746]
	Stratum 10	1.19* [.556]	-.037 [.518]	1.13* [.526]	1.13* [.525]	.557 [.772]
Racial Group:	White	—	—	—	—	—
	Black	—	—	—	—	.033 [.194]
	Other	—	—	—	—	.671† [.398]
Age Group:	18 to 20	—	—	—	—	—
	21 to 22	—	—	—	—	-.247 [.181]
	23 and up	—	—	—	—	-.504* [.209]
Education:	Less than HS	—	—	—	—	.944*** [.291]
	High School Grad	—	—	—	—	.544** [.193]
	Some College (modal cat.)	—	—	—	—	—
	College Grad	—	—	—	—	.082 [.232]
Wave 1 Violence		—	—	—	—	-.241 [.461]
Wave 1 Suicide Ideation		—	—	—	—	1.23*** [.276]
Wave 1 College Aspiration		—	—	—	—	-.103 [.091]
	Constant	3.05	4.13	2.95	2.92	3.49
	R^2	0.08	0.12	0.15	0.15	0.17

of a point change in predicted CES-D score at Wave 3 ($p < 0.001$). However, the estimated effect of Wave 2 violence on Wave 3 CES-D score remains virtually unchanged, at .664 of a point ($p < 0.01$). This finding is both substantively meaningful and statistically significant: even after controlling for preexisting depressive symptomology, exposure to violence still engenders a negative effect on later depression status. Once again, tests of stratum-specific effects showed no significant differences.

Model 3 additionally controls for gender, again with unsurprising results. Consistent with expectations, females show an expected average CES-D score approximately 1.67 points higher than males ($p < 0.001$), which is in line with existing literature on depression and gender (Hankin et al 1998, Nolen-Hoeksema 2001, Piccinelli & 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 around .238 of a point on the CES-D scale at Wave 3 per point at Wave 1 ($p < 0.001$), very close to the estimate from the previous model. Crucially, the effect of Wave 2 violence exposure on Wave 3 CES-D score shrinks only slightly to .587 of a point and remains significant ($p < 0.05$). 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 × female interaction term with surprising results. In this model, the regression coefficients for gender and previous depression score remain virtually unchanged, at 1.71 and .238 respectively (both $p < 0.001$). The estimate for the effect of Wave 2 violence exposure on Wave 3 CES-D score increases slightly to .655 of a point ($p < 0.05$), which represents .136 of a standard deviation – still a substantively meaningful effect. However, the

coefficient for the violence × female term – which can be interpreted as the difference between males and females in how much Wave 2 violence is expected to affect their Wave 3 CES-D score net of propensity score stratum and other variables – is not statistically significant, though at about 1/3 the size of the overall treatment effect it is a substantial difference. Also interesting is that the coefficient is negative, which (contrary to Hypothesis 2) indicates that if anything the effect of violence exposure is smaller for women than for men. With an estimated value of -.201 ($p > 0.1$), this result suggests that the effect of violence exposure on depression may not vary by gender – that is, male and female students exposed to violence manifest similarly poor mental health responses years after the fact – but that if there is variation, it is men who fare worse.

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 two-thirds of a point higher on average. As expected, depression seems to decline with age, as 21-22 year-olds have a 0.247-point lower average CES-D score than 18-21 year-olds, and 23-25 year-olds have 0.504-point lower average CES-D score than 18-21 year-olds ($p < 0.05$). 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 0.944 ($p < 0.001$) and 0.544 ($p < 0.01$) – 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 degree are indistinguishable statistically from those who graduated from college. This may be due to the fact that the subjects are still young: many who report “some college” may still be in the process of completing their degree, 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 shows a strong association with depression ($b = 1.23$, $p < 0.001$), and Wave 1 college aspirations shows a weak link ($b = .103$, $p > 0.1$), both in the predicted direction. 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 (more likely) that most of the people who were exposed at Wave 2 experienced a chronic pattern of exposure that was already 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 covariate adjustment: Wave 2 violence exposure still seems to affect Wave 3 CES-D score by about 0.650 of a point ($p < 0.05$); Wave 1 CES-D score is still strongly associated with Wave 3 CES-D score ($b = 0.208$, $p < 0.001$); females still have CES-D scores an average of 1.57 points higher than males ($p < 0.001$); and the violence × female term ($b = -.227$, $p > 0.1$) remains substantively about 1/3 of the overall effect size but statistically insignificant, suggesting that men may fare worse even though the difference between violence's effect on males and females is not statistically distinguishable.

In light of these findings, it is instructive to compare straightforward differences in depression score within each stratum for both males and females. These comparisons, weighted for probability of retention, are presented in Table 2.6.

A number of clarifications come to light in this comparison. First of all, both for the full sample and for the sex groups separately, most of the stratum-specific effects point in the hypothesized direction (with those who were exposed at Wave 2 faring worse than those who were not), and all of the significant effects (at $p < 0.05$) are in that direction – though because of

TABLE 2.6: Propensity Score Stratum-Specific Treatment Effects [95% CIs] for Full Sample, Males Alone, and Females Alone

	Full sample			Males			Females		
Strat	Z = 0	Z = 1	Diff (t, p)	Z = 0	Z = 1	Diff (t, p)	Z = 0	Z = 1	Diff (t, p)
1	4.822 [4.54-5.11]	6.108 [4.45-7.77]	1.286 (1.50, 0.135)	3.592 [3.09-4.09]	2.964 [0.60-5.33]	-0.628 (-0.51, 0.612)	5.186 (4.85-5.52)	7.265 (5.70-8.83)	**2.079 (2.56, 0.011)**
2	5.278 [4.91-5.65]	4.913 [3.20-6.63]	-0.365 (-0.41, 0.683)	4.013 [3.58-4.45]	4.405 [1.38-7.43]	0.392 (0.25, 0.801)	6.291 (5.76-6.83)	5.39 (3.65-7.13)	-0.902 (-0.97, 0.330)
3	5.317 [4.87-5.76]	6.344 [4.50-8.19]	1.027 (1.06, 0.290)	4.248 [3.77-4.73]	5.833 [3.57-8.10]	1.585 (1.34, 0.182)	6.729 (5.99-7.47)	7.088 (4.03-10.1)	0.359 (0.22, 0.823)
4	5.149 [4.65-5.65]	5.165 [4.14-6.19]	0.016 (0.03, 0.979)	4.355 [3.86-4.85]	4.675 [3.62-5.73]	0.319 (0.54, 0.591)	7.026 (5.93-8.12)	6.653 (4.19-9.12)	-0.374 (-0.27, 0.787)
5	5.543 [4.88-6.21]	6.046 [4.85-7.24]	0.503 (0.72, 0.474)	4.698 [4.00-5.39]	5.643 [4.06-7.22]	0.946 (1.07, 0.286)	7.041 (5.76-8.32)	7.002 (5.52-8.49)	-0.039 (-0.04, 0.969)
6	5.277 [4.35-6.21]	6.391 [5.21-7.58]	1.114 (1.44, 0.151)	4.843 [3.54-6.15]	5.100 [3.63-6.57]	0.257 (0.25, 0.800)	5.858 (4.56-7.16)	7.830 (6.11-9.55)	*1.972 (1.78, 0.078)*
7	5.546 [4.71-6.38]	5.507 [4.70-6.32]	-0.039 (-0.07, 0.948)	4.209 [3.46-4.96]	4.666 [3.82-5.52]	0.457 (0.79, 0.432)	9.153 (7.34-11.0)	7.282 (5.62-8.94)	-1.871 (-1.48, 0.142)
8	4.894 [3.64-6.15]	6.303 [4.46-8.14]	1.409 (1.23, 0.225)	4.327 [3.19-5.46]	5.318 [3.28-7.35]	0.991 (0.82, 0.416)	7.220 (3.24-11.2)	9.508 (6.23-12.8)	2.288 (0.83, 0.420)
9	5.943 [4.59-7.30]	8.286 [7.15-9.42]	**2.344 (2.58, 0.011)**	5.797 [4.25-7.35]	7.444 [6.08-8.81]	1.646 (1.55, 0.124)	6.851 (5.35-8.35)	10.338 (8.39-12.2)	**3.487 (2.72, 0.010)**
10	5.907 [4.08-7.73]	6.761 [5.61-7.92]	0.854 (0.77, 0.447)	5.183 [3.47-6.90]	6.459 [5.20-7.72]	1.275 (1.16, 0.252)	11.1 (8.65-13.5)	7.952 (5.38-10.5)	-3.140 (-1.64, 0.122)
ATE			**0.701 (2.70, 0.007)**			**0.793 (2.57, 0.010)**			0.346 (0.80, 0.423)

t-test of difference between Male and Female ATEs: diff = -0.447, t = -0.843, p = 0.399

the small sample sizes and high variances within each stratum, few of these effects achieve statistical significance. Interestingly, the effects for women are much less consistent in this regard than are the findings for men; the men's results have only one stratum-specific effect pointing in the "wrong" direction, whereas the women's results have four such unexpected negative effects. Perhaps because of this, the average treatment effect (ATE) across strata for

women is not statistically distinguishable from 0 (b = 0.346, t = 0.80, p = 0.423). The ATE for men (b = 0.793, t = 2.57, p = 0.010) is both substantial and significant, but a direct test for difference between the effect sizes reveals that the ATEs for men and women are not statistically distinguishable (diff = -0.447, t = -0.843, p = 0.399). This is in agreement with the interaction-effect tests in the models above – the effect sizes for men and women are statistically identical, but substantively it appears that violence's effect is worse for men. We may consider these results as adding further weight to the evidence against Hypothesis 2, that the effect of violence exposure will be higher for women than it is for men: in fact, the two sex groups may not differ much in their response to violence, and when considered separately it is only among the *men* that Wave 2 violence exposure produces a distinct effect on Wave 3 depression score.

DISCUSSION AND CONCLUSION

Summarizing these findings with respect to the chapter'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 generally uphold this prediction. Across all model specifications in Table 2.5, we note a distinct harmful effect of violence on depression score – between .701 and .587 of a point on the CES-D scale, representing between .146 and .122 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. The results from Table 2.6 complicate this somewhat, but still show that there is an overall effect of violence on depression.

Also upheld is the prediction, based on extensive previous findings, that women in general will 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 scale 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. And the results in Table 2.6 show that within each stratum and treatment assignment condition, women's average CES-D scores are higher than men's.

Last, and possibly most interesting, are the results regarding the second hypothesis. Surprisingly, the violence × female interaction effect never approaches statistical significance in any of the estimated propensity score models, though the size and direction of the estimate suggest the possibility that violence's effect on men's depression score is worse than on women's. The results in Table 2.6 also show that the cross-stratum ATEs of violence exposure on depression are statistically identical for females and males, and further reveal that when the two groups are considered separately it is the men who show a higher average effect of violence exposure on depression. This implies that violence has a negative causal effect on depression score and females have a higher level of depression overall, but the effect of violence exposure is either equally bad for both sexes or slightly worse for men, which would be reasonable if it is the case (as much research has suggested) that men's physical violence victimization is typically more severe than women's (Popp & Peguero 2011, Robers et al 2012). This finding is broadly consistent with Nishina et al (2005) and some others (Biggs et al 2010, Lehman & Repetti 2007, Nishina & Juvonen 2005) who find that the pathways between peer victimization and negative mental health outcomes are the same for boys and girls; it is certainly sufficient to reject the hypothesis that women's exposure to violence is more harmful than men's.

These findings, while 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, and that the size of the effect is similar for both. This suggests that many earlier findings of sex group differences may be detecting non-causal associations, due to the use of cross-sectional (e.g. DuRant et al 2000, Lopez & DuBois 2005) or prospective cohort data designs (e.g. Bond et al 2001, Copeland et al 2013) and an attendant methodological inability to adequately address endogenous selection for violence exposure based on 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 findings in this context may speak to the nature of the sex differences observed in previous studies. The results obtained here suggest that minimal differences exist in the size of any causal effect defined in counterfactual terms.

It is necessary to stress the limitations of a study like 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 part of a long-term pattern of chronic or repeated exposure that begins at or before Wave 1 and lasts over many years. This is underscored by the relative lack of overlap across propensity score strata between those who were exposed to violence at Wave 1 and those who were not: strata 1 through 3 contain only people who experienced no violence at Wave 1, stratum 4 contains only 10 people who were exposed at Wave 1, strata 7 through 9 each contain 15 or fewer people who were unexposed at Wave 1, and stratum 10 contains only people who were exposed at Wave 1. It seems that the risk patterns for these two groups are, and should be treated as, qualitatively different. This also gives us reason to suspect that these different forms of exposure will have different sequelae for young people's mental health. Researchers ought to

separate out the effects of chronic and isolated exposure to violence, a task which I will undertake directly in the next chapter.

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, since we detected no consistent patterns of stratum-specific effects, it is possible that violence's effects on health may be similar across the spectrum of propensity for exposure. If anything, since 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 findings regarding gender difference. While it is possible that men and women react similarly to the kinds of violence measured here, it is still plausible that these kinds of violence are more specific to the male experience (explaining the higher overall ATE for men and the inconsistent patterns in women's stratum-specific ATEs), and that other forms of victimization – such as intimate partner violence, sexual assault, and other forms of interpersonal aggression – would differentially impact females.

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 at least one measure of emotional well-being 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 similarly detrimental for men and women – and if we take seriously findings like 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 post-traumatic 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.

CHAPTER THREE

Effects of Isolated vs. Repeated Violence Exposure on Mental Health and Academic Attainment

Links between traumatic violence exposure and academic and mental health outcomes are of serious concern to educational research. Both education and violence exposure are considered critical factors in life course development, especially with regard to health. Education is known to be a powerful predictor of later mental and physical health outcomes (Mirowsky & Ross 2003) and is considered a "fundamental cause" of good health throughout the life course (Link & Phelan 1995; Phelan, Link, & Tehranifar 2010). Violence exposure, by contrast, is strongly associated with poorer health along virtually all relevant dimensions – including mental health concerns like suicidality (Klomek et al 2010), emotional maladjustment (Arseneault et al 2006), and PTSD-like symptoms (Osofsky 1995), as well as somatic health complaints like headache, stomachache, backache, morning fatigue, sleep disturbance, bed wetting, dizziness, irritability, and poor appetite (Due et al 2005, Fekkes et al 2006, Ghandour et al 2004, Williams et al 1996). Indeed, in a recent technical report published in the journal *Pediatrics*, Shonkoff and colleagues (2012) cite early childhood violence exposure as a cause of the kind of toxic stress that can lead to lifelong health problems. Likewise, violence is associated with poorer cognitive functioning (Sharkey 2010) and lower academic success (Burdick-Will 2013, Tanaka et al 2014), suggesting that violence exposure may also indirectly affect health and related behaviors by reducing educational investment and persistence. But the specific role played by school violence exposure in altering developmental trajectories and changing health-related behaviors is not as well examined.

Results from the previous chapter strongly indicated the existence of at least two distinct patterns of violence exposure – early and frequent exposure on the one hand, and intermittent or isolated exposure on the other – and also suggested that different exposure patterns will have different socio-emotional health-related sequelae. This chapter will address the effects of school violence exposure on individual mental health and academic outcomes, and will particularly investigate whether these effects are contingent on individuals' previous history of violence.

Besides addressing an interesting theoretical question raised in the previous chapter, such an investigation could yield important practical results by suggesting likely benefits of different antiviolence policy emphases. Given the current constrained state of resources available for school violence prevention programs, policymakers and educators must prioritize interventions that will do the most good. In particular, results here may help us determine whether students' lives will be better served by focusing on the intensive margin or on the extensive margin of violence exposure – that is, whether we could more effectively mitigate harm by targeting those who suffer repeat victimization or by minimizing the share of youth who are exposed to violence at all. Results showing that repeat exposures exert cumulative harm suggests the former strategy; results showing diminishing effects of later exposures would suggest the latter.

In this chapter I will demonstrate a heterogeneous causal link between school violence exposure and later negative mental health and academic outcomes as a function of individuals' previous history of violence exposure, showing that those who experience isolated victimization manifest different outcome patterns than those who experience repeated victimization.

BACKGROUND

This analysis is informed by the theoretical perspectives on the life course, cumulative inequality, and the contingency of stress reactions. Life course theory (e.g. Elder & Shanahan

2007) predicts that a single event at a single moment in time – here, violence exposure – has different effects depending on individuals' histories, and that we must understand what individuals have already been through when interpreting how they react to a stimulus. Literature applying this perspective to violence and the school setting is usually concerned with academic outcomes (Lauen & Gaddis 2013; Sampson, Sharkey, & Raudenbush 2008; Wodtke, Harding, & Elwert 2011) and typically finds negative effects of violence on educational persistence and attainment, but portions of this literature also address health-related consequences of adverse exposures (e.g. Crane 1991, Harding 2003); I take up both these concerns in this analysis.

Cumulative inequality theory (e.g. Ferraro & Shippee 2009) predicts that exposure to negative events or contexts exerts a twofold harmful effect: those who suffer early adverse experiences develop poorer outcomes (Dannefer 1987), and also manifest different, typically less adaptive responses to other stressors later in life (O'Rand 2003). This underscores the prediction that the effects of violent victimization at a point in time may vary as a function of the victim's previous history of exposure: those who suffer repeat victimization may have both a worse starting point and a worse response to later events. This is also consistent with Shonkoff et al's (2012) argument that, because of the developing brain's unique susceptibility to the effects of toxic stress, early childhood adversity has a more harmful effect on adult mental health than do exposures that occur later in life.

Theories about the contingency of stress reaction, in particular Aneshensel's (Aneshensel, Rutter, & Lachenbruch 1991; Aneshensel 1992), argue that the impact of stressors on individual health depend on the type of stressor and characteristics of the individual; because members of different social groups differ in how they experience stress and manifest reactions, the same stressor – here, school violence – may produce different outcomes for different types of people.

To the extent that previous exposure is considered a characteristic of the individual, this too suggests that we must account for individuals' previous victimization experiences in predicting their responses to any particular exposure.

Also pertinent to this topic is the extensive literature on re-victimization: the well-established empirical pattern that those who experience victimization once are at higher risk of experiencing subsequent victimization (Finkelhor, Ormrod, & Turner 2007; Weisel 2005). Two branches of this literature are relevant to my purpose here. One strand of this literature analyzes empirical determinants for the occurrence of two or more victimizations within close temporal proximity, such as multiple violence exposures within a few years of one another (Lauritsen & Quinet 1995; Outlaw, Ruback, & Britt 2002). Because my methodological strategy will leverage violent events occurring approximately a year apart, understanding the causes of such situations is important. A second, more conceptual component of the literature advances the argument that for many children victimization should be viewed as a condition rather than an event (Finkelhor et al 2007). Much victimization among students is ongoing; studies of peer relationships suggest that some vulnerable students are repeatedly victimized by many different types of attacks from many different types of aggressors (Kochenderfer-Ladd 2003). If vulnerability may be an enduring state, our attention is compelled towards differences between those whose victimization is part of such a pattern and those whose victimization represents an isolated incident.

The key insight I draw from these theories is that in order to reach a more sophisticated understanding of its effects, violence exposure must be considered in the context of students' lives: differences in background characteristics may lead to differences in the patterns of outcomes manifested following a particular instance of violence exposure.

The particular outcomes on which I focus include academic success, captured by high school graduation and college attendance, and mental health outcomes such as depression and suicidality. Research indicates that these are domains in which the effects of violence exposure, and chronic violence exposure in particular, may be especially pernicious. Among many others, Bond and colleagues (2001) use longitudinal data to show that past victimization predicts anxiety and depression in late adolescence but preexisting emotional problems do not predict later victimization; Boynton-Jarrett and colleagues (2008) demonstrate a longitudinal association between violence exposure and poor health which gets worse in proportion to both the number of different types of exposures reported and the length of time over which exposures occur; Copeland and colleagues (2013) find that victims of violent bullying face elevated risk of psychiatric problems such as depression, anxiety, and suicidal ideation or attempt; and Lehman and Repetti (2007) find that school victimization produces changes in students' mood and self-esteem which negatively color later perceptions of interactions with parents and others. Indeed, the theory of hostile attribution bias (Dodge 1980; de Castro et al 2002) holds that children who are victimized are more likely to misinterpret others' later behaviors as having aggressive intention, especially under conditions of threat (Dodge & Somberg 1987); this has serious implications for children's ability to form friendships and other meaningful social ties (Dodge 1980), and the resultant isolation or exclusion can negatively affect social/emotional health.

As pertains to academic attainment, Tanaka et al (2014) find that severe physical victimization in childhood is associated with fewer years of attained education even after controlling for a variety of background characteristics, but sexual victimization and non-severe physical victimization have no such effects net of those attributes. Burdick-Will (2013) shows that higher rates of violent incidents in schools lead to lower student test scores, which she

suggests is due to direct effects on learning via mechanisms like classroom disruption and cognitive stress. And Wodtke, Harding, and Elwert (2011) find that sustained exposure to disadvantage in the neighborhood context exerts a tremendous negative effect on the likelihood of high school graduation.

Based on this, I expect that adolescents' responses to school violence exposure will vary depending on whether their exposure is isolated or part of a pattern of multiple exposures. Specifically, I make three predictions about the effects of exposure to violence at a point in time as a function of previous history of exposure: 1. Compared to those who experience an isolated incident of violence only later in life, those who experience violence earlier will show lower rates of college enrollment and higher rates of depression and suicidal ideation; 2. Among those who do not report previous violence exposure, a single instance of exposure to school violence will cause a sharp increase in negative outcomes (i.e. lower college enrollment rates and higher rates of depression and suicidality); and 3. Those who do report previous violence exposure will to be less susceptible to negative effects from a single instance of exposure to school violence – that is, an additional exposure will not significantly alter their college enrollment or mental health patterns. I test these hypotheses using data from the National Longitudinal Study of Adolescent Health ("Add Health"); to preview the findings, I obtain support for Hypotheses 1 and 3 and partial support for Hypothesis 2.

METHOD

Four major methodological obstacles crop up in the attempt to analyze the effects of isolated and repeated violence exposure on academic and mental health outcomes later in life: 1. accounting for the non-random assignment of violence exposure to individuals; 2. accounting for the non-independence of individuals' propensity for violence exposure within schools; 3.

determining the correct temporal ordering for pre-exposure traits, exposure itself, and outcomes; and 4. drawing the distinction between those whose exposure is an isolated shock, and those whose exposure is part of a pattern of repeat victimization.

Non-random treatment assignment is among the biggest problems for causal inference in social science (Schneider et al 2007). This is especially the case for a treatment, like violence exposure, where the characteristics on which subjects are "selected" for treatment may also independently dispose them to adverse outcomes. Violence is not distributed randomly; factors such as obesity (Swearer, Espelage, Vaillancourt, & Hymel 2010), physical development (Olweus 1993), drug use (Saner & Ellickson 1996), gender non-conforming behavior (Pascoe 2011), neighborhood disadvantage (Harding 2003, 2009; Sampson, Sharkey, & Raudenbush 2008), and more all affect adolescents' chances of exposure, and may also directly affect mental health and academic functioning via other mechanisms (Burdick-Will 2013, Sharkey 2010).

The gold standard for proving causal effects is the controlled randomized trial (CRT), which uses random treatment allocation to ensure that all links between pre-treatment characteristics and treatment assignment status are broken. However, even if it were practically possible, no one would suggest randomly assigning adolescents to violence exposure. In place of a CRT, I estimate causal effects using propensity score stratification (Rosenbaum & Rubin 1983, Rosenbaum 1984). Assuming that violence exposure results from both structural and random factors, every individual can be assumed to have an underlying likelihood of being exposed to violence – a "propensity score," π, which theoretically ranges from zero (no chance of violence exposure) to one (absolute certainty of exposure). Propensity scores can be estimated ($\hat{\pi}$s) for each individual using binary logistic regression, with violence exposure as the outcome and relevant observable characteristics as predictors. Once the $\hat{\pi}$s are known, individuals can be

divided into strata where each stratum contains individuals with similar values of $\hat{\pi}$ and other pretreatment characteristics, but some of whom did and some of whom did not actually receive the treatment. Although the models are directly based only on subjects' observable traits, Rosenbaum (1984) shows that under these conditions, and given proper model specification, conditioning on propensity score is sufficient to control for both observed and unobserved characteristics. Thus, because each stratum contains similar people with similar chances of receiving the treatment, but some of whom were treated and some of whom were not based on factors having nothing to do with their structural characteristics, each stratum functions as a miniature pseudo-CRT; the mean difference in outcomes between treated and untreated individuals within each stratum gives an unbiased estimate of the average treatment effect.

Non-independence of treatment assignment is another major problem. Students are nested within social settings – such as schools and neighborhoods – and characteristics of these settings may predict violence exposure independently of individual characteristics. For example, two students with similar values on all individual-level variables may have widely different chances of exposure to violence if one of them attends a high-violence school while the other attends a school with low violence rates. The exposure to common social settings shared by students in the same schools leads to interdependence of error terms, which violates a central assumption of the standard GLM regression paradigm (Burstein 1980, Raudenbush & Bryk 1986).

To account for this, I estimate propensity scores using a hierarchical model (Raudenbush & Bryk 2002) with students at level 1 nested within schools at level 2. Because the treatment is a binary indicator of violence exposure, I specify a hierarchical binary logistic regression model – a type of the hierarchical generalized linear model (HGLM). This allows me to calculate $\hat{\pi}$s as a function of both individual- and school-level characteristics.

Temporal ordering of events presents a third major problem for any causal investigation, especially those employing cross-sectional data. Cross-sectional studies, by their nature, cannot confirm whether the ontological ordering of events actually aligns with our conceptual ordering. Did the covariates (school- or individual-level) actually antecede the treatment? Or could they have been influenced by the treatment? Or did experiencing the treatment lead some subjects to report their pretreatment covariates differently? Either of the latter two possibilities presents a serious problem for the attempt to control for pretreatment characteristics; if, say, a student reevaluated his/her self-perception or switched schools as a function of his/her experience with violence, then what we believe to be pre-exposure states may actually be effects of exposure. Similarly, we have to ask whether the treatment actually anteceded the outcomes, or if the putative outcomes really cause the treatment itself. If a student who is already depressed experiences violence, it would be a mistake to point to violence exposure as the etiological source of the depression. Unfortunately, cross-sectional data – even with retrospective questions – offers no way around this obstacle.

To avoid these problems I employ three waves of longitudinal data in a cross-lagged structure. I use covariates from wave 1 to predict violence exposure at wave 2, then use the propensity scores derived from this model to predict outcomes at wave 3. This removes the problem of reverse causality; it is impossible for a student's violence exposure at time 2 to be the cause of answers given to questions at time 1. The most appropriate data source for this sort of investigation is the National Longitudinal Study of Adolescent Health, or "Add Health," described in more detail below.

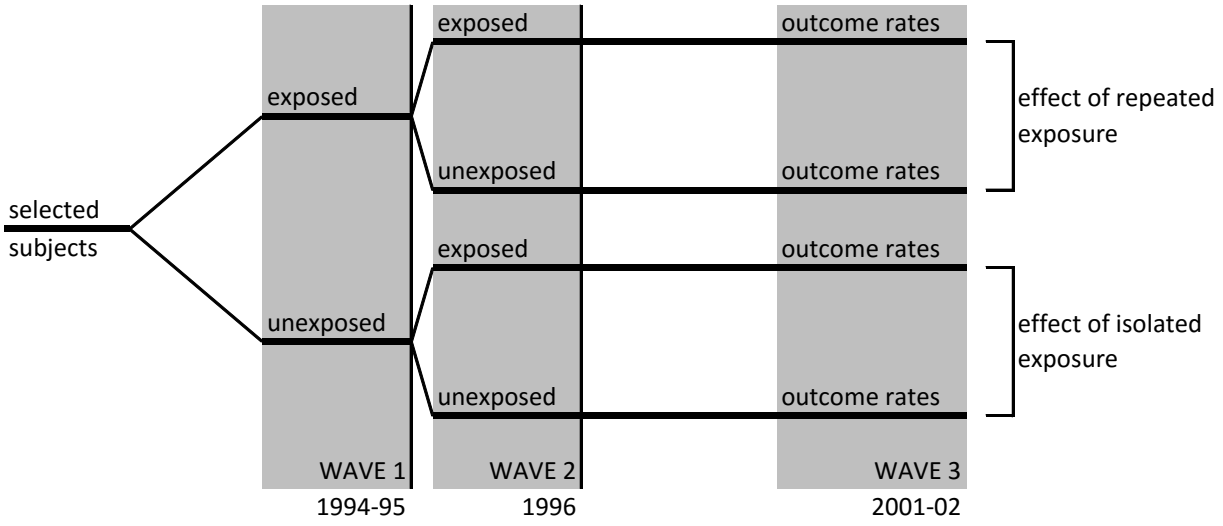
Last, I disentangle the effects of acute and chronic violence exposure. The treatment here is violence exposure at wave 2, but some subjects also experienced violence at wave 1 (i.e. their

wave 2 exposure is part of an ongoing pattern of violence) while some did not (i.e. their wave 2 exposure is relatively isolated). In short, among those who were unexposed at wave 1, some suffer an isolated exposure at wave 2 and some do not; among those who were exposed at wave 1, some suffer a repeat exposure at wave 2 and some do not.

To account for this, and for the fact that the groups likely have other systematic differences and thus different relevant pretreatment predictors, I separate the sample into two subsamples: those who did, and those who did not, experience wave 1 violence. I can then estimate full propensity score models separately for each group, allowing them to have unique patterns of relevant pretreatment covariates as well as group-specific effects of academic and mental health outcomes.

In summary: I use an estimation strategy based on propensity score stratification with cross-lagged predictors, where individual- and school-level covariates from Add Health Wave 1 are entered in a 2-level HGLM to predict propensity for violence exposure at Wave 2, and the resulting propensity scores are used to sort students into strata and predict the effect of exposure on outcomes at Wave 3. I take the additional step of splitting the sample into subgroups defined by the presence or absence of any reported school violence exposure (defined as witnessing or direct victimization) at Wave 1. Because Add Health asks about violence over the last 12 months, and Wave 2 was collected approximately 1 year after Wave 1, violence exposure at Wave 2 among those who were not exposed at Wave 1 should represent an isolated shock; by contrast, Wave 2 violence exposure for those who were also exposed at Wave 1 represents a repeated pattern of at least two serious exposures over two years. Figure 3.1 represents this design graphically.

FIGURE 3.1: Conceptual Design of Propensity-Stratified Longitudinal Study



DATA

This study uses data from the Public Use subsample of the National Longitudinal Study of Adolescent Health (Add Health), one of the largest and most comprehensive studies of adolescents ever undertaken. Add Health has followed a nationally representative sample of adolescents in the United States over four waves, beginning during the 1994-95 school year with respondents in grades 7-12, and continuing in 1996, 2001-02, and 2007-08. Eighty high schools and 52 middle schools from the US were chosen with systematic sampling methods and implicit stratification incorporated to ensure that the sample is representative of US schools with respect to school size, school type, region of country, urbanicity, and ethnicity. In-school and in-home surveys were given to the study subjects, and surveys were also given to their parents, siblings, fellow students, school administrators, and romantic partners. These data were matched with information about neighborhoods and communities in extant databases (Harris et al 2009).

Here, as in the previous chapter, I draw on the first three waves of Add Health data, using individual- and school-level covariates from Wave 1 to predict propensity for school violence exposure at Wave 2, then propensity scores from Wave 2 to predict outcomes at Wave 3. I also

reduce the sample by retaining only students who report that they are enrolled in school as of Wave 2. I further drop any observations who do not provide data at all three waves or whose violence exposure status at Wave 2 cannot be determined. This leaves me with two subsamples including 892 students who were exposed to violence at Wave 1 (of whom 436 are exposed again at Wave 2) and 2874 students who were not exposed at Wave 1 (of whom 254 are later exposed at Wave 2). To account for biases caused by differential probability of censoring, I calculate weights based on inverse probability of retention in the later waves and apply them to the estimates reported below.

Key Variables

The key treatment variable here, as in the previous chapter, is exposure to violence at Wave 2. As before, I focus on the witnessing and victimization types of exposure. Respondents were counted as "exposed" if they answered anything other than "never" to any of the following questions at Wave 2: "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."

This analysis requires assessing exposure status at Wave 1 as well as at Wave 2. The questions used to measure exposure at Wave 1 are similar to those used at Wave 2; at Wave 1, respondents were asked "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 got into a physical fight; You were jumped." As above, students were counted as exposed – in this case, "previously exposed" – if they answered anything other than "never" to any of these questions.

As before, some caveats attend on the use of this definition of violence exposure for the analysis. For one, this operationalization does not distinguish between varying levels of severity of violence exposure within a data collection wave – a student who reports "got into a physical fight" once in the last 12 months receives the same score as one who answered "more than once" to several of the questions. However, because this analysis is primarily concerned with exposure status over time rather than severity of exposure, this does not pose a major substantive threat. Indeed, the decision to track violence exposure status over multiple waves of data seems at least partially to obviate one concern raised in the previous chapter: the operationalization of violence exposure as a single traumatic incident rather than as a persistent stressor or a chronic condition.

I analyze the potentially heterogeneous effect of violence on three key outcomes measured at Wave 3: college attendance, depression score, and self-reported suicidal ideation. College attendance is coded as a binary outcome based on subjects' responses to the question "What is the highest grade or year of regular school you have completed?" Those who indicate any college attendance were coded as 1; others were coded as 0. Add Health also asks directly about suicidal ideation with the question "During the past 12 months, have you ever seriously thought about committing suicide?" This outcome was coded as 1 if respondents indicated "yes" and 0 if they indicated "no." Depression was measured using a modified version of the Center for Epidemiologic Studies Depression (CES-D) scale built into the Add Health questionnaire. The standard CES-D instrument consists of 20 self-report items, forming a scale that measures symptoms of several aspects of depression, including negative affect, positive affect, somatic complaints, and interpersonal relations (Radloff 1977). The Add Health survey instrument contains a simplified 10-question battery to assess the different categories of the CES-D scale; these items 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.

Propensity Score Models

I identified approximately 125 pre-treatment covariates in the Wave 1 data which I tested for inclusion in the propensity score estimation model. These included several categories of pre-exposure characteristics: demographic traits, temperament and behaviors, home environment, school environment, mental health, physical health, peer influences, social support, and academic characteristics. I also aggregated student data to the school level in order to create 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.

After separating the sample into the two subgroups based on Wave 1 exposure and restricting the subsamples based on the criteria outlined above, I estimated propensity scores $\hat{\pi}$ for each group with separate maximum-likelihood hierarchical logistic regression models. As noted, in order to maximize within-stratum balance on both observable and unobservable traits it is advisable to use a wide variety of predictor variables. I therefore selected variables for the propensity score models using iterative stepwise regression. At each iteration, the procedure either added to the model the variable most strongly associated with Wave 2 violence exposure conditional on the covariates in the previous step, if the resulting coefficient was significant at the $\alpha = 0.15$ level; or removed from the model any variables no longer associated with Wave 2 violence exposure at the $\alpha = 0.15$ level. I ran this procedure separately for each subsample, using Stata for the level-1 variables, then employing the resulting set of variables in 2-level models estimated with the HLM software, testing level-2 variables for significant association with Wave

2 violence exposure net of the other covariates and retaining those that added to the model's explanatory power. In the final models I reintroduced measurements for conditions which were not significant at $\alpha = 0.15$ in the stepwise model but which extant research identifies as relevant for violence exposure, such as age, home condition, and temperament.

This resulted in two final models, one for each violence exposure subsample. The model for those who were not exposed to violence at Wave 1 estimated each subject's $\hat{\pi}$ using 35 level-1 covariates and one level-2 covariate. The model for those who were exposed to violence at Wave 1 estimated each subject's $\hat{\pi}$ using 33 level-1 covariates and one level-2 covariate. Restricting each subsample to its region of common support on $\hat{\pi}$ subsequent to estimating the propensity score models further reduces the size of the two subsamples, leaving 2674 subjects who were unexposed at Wave 1 (of whom 219 will go on to be exposed at Wave 2) and 798 subjects who were exposed at Wave 1 (of whom 377 are exposed again at Wave 2).

With estimated scores in hand, the next step of propensity score modelling is to divide the samples into strata such that the treated (i.e. exposed to violence at Wave 2) and untreated groups in each stratum are statistically indistinguishable on $\hat{\pi}$ and all other pretreatment covariates. The strata are summarized in Table 3.1. The Wave 1 unexposed subsample was divided into five

TABLE 3.1: Frequency Counts per Propensity Score Stratum

	Panel 1: Unexposed at Wave 1				**Panel 2: Exposed at Wave 1**			
Strat	**P-score (range)**	**Untreated (N)**	**Treated (N)**	**Total (N)**	**P-score (range)**	**Untreated (N)**	**Treated (N)**	**Total (N)**
1	[0.0-0.03)	907	14	921	[0.0-0.3)	108	31	139
2	[0.03-0.055)	585	18	603	[0.3-0.355)	74	43	117
3	[0.055-0.1)	492	46	538	[0.355-0.425)	74	51	125
4	[0.1-0.25)	409	78	487	[0.425-0.47)	40	49	89
5	[0.25-1.0)	62	63	125	[0.47-0.575)	65	73	138
6	–	–	–	–	[0.575-0.695)	43	86	129
7	–	–	–	–	[0.695-1.0)	17	44	61
total	–	2,455	219	2,674	–	421	377	798

different propensity score strata, balanced on $\hat{\pi}$ and on all other covariates; the Wave 1 exposed subsample was divided into seven balanced strata. In both cases, cut points were determined empirically to optimize balance on propensity scores and all other pretreatment covariates.

Table 3.2 shows the average estimated propensity scores within each of these strata for both subsamples, with tests of significance calculated for the differences between the treated and untreated in each, to demonstrate that the balancing property has been satisfied.

TABLE 3.2: Mean [SE] of Propensity Scores within Strata, by Treatment Assignment, by Wave 1 Violence Exposure Status

	Unexposed at Wave 1				Exposed at Wave 1			
Strat	Range	Z = 0	Z = 1	Diff (t, p)	Range	Z = 0	Z = 1	Diff (t, p)
1	[0.0-0.03)	0.018 [.0002]	0.021 [0.002]	0.003 (1.44, 0.149)	[0.0-0.3)	0.247 [0.004]	0.257 [0.006]	0.011 (1.40, 0.164)
2	[0.03-0.055)	0.041 [.0003]	0.041 [0.002]	0.00005 (0.03, 0.980)	[0.3-0.355)	0.332 [0.002]	0.331 [0.003]	-0.0004 (-0.14, 0.886)
3	[0.055-0.1)	0.074 [.001]	0.077 [0.002]	0.003 (1.23, 0.221)	[0.355-0.425)	0.391 [0.002]	0.399 [0.003]	0.007 (1.73, 0.085)
4	[0.1-0.25)	0.155 [.002]	0.161 [0.005]	0.006 (1.16, 0.248)	[0.425-0.47)	0.447 [0.002]	0.444 [0.002]	-0.002 (-0.89, 0.376)
5	[0.25-1.0)	0.384 [.013]	0.414 [0.015]	0.030 (1.54, 0.127)	[0.47-0.575)	0.514 [0.004]	0.527 [0.004]	0.012 (1.73, 0.086)
6	–	–	–	–	[0.575-0.695)	0.628 [0.006]	0.630 [0.004]	0.002 (0.21, 0.832)
7	–	–	–	–	[0.695-1.0)	0.752 [0.010]	0.772 [0.006]	0.020 (1.61, 0.112)

RESULTS

Descriptive Findings

Tables 3.3a and 3.3b offer a descriptive summary of the distribution of key variables within propensity score strata across the two subsamples. Table 3.3a, which describes the subsample that was unexposed to violence at Wave 1, reveals interesting trends across propensity score strata. Since the propensity score is itself an estimate of the likelihood of violence exposure at Wave 2, it is not surprising that the proportion of subjects actually exposed to Wave 2 violence

increases across strata. Some similar trends are evident among the three outcome variables: there is a strong, monotonically decreasing trend in the likelihood of college attendance from the less risky to the more risky strata; there is a non-monotonic but generally upwards trend in average depression score from the less risky to the more risky strata; and interestingly the pattern for likelihood of suicidal ideation increases incrementally across the first four strata and drops sharply in the last. The patterns among the control variables are interesting as well. Several of

TABLE 3.3a: Weighted Means of Treatment, Outcome, and Selected Control Variables (Measured at Wave 1) by Propensity Score Stratum, for Unexposed at Wave 1

	Stratum					
	1	**2**	**3**	**4**	**5**	**TOT**
p-score range:	[0.0-0.03)	[0.03-0.055)	[0.055-0.1)	[0.1-0.25)	[0.25-1.0)	**0.001-0.732**
Treatment						
Viol2	0.019	0.029	0.094	0.177	0.570	**0.082**
Outcome						
College Attend	0.729	0.638	0.546	0.422	0.234	**0.575**
Suicidality-3	0.056	0.056	0.062	0.065	0.048	**0.059**
CES-D3	5.018	5.323	4.972	5.163	6.400	**5.194**
Controls						
Female	0.773	0.579	0.414	0.293	0.198	**0.517**
White	0.772	0.770	0.696	0.659	0.466	**0.712**
Black	0.149	0.167	0.218	0.257	0.393	**0.206**
18-20	0.278	0.341	0.356	0.294	0.253	**0.309**
21-22	0.432	0.413	0.405	0.454	0.460	**0.429**
23-25	0.290	0.245	0.238	0.252	0.288	**0.262**
CES-D1	2.506	3.437	3.822	5.046	6.661	**3.789**
Suicidality-1	0.108	0.128	0.092	0.105	0.089	**0.107**
N	921	603	538	487	125	**2,674**

them show intuitively predictable patterns: moving from the least to the most risky strata, the composition of each stratum becomes monotonically more male-dominated, monotonically less white and more black, and monotonically higher on depression score at Wave 1. Age and Wave 1 suicidality show no obvious trends across propensity score strata.

Table 3.3b provides the same descriptive statistics across propensity score strata for the subsample that was exposed to violence at Wave 1, and some similar patterns emerge. As before, there is a monotonic upwards trend in the rate of Wave 2 violence exposure. Reaffirming findings from the previous chapter, we also see across all strata that the risk of Wave 2 violence exposure among those who were exposed at Wave 1 is much higher than among those who were not exposed at Wave 1. As in the other subsample, as we move from the least to the most risky stratum there is a generally decreasing trend in the rate of college attendance, a non-monotonic

TABLE 3.3b: Weighted Means of Treatment, Outcome, and Selected Control Variables (Measured at Wave 1) by Propensity Score Stratum, for Exposed at Wave 1

	Stratum							
	1	**2**	**3**	**4**	**5**	**6**	**7**	**TOT**
p-score range:	[0.0-0.3)	[0.3-0.355)	[0.355-0.425)	[0.425-0.47)	[0.47-0.575)	[0.575-0.695)	[0.695-1.0)	**0.160-0.843**
Treatment								
Viol2	0.289	0.374	0.427	0.532	0.537	0.700	0.764	**0.511**
Outcome								
College	0.459	0.460	0.480	0.379	0.318	0.291	0.235	**0.377**
Suicidality-3	0.100	0.077	0.030	0.092	0.060	0.081	0.088	**0.073**
CES-D3	5.504	5.069	4.862	6.384	5.794	7.804	6.833	**5.905**
Controls								
Female	0.474	0.348	0.224	0.361	0.352	0.191	0.381	**0.323**
White	0.554	0.550	0.566	0.468	0.513	0.548	0.589	**0.541**
Black	0.383	0.292	0.359	0.433	0.358	0.307	0.319	**0.348**
18-20	0.302	0.253	0.208	0.214	0.210	0.206	0.187	**0.227**
21-22	0.383	0.431	0.478	0.453	0.439	0.394	0.546	**0.437**
23-25	0.315	0.317	0.314	0.333	0.351	0.400	0.267	**0.336**
CES-D1	4.344	4.309	3.476	4.575	6.275	6.067	9.511	**5.356**
Suicidality-1	0.149	0.129	0.070	0.197	0.226	0.188	0.223	**0.167**
N	139	117	125	89	138	129	61	**798**

but generally upwards trend in Wave 3 CES-D score, and no linear pattern with respect to rates of suicidal ideation. The patterns among the control variables are less clear here than in the other subsample, but some trends emerge: in particular, there are generally (though by no means monotonically) upwards trends in depression score and in rates of suicidal ideation as we move

from the least to the most risky strata. The patterns with respect to age remain indistinct here, and patterns for sex and race compositions of the strata are far less evident than previously.

Comparing the two subsamples descriptively on the basis of these tables, though, highlights key differences between them. Perhaps the biggest difference is the distribution of the propensity score itself: among those who were unexposed at Wave 1, the propensity scores for exposure at Wave 2 range from about 0.001 to 0.732, with the vast majority (over 95% of the subsample) having propensity scores below 0.25; whereas among those who were exposed at Wave 1, the propensity scores for Wave 2 re-exposure range from 0.16 to 0.843, with 82.6% having propensity scores higher than 0.3. In other words, even the least at-risk among those who were exposed at Wave 1 have a higher chance of Wave 2 exposure than nearly all of those who were not exposed at Wave 1. The overlap in propensity score distributions among the two subsamples is so slight that I feel fully justified in the decision to treat them as separate groups with substantively different risk patterns. This is consistent with the finding in the previous chapter that Wave 1 violence exposure was by far the factor most obviously associated with higher risk of Wave 2 violence exposure. This is also consistent with the literature on re-victimization patterns (Lauritsen & Quinet 1995), suggesting that for many of these individuals victimization may not be a recurring problem so much as a persistent condition (Finkelhor et al 2007). Other patterns appear via such comparisons: as a whole, the Wave 1 unexposed subsample has a higher proportion of women, a higher proportion of whites and a lower proportion of blacks, and substantially higher average Wave 1 CES-D scores and rates of suicidality than the Wave 1 exposed subsample, reinforcing findings (e.g. Swearer et al 2010) that violence is unevenly distributed across certain individual and group characteristics.

The descriptive comparisons offered by Tables 3.3a and 3.3b are suggestive, but as a direct test of Hypothesis 1, which predicts that those who are exposed at Wave 1 will have worse overall outcomes at Wave 3 than those who are unexposed on Wave 1, it is important to establish and test direct differences in outcome patterns between those who are exposed and unexposed at Wave 1. It is also valuable, in light of theories regarding re-victimization and the upshot of evidence thus far, to examine the differences between those who are exposed and unexposed to violence at Wave 1 in the rates at which they suffer Wave 2 violence exposure. Table 3.4 displays direct comparisons between the two main subsamples in terms of their rates of exposure

TABLE 3.4: Weighted Descriptive Statistics for Treatment and Outcome Variables across Subgroups Based on Wave 1 Violence Exposure, and Tests of Difference

	Panel 1: Unexposed at Wave 1				**Panel 2: Exposed at Wave 1**				**Panel 3: Diff**	
	$\bar{X}_1$	S_1	range	N	$\bar{X}_2$	S_2	Range	N	$\bar{X}_2$-$\bar{X}_1$	t (p)
Wave 2 violence	0.107	0.364	0 – 1	2674	0.516	0.534	0 – 1	798	0.409	23.92 (0.000)
Wave 3 college enrollment	0.570	0.536	0 – 1	2673	0.369	0.504	0 – 1	798	-0.201	-9.10 (0.000)
Wave 3 suici-dal ideation	0.059	0.249	0 – 1	2615	0.073	0.275	0 – 1	777	0.014	1.30 (0.195)
Wave 3 CES-D score	5.224	4.512	0 – 28	2656	5.952	5.055	0 – 23	797	0.728	3.75 (0.000)

to the key "treatment" variable (Wave 2 violence exposure) and their patterns on the outcome variables at Wave 3 (college enrollment, suicidal ideation in the last 12 months, and depression score on the CES-D scale), as well as significance tests of difference between the groups' means.

Probably most immediately evident and distressing among the descriptive findings is the enormous difference in the rates at which the two subsamples are exposed to school violence at Wave 2. The first row of Table 3.4 shows that the rate of Wave 2 violence exposure is nearly five times higher among those who exposed at Wave 1 than among those who were not: only 10.7% of those who were unexposed at Wave 1 are exposed at Wave 2, whereas over 51% of those who were exposed at Wave 1 are exposed again at Wave 2. This is again consistent with

other findings on re-victimization patterns, reinforcing the finding from the earlier tables and from the previous chapter that prior victimization is a strong predictor of future risk (Lauritsen & Quinet 1995) and giving further support to the notion that members of this subset may be trapped in a "victimization condition" (Finkelhor et al 2007). This also provides implicit justification for the life-course framing applied to this investigation. It is clear that these two groups have very different relationships with violence exposure, and that this plays out over many years of experience and likely accrues further differences in other forms of disadvantage (cf. Sampson, Sharkey, & Raudenbush 2008; Wodtke, Harding, & Elwert 2010 for analyses of the effects of long-term exposure to concentrated disadvantage).

More directly relevant to the hypotheses stated earlier, the results in Table 3.4 reveal consistently worse patterns for those who were exposed to violence at Wave 1 in terms of both academic and mental health outcomes. Compared to those who did not experience violence at Wave 1, those with previous exposure are less than 2/3 as likely to enroll in college (36.9% vs 57%). Those who were exposed at Wave 1 also have about 1/4 higher likelihood of reporting suicidal ideation (7.3% vs. 5.9%), and show higher average depression score by about 3/4 of a point (5.224 vs. 5.952, representing a modest difference of about 0.16 of a joint standard deviation). The differences between the groups are highly statistically significant for college enrollment rates and depression scores, though not significant for suicidal ideation.

These results provide qualified but consistent support for Hypothesis 1: the overall rates of negative outcomes are higher for those who were exposed to violence at Wave 1 than for those who were not exposed at Wave 1, in ways that are both substantively meaningful and generally statistically significant. This finding is entirely separate from the questions of whether

Wave 2 violence exposure exerts causal effects on these outcomes and whether the size of this effect varies across these two groups, which will be examined in the next section.

Propensity Score Model Results

For all three outcomes presented here – college enrollment, suicidal ideation, and depression index score – I present an analysis in two stages. The first stage for each outcome features direct comparisons of the stratum-specific treatment effects as well as the overall average treatment effects (ATEs) for each Wave 1 exposure subsample. The second includes the propensity score strata as controls in a series of multivariate models in order to examine simultaneous associations with other relevant covariates. All the following tables feature two panels: panel 1 contains the analyses for those who were unexposed at Wave 1, and panel 2 includes the analyses for those who were exposed Wave 1. For the direct comparison tables, the ATEs are presented in a separate row; for the multivariate models, the coefficient estimates for the main effect of Wave 2 violence exposure can be interpreted as the ATE of isolated exposure in panel 1, and as the ATE of repeated exposure in panel 2. In both panels for the multivariate model tables, Model 1, the naïve model, directly controls only for propensity score stratum assignment and provides ATE estimates identical to the direct comparisons; Model 2 also controls for several demographic factors; and Model 3 additionally introduces some other relevant individual-level academic and behavioral characteristics.

Table 3.5 presents weighted estimates of stratum-specific effects of Wave 2 violence exposure on Wave 3 college enrollment, separately for those who were and were not exposed at Wave 1. The results are broadly consistent with my Hypotheses 2 and 3. Panel 1 shows that among those who were not exposed at Wave 1, the differences between the treated and untreated all run in the negative direction: that is, among those who had no previous exposure, being

victimized by violence consistently reduces the likelihood of enrolling in college. Overall, we see an odds ratio of 0.747, meaning that Wave 2 violence is associated with an approximately 25% reduction in the odds of college attendance for this population. While none of the stratum-specific effects achieve statistical significance, the ATE is marginally significant at the 0.1 level. By contrast, Panel 2 shows no consistent pattern for the effect of Wave 2 victimization on Wave 3 college enrollment among those who were already exposed by Wave 1. The directions of the stratum-specific effects are both negative and positive, and the ATE (with an odds ratio of 1.1) is nowhere near statistical significance.

TABLE 3.5: Weighted Propensity Score Stratum-Specific Treatment Effects [95% CIs] of Wave 2 Violence Exposure on Wave 3 College Enrollment, Separately by Wave 1 Exposure Status

	Panel 1: Not Exposed at Wave 1			Panel 2: Exposed at Wave 1		
Strat	Z = 0	Z = 1	Diff, OR (t, p)	Z = 0	Z = 1	Diff, OR (t, p)
1	0.733 [0.700-0.765]	0.722 [0.472-0.972]	-0.011, 0.986 (-0.08, 0.934)	0.494 [0.390-0.598]	0.346 [0.171-0.522]	-0.147, 0.702 (-1.41, 0.161)
2	0.637 [0.594-0.681]	0.441 [0.187-0.696]	-0.196, 0.692 (-1.50, 0.135)	0.483 [0.357-0.608]	0.423 [0.260-0.586]	-0.060, 0.876 (-0.57, 0.572)
3	0.541 [0.493-0.590]	0.481 [0.324-0.638]	-0.061, 0.888 (-0.73, 0.467)	0.480 [0.357-0.603]	0.479 [0.327-0.632]	-0.001, 0.998 (-0.01, 0.991)
4	0.423 [0.370-0.475]	0.380 [0.263-0.496]	-0.043, 0.898 (-0.67, 0.506)	0.403 [0.243-0.564]	0.339 [0.196-0.482]	-0.064, 0.841 (-0.58, 0.564)
5	0.284 [0.159-0.410]	0.197 [0.097-0.297]	-0.088, 0.692 (-1.07, 0.288)	0.278 [0.164-0.392]	0.336 [0.216-0.456]	0.058, 1.210 (0.69, 0.493)
6	–	–	–	0.123 [0.024-0.220]	0.357 [0.249-0.465]	**0.234, 2.906 (3.14, 0.002)**
7	–	–	–	0.203 [0.006-0.411]	0.236 [0.106-0.365]	0.033, 1.163 (0.26, 0.795)
ATE			*0.747 (-1.67, 0.095)*			1.100 (0.57, 0.566)

We can examine these patterns in some more detail in Table 3.6, which presents the results estimating the effects of Wave 2 violence on Wave 3 college enrollment. The results from Model 1 (the naïve model) repeat the ATEs from the previous table: in panel 1, the effect of isolated violence exposure at Wave 2 on those who were unexposed at Wave 1 is to reduce the odds of college enrollment by approximately 25% (OR: 0.747, 95% CI: 0.53-1.05, p = 0.095),

TABLE 3.6: Odds Ratios [95% CIs] for Effect of Wave 2 Violence on Wave 3 College Enrollment

	Panel 1: Unexposed at Wave 1			Panel 2: Exposed at Wave 1		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Wave 2 Violence	**0.747˜ [0.53-1.05]**	**0.66* [0.46-0.96]**	**0.75 [0.50-1.13]**	**1.10 [0.79-1.53]**	**1.13 [0.80-1.62]**	**1.04 [0.68-1.58]**
P-Score: stratum 1	–	–	–	–	–	–
2	0.63*** [0.49-0.80]	0.65** [0.50-0.85]	0.78˜ [0.57-1.05]	1.02 [0.59-1.76]	1.04 [0.60-1.82]	0.81 [0.42-1.56]
3	0.43*** [0.33-0.55]	0.53*** [0.40-0.71]	0.79 [0.57-1.10]	1.10 [0.65-1.88]	1.05 [0.59-1.89]	0.87 [0.45-1.70]
4	0.27*** [0.21-0.35]	0.39*** [0.29-0.54]	0.57** [0.40-0.82]	0.69 [0.38-1.26]	0.74 [0.38-1.43]	0.82 [0.40-1.68]
5	0.13*** [0.08-0.21]	0.24*** [0.13-0.42]	0.49* [0.25-0.94]	0.53* [0.31-0.91]	0.60˜ [0.34-1.08]	0.60 [0.31-1.18]
6	–	–	–	0.46** [0.26-0.81]	0.62 [0.33-1.14]	0.44* [0.20-0.96]
7	–	–	–	0.34** [0.16-0.72]	0.63 [0.27-1.47]	0.75 [0.28-2.00]
Female	–	0.85 [0.68-1.05]	0.86 [0.67-1.10]	–	0.86 [0.60-1.25]	0.74 [0.48-1.14]
Race: white	–	–	–	–	–	–
black	–	0.94 [0.73-1.20]	1.04 [0.78-1.37]	–	0.66* [0.46-0.96]	0.70 [0.45-1.09]
other	–	0.94 [0.61-1.43]	0.74 [0.43-1.25]	–	0.75 [0.40-1.39]	0.57 [0.27-1.21]
Age: 18-20	–	–	–	–	–	–
20-21	–	1.36** [1.10-1.70]	1.85*** [1.44-2.39]	–	1.04 [0.69-1.59]	1.35 [0.83-2.22]
22-25	–	1.31* [1.02-1.68]	2.10*** [1.65-2.13]	–	0.92 [0.59-1.43]	1.40 [0.81-2.44]
Felt college is likely, Wave 1	–	2.23*** [1.99-2.51]	1.87*** [1.65-2.13]	–	2.05*** [1.73-2.41]	1.73*** [1.42-2.10]
Approximate GPA, Wave 1	–	–	2.73*** [2.28-3.25]	–	–	2.14*** [1.58-2.90]
Ever repeat a grade, Wave 1	–	–	0.42*** [0.30-0.58]	–	–	0.43*** [0.26-0.70]
Ever expelled from school, Wave 1	–	–	0.37˜ [0.12-1.16]	–	–	0.67 [0.31-1.48]
Feel safe at school, Wave 1	–	–	0.92 [0.81-1.04]			1.04 [0.87-1.24]
Constant	2.758	0.067	0.007	0.804	0.051	0.017

Note: "Ever enrolled in college" defined by the question "What is the highest grade or year of regular school you completed?" Outcome is coded as 1 if respondent indicated attending at least one year of post-secondary education (regardless of whether a degree was obtained) and 0 otherwise. ˜ = p<0.1, * = p<0.05, ** = p<0.01, *** = p<0.001

and the results from Model 1 in panel 2 show that repeated exposure at Wave 2 appears to exert no statistically or substantively significant effect on the likelihood of college enrollment (OR: 1.1, 95% CI: 0.79-1.53, $p = 0.566$). It is interesting to note that net of Wave 2 violence exposure, odds of college enrollment generally decrease from the lowest- to the highest-risk propensity score strata in both subsamples, suggesting that risk propensity itself predicts lower college enrollment success even if it does not affect the size of the effect of exposure on enrollment.

Further interesting findings are revealed by the two versions of Model 2 in Table 3.6, controlling for gender, age, race, and respondents' Wave 1 self-reports of whether they feel themselves likely to go to college. The first evident result is that the estimates of violence's main effects are largely unchanged: panel 1 shows that net of propensity score stratum and other variables, the effect of isolated Wave 2 violence is to reduce the odds of college enrollment by about 34% (OR: 0.66, 95% CI: 0.47-0.96, $p<0.05$), and the effect of repeated violence exposure shown in panel 2 remains both substantively and statistically insignificant (OR: 1.13, 95% CI: 0.80-1.62, $p>0.10$). These models additionally reveal that, for both groups, gender and race are not consistently significant predictors of college enrollment net of the other factors – though the difference between blacks and whites is significant among the group that was exposed at Wave 1 (OR: 0.66, $p<0.05$) – suggesting that violence exposure itself may be a major factor contributing to the extant pattern of black-white educational attainment gaps (cf. Gregory, Skiba, & Noguera 2010). We also see that Wave 1 self-assessment of the likelihood of college attendance is a hugely important predictor of actual college attendance at Wave 3 for both groups (among the unexposed, OR: 2.23, 95% CI: 1.99-2.51, $p<0.001$; among the exposed, OR: 2.05, 95% CI: 1.73-2.41, $p<0.001$). Last, and surprising, are the results regarding age category: whereas older respondents among the unexposed in panel 1 are more likely to enroll in college, the age

categories among the exposed in panel 2 show no significant relationship with enrollment. The pattern among the unexposed makes sense if we suppose, reasonably, that some students who defer college attendance after high school will choose to enroll at a later age; the pattern among the exposed is more surprising, though it accords with other findings that exposure to violence stifles educational success (e.g. Harding 2009; Wodtke, Harding, & Elwert 2011).

The final points with regard to college enrollment are given by the results from Model 3 in panels 1 and 2 of Table 3.6. These models, which control for individual predictors in addition to those included in Model 2, add interesting new color to the picture so far presented. In the first place, the effect of Wave 2 violence exposure on Wave 3 college enrollment among those who were unexposed at Wave 1, shown in panel 1, is no longer statistically significant (OR: 0.75, 95% CI: 0.50-1.13, $p>0.10$), despite being substantively similar to the effect size estimated in Model 1. The impact on those who were exposed at Wave 1, shown in panel 2, remains insignificant as predicted (OR: 1.04, 95% CI: 0.68-1.58, $p>0.10$). As in Model 2, gender and race do not significantly impact the likelihood of college attendance, net of other factors, for either subsample; the black-white gap from Model 2 in Panel 2 has disappeared. Age category presents a more intuitive pattern here than it did in Model 2, being associated with monotonically increasing odds of college enrollment for both the previously exposed and the previously unexposed, though the panel 2 results again find the differences not to be significant. As before, Wave 1 college aspirations have a huge impact on both groups, raising the predicted odds of enrollment by between 87% and 73%. Other academic and behavioral characteristics, newly introduced in Model 3, are powerfully associated with college enrollment. Approximate self-reported GPA (derived from academic questions in the Add Health survey) is a hugely important predictor of college enrollment, with each one-point increase in GPA more than doubling the

odds of enrollment among both subsamples. Consistent with other research which shows a negative effect of grade retention on academic attainment (e.g. Jacob & Lefgren 2007, Roderick 1994), Model 3 finds that grade retention as of Wave 1 dramatically reduces the chances of later successful enrollment in college – about a 57% decline in odds for both groups. Interestingly, ever being expelled from school as of Wave 1 reduces the odds of college enrollment among those who were not exposed at Wave 1 (OR: 0.37, 95% CI: 0.12-1.16, $p<0.1$), and is associated with substantive though not significantly lower odds of enrollment among those who were exposed at Wave 1 (OR: 0.67, 95% CI: 0.31-1.24, $p>0.10$) – possibly because the rarity of this sanction makes for a subgroup too small for a reliable estimate. Finally, self-reported feeling safe at school appears, surprisingly, to be largely unimportant net of other factors; once actual exposure, demographics, and academic characteristics are taken into account, subjective sense of safety at school does not appear to affect odds of college enrollment.

To summarize the results regarding college enrollment: isolated exposure at Wave 2 on those who were unexposed at Wave 1 sharply reduces the likelihood of ever attending college. This effect is at least marginally significant in Models 1 and 2, and persists though fails to achieve statistical significance in Model 3 when pretreatment academic characteristics are taken into account. Among those who were exposed at Wave 1, re-exposure at Wave 2 exerts no statistically or substantively significant effect on college enrollment under any specification. This is consistent with predictions from Hypotheses 2 and 3. Unsurprisingly, age, Wave 1 college aspirations, and academic characteristics strongly predict enrollment for both groups.

Turning now to suicidal ideation, we begin with direct comparisons of stratum-specific treatment effects, presented in Table 3.7. Here the patterns with respect to Hypotheses 2 and 3 are, if anything, generally clearer than they were in the analyses of college enrollment. For one,

TABLE 3.7: Weighted Propensity Score Stratum-Specific Treatment Effects [95% CIs] of Wave 2 Violence Exposure on Wave 3 Suicidal Ideation, Separately by Wave 1 Exposure Status

	Panel 1: Not Exposed at Wave 1			Panel 2: Exposed at Wave 1		
Strat	Z = 0	Z = 1	Diff, OR (t, p)	Z = 0	Z = 1	Diff, OR (t, p)
1	0.051 [0.036-0.067]	0.297 [0.010-0.584]	*0.254, 5.776* *(1.69, 0.092)*	0.115 [0.048-0.181]	0.064 [0.036-0.298]	-0.050, 0.561 (-0.99, 0.325)
2	0.057 [0.037-0.077]	0.073 [0.065-0.211]	0.015, 1.269 (0.22, 0.827)	0.099 [0.018-0.179]	0.041 [0.027-0.231]	-0.057, 0.418 (-1.13, 0.260)
3	0.060 [0.037-0.084]	0.086 [0.008-0.180]	0.026, 1.424 (0.52, 0.603)	0.017 [0.003-0.094]	0.048 [0.022-0.189]	0.031, 2.891 (1.03, 0.305)
4	0.059 [0.033-0.085]	0.092 [0.026-0.157]	0.032, 1.547 (0.91, 0.364)	0.130 [0.013-0.247]	0.058 [0.013-0.172]	-0.072, 0.444 (-1.05, 0.297)
5	0.031 [0.013-0.075]	0.059 [0.003-0.115]	0.028, 1.895 (0.77, 0.445)	0.070 [0.006-0.134]	0.052 [0.009-0.124]	-0.019, 0.735 (-0.42, 0.676)
6	–	–	–	0.144 [0.004-0.284]	0.053 [0.013-0.117]	-0.090, 0.371 (-1.18, 0.240)
7	–	–	–	0.085 [0.002-0.319]	0.082 [0.026-0.221]	-0.003, 0.970 (-0.03, 0.979)
ATE			**1.960** **(2.16, 0.031)**			*0.562* *(-1.73, 0.084)*

among the unexposed at Wave 1, Wave 2 exposure is associated with consistent and generally large increases in the risk of suicidal ideation – especially those in the least risky stratum, whose risk jumps from 5% to 30% (p = 0.092). The ATE among the Wave 1 unexposed suggests that Wave 2 violence exposure is associated with a near-doubling in the odds of suicidal ideation at Wave 3 (OR = 1.96, p = 0.031). By contrast, there is no consistent pattern in the size or direction of the stratum-specific effects of Wave 2 violence exposure on Wave 3 suicidal ideation among those who were exposed at Wave 1. Oddly, most effects are negative in direction – that is, those who were exposed report lower average rates of suicidal ideation – to the point that the ATE indicates a marginally significant reduction in odds of suicidality (OR = 0.562, p = 0.084).

The estimated effects of Wave 2 violence exposure on Wave 3 suicidal ideation, shown in Table 3.8, are also generally consistent with Hypotheses 2 and 3. Model 1, the naïve model, repeats the ATEs from Table 3.7: among those with no violence exposure at Wave 1, exposure at Wave 2 causes a 96% increase in the odds of self-reporting suicidal ideation at Wave 3 (OR:

TABLE 3.8: Odds Ratios [95% CIs] for Effect of Wave 2 Violence on Wave 3 Suicidal Ideation

	Panel 1: Unexposed at Wave 1			Panel 2: Exposed at Wave 1		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Wave 2 Violence	**1.96* [1.06-3.61]**	**1.89* [1.03-3.47]**	**1.99* [1.08-3.71]**	**0.56˜ [0.29-1.08]**	**0.54˜ [0.28-1.05]**	**0.54˜ [0.27-1.06]**
P-Score: stratum 1	–	–	–	–	–	–
2	1.02 [0.63-1.66]	0.94 [0.57-1.56]	0.80 [0.48-1.35]	0.78 [0.30-2.06]	0.75 [0.28-1.98]	0.74 [0.28-1.97]
3	1.06 [0.63-1.77]	0.95 [0.53-1.70]	0.76 [0.42-1.35]	0.30* [0.10-0.88]	0.32* [0.10-0.97]	0.32* [0.11-0.99]
4	1.03 [0.59-1.78]	0.93 [0.49-1.77]	0.63 [0.33-1.19]	1.03 [0.38-2.78]	1.13 [0.40-3.17]	1.02 [0.37-2.84]
5	0.55 [0.20-1.48]	0.52 [0.18-1.50]	0.28* [0.09-0.82]	0.66 [0.25-1.70]	0.71 [0.26-1.92]	0.60 [0.21-1.77]
6	–	–	–	0.99 [0.36-2.75]	1.15 [0.41-3.23]	1.08 [0.37-3.20]
7	–	–	–	1.08 [0.34-3.41]	1.09 [0.33-3.59]	0.88 [0.24-3.30]
Female	–	1.04 [0.69-1.57]	0.77 [0.50-1.17]	–	1.46 [0.82-2.60]	1.02 [0.53-1.96]
Race: white	–	–	–	–	–	–
black	–	0.68 [0.40-1.16]	0.74 [0.43-1.27]	–	0.51˜ [0.25-1.02]	0.52~ [0.25-1.11]
other	–	1.71 [0.84-3.48]	1.66 [0.80-3.43]	–	1.63 [0.69-3.85]	1.90 [0.77-4.69]
Age: 18-20	–	–	–	–	–	–
20-21	–	0.57** [0.38-0.85]	0.51*** [0.34-0.76]	–	0.87 [0.44-1.74]	0.87 [0.43-1.75]
22-25	–	0.35*** [0.19-0.63]	0.27*** [0.15-0.50]	–	0.53 [0.23-1.22]	0.54 [0.23-1.28]
Educ: less than HS	–	–	–	–	–	–
HS grad	–	0.75 [0.43-1.32]	0.88 [0.49-1.55]	–	0.72 [0.29-1.79]	0.69 [0.26-1.81]
Some college	–	0.63 [0.36-1.10]	0.85 [0.48-1.52]	–	1.26 [0.55-2.91]	1.13 [0.47-2.70]
College grad	–	0.53 [0.36-1.20]	0.76 [0.33-1.77]	–	0.97 [0.22-4.18]	0.90 [0.19-4.25]
Suicidal ideation, Wave 1	–	–	2.42*** [1.46-4.02]	–	–	2.40* [1.20-4.80]
Depression score, Wave 1	–	–	1.06*** [1.03-1.10]	–	–	1.05~ [0.999-1.10]
Friend or relative committed suicide	–	–	1.79 [0.58-5.53]	–	–	1.71 [0.62-4.73]
Felt college is likely, Wave 1	–	–	0.85~ [0.71-1.01]	–	–	1.24 [0.92-1.66]
Constant	0.058	0.142	0.227	0.128	0.163	.053

Note: "Suicidal Ideation" defined by the question "During the past 12 months, have you ever seriously thought about committing suicide?" Outcome is coded as 1 if respondent responded "yes," and 0 if "no". ˜ = p<0.1, * = p<0.05, ** = p<0.01, *** = p<0.001

1.96, 95% CI: 1.06-3.61, p<0.05); among those who were exposed to violence at Wave 1, re-exposure at Wave 2 has a marginally significant negative effect on the odds of suicidal ideation at Wave 3 (OR: 0.56, 95% CI: 0.29-1.08, p<0.10). Unlike in the results for college enrollment, there appear to be no particular trends in the odds of suicidality across strata for either group.

Model 2, which introduces additional control variables, adduces substantively similar results. Most importantly, the main effects of violence exposure on odds of suicidal ideation remain virtually unchanged for both groups: isolated Wave 2 violence exposure increases by 89% the odds of Wave 3 suicidal ideation among those who were not exposed at Wave 1 (OR: 1.89, 95% CI: 1.03-3.47, p<0.05), and repeated exposure has a marginally significant negative impact on those who were exposed at Wave 1 (OR: 0.54, 95% CI: 0.28-1.05, p<0.10). Here we also see that for both groups, the likelihood of suicidal ideation net of other factors is the same for both sexes – consistent with other research showing that women have idle suicidal thoughts more often than men, but that the two sexes give suicide serious consideration at similar rates (e.g. Crosby et al 2011). Race differences, net of other factors, are insignificant for both groups; but age is a key factor, with both subgroups' odds of suicidal ideation declining monotonically from younger to older age groups (though the differences are not significant in Panel 2). And education, quite oddly, shows diverging trends: among those unexposed at Wave 1, higher education is monotonically (though not significantly) associated with lower odds of suicidal ideation; but among those who were exposed at Wave 1, higher education show no consistent pattern of association with the odds of suicidal ideation.

Moving from Model 2 to Model 3 across panels of Table 3.8, the main effects of violence exposure on odds of suicidal ideation remain remarkably stable for both groups. Among the unexposed at Wave 1, Model 3 shows that the effect of isolated Wave 2 exposure increases the

odds of Wave 3 suicidal ideation by 99% (OR: 1.99, 95% CI: 1.08-3.71, $p<0.05$), virtually identical to the 1.96 odds ratio observed for this effect in Model 1. Among those who did suffer Wave 1 exposure, Wave 2 re-exposure still exerts only a marginally significant effect on the odds of suicidal ideation at Wave 3 (OR: 0.54, 95% CI: 0.27-1.06, $p<0.10$). As before, gender and race predict no significant differences in the odds of Wave 3 suicidal ideation for either group net of the other factors. Age, too, retains its association: older subjects, regardless of previous exposure, were less likely to report suicidal ideation, though the differences were only significant in Panel 1. And the diverging pattern of association with educational attainment – with more education predicting lower odds of suicidal ideation among those who were unexposed at Wave 1, but no particular pattern among those who were exposed at Wave 1 – persists in Model 3, with none of the coefficient estimates achieving statistical significance. The additional background variables added to Model 3 also reveal interesting, and in some cases surprising, estimates. Predictably, previous suicidal ideation reported during Wave 1 strongly predicts suicidal ideation at Wave 3 for both the previously exposed (OR: 2.42, 95% CI: 1.46-4.02, $p<0.01$) and the previously unexposed (OR: 2.40, 95% CI: 1.20-4.80, $p<0.05$). Depression score reported at Wave 1 also predicts the likelihood of suicidal ideation at wave 3 for both the previously exposed (OR: 1.06, 95% CI: 1.03-1.10, $p<0.001$) and the previously unexposed (OR: 1.05, 95% CI: 0.999-1.10, $p<0.1$). Interestingly, though, having a friend or relative commit suicide increased the odds of suicidal ideation for both groups but neither to a conventional level of significance, perhaps because of the rarity of this event. Also perhaps surprising is that Wave 1 self-assessed likelihood of college attendance – often used as a proxy for future-orientation – had only a marginally significant negative association with odds of Wave 3 suicidal ideation for the unexposed at Wave 1, and no effect among the exposed at Wave 1, net of the other factors.

To summarize the results regarding suicidal ideation: isolated Wave 2 violence exposure sharply increases the odds of Wave 3 suicidal ideation among those who were unexposed at Wave 1. This effect is significant and remarkably stable across all model specifications. Among those who were exposed at Wave 1, re-exposure at Wave 2 appears, if anything, to slightly reduce the likelihood of Wave 3 suicidal ideation, though these results are only marginally significant under all model specifications. The results for both groups are broadly consistent with predictions from Hypotheses 2 and 3. Unsurprisingly, age, Wave 1 depression, and Wave 1 suicidal ideation strongly predict Wave 3 suicidal ideation for both groups. Level of education attained by Wave 3 is also associated with suicidal ideation, but in opposite directions for both groups: more education is associated with lower odds of suicidal ideation among those who were unexposed at Wave 1, and higher odds among those who were exposed at Wave 1.

Table 3.9 shifts to focus on depression, measured by CES-D score. The stratum-specific treatment effects here are surprising. Among those who were unexposed at Wave 1, we observe

TABLE 3.9: Weighted Propensity Score Stratum-Specific Treatment Effects [95% CIs] of Wave 2 Violence Exposure on Wave 3 Depression Score, Separately by Wave 1 Exposure Status

	Panel 1: Not Exposed at Wave 1			Panel 2: Exposed at Wave 1		
Strat	Z = 0	Z = 1	Diff (t, p)	Z = 0	Z = 1	Diff (t, p)
1	5.045 [4.748-5.342]	6.057 [3.529-8.584]	1.012 (0.78, 0.433)	5.587 [4.778-6.397]	5.240 [3.688-6.792]	-0.347 (-0.39, 0.699)
2	5.364 [4.973-5.754]	5.240 [3.994-6.485]	-0.124 (-0.19, 0.852)	5.009 [3.591-6.427]	5.111 [3.903-6.320]	0.102 (0.11, 0.915)
3	4.976 [4.582-5.370]	5.057 [3.745-6.368]	0.081 (0.12, 0.908)	4.455 [3.542-5.367]	5.324 [4.083-6.565]	0.869 (1.10, 0.273)
4	5.069 [4.639-5.499]	5.841 [4.691-6.992]	0.772 (1.24, 0.216)	5.745 [3.980-7.510]	7.449 [5.887-9.011]	1.704 (1.41, 0.164)
5	6.414 [5.187-7.642]	6.296 [5.080-7.513]	-0.118 (-0.13, 0.894)	5.231 [4.100-6.362]	6.283 [4.976-7.591]	1.052 (1.19, 0.237)
6	–	–	–	7.119 [5.628-8.610]	7.117 [5.902-8.331]	-0.002 (-0.00, 0.998)
7	–	–	–	5.133 [3.424-6.842]	7.408 [6.086-8.731]	**2.275** **(2.04, 0.046)**
ATE			0.374 (1.02, 0.309)			*0.679* *(1.83, 0.068)*

little consistent pattern in the effect sizes and directions: the differences are as high as a full point and as small as 0.08 of a point, and they run both positive and negative; the ATE is 0.374, but this is not statistically significant. On the other hand, the effects on those who were exposed at Wave 1 are generally positive, though still varying in size from nearly zero to as much as 2.275, and the ATE is a marginally significant 0.679. These results are generally not consistent with Hypothesis 2, though we can still interpret this as qualified support for Hypothesis 3.

Table 3.10 shows the multivariate models predicting effects of Wave 2 victimization on Wave 3 depression, measured by CES-D score. In all three models, again, the results are rather less consistent with Hypothesis 2, though Hypothesis 3 is generally supported. Model 1, the naïve model, repeats the null effect of violence exposure on CES-D score in panel 1 (B=0.374, SE=0.367, $p>0.05$) and a marginally significant effect in panel 2 (B=0.679, SE=0.372, $p<0.10$); this is to say, neither isolated nor repeated exposure at Wave 2 appears to exert a statistically or substantively meaningful average effect on depression at Wave 3. There are also no consistent patterns in average CES-D scores across propensity score strata.

Introducing additional background variables in Model 2 reveals some associations that are both predictable and meaningful, but leaves the estimated main effects of both isolated (B=0.323, SE=0.342, $p>0.05$) and repeated violence exposure (B=0.605, SE=0.343, $p<0.1$) mostly unchanged from the previous model. The analyses also reveal that, net of all other factors, women have higher average CES-D scores than men among both the Wave 1 unexposed (B=1.416, SE=0.183, $p<0.001$) and the Wave 1 exposed (B=1.455, SE=0.385, $p<0.001$). This is consistent with the results from the previous chapter, as well as a wealth of previous research showing higher average depression levels among women (e.g. Nolen-Hoeksema 2001, Piccinelli & Wilkinson 2000, Weissman et al 1996). The sex differences in depression score for both

TABLE 3.10: OLS Coefficients (Std Errors) for Effect of Wave 2 Violence on Wave 3 CES-D Score

	Panel 1: Unexposed at Wave 1			Panel 2: Exposed at Wave 1		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Wave 2 Violence	**0.373**	**0.323**	**0.294**	**0.679˜**	**0.605˜**	**0.609˜**
	(0.367)	**(0.342)**	**(0.343)**	**(0.372)**	**(0.343)**	**(0.344)**
P-Score: stratum 1	–	–	–	–	–	–
2	0.291	0.256	0.268	-0.512	-0.305	-0.274
	(0.246)	(0.238)	(0.237)	(0.636)	(0.603)	(0.608)
3	-0.109	-0.073	-0.064	-0.763	-0.119	-0.074
	(0.246)	(0.245)	(0.244)	(0.538)	(0.500)	(0.503)
4	0.082	-0.099	-0.157	0.994	1.071˜	1.037
	(0.259)	(0.279)	(0.290)	(0.721)	(0.641)	(0.633)
5	1.072*	0.476	0.422	0.140	-0.157	-0.163
	(0.500)	(0.493)	(0.510)	(0.592)	(0.559)	(0.566)
6	–	–	–	1.351*	1.285*	1.256*
				(0.632)	(0.599)	(0.616)
7	–	–	–	1.037	-0.162	-0.312
				(0.698)	(0.746)	(0.747)
Female	–	1.416***	1.399***	–	1.455***	1.346***
		(0.183)	(0.182)		(0.385)	(0.394)
Race: white	–	–	–	–	–	–
black	–	-0.006	0.165	–	0.113	0.162
		(0.224)	(0.224)		(0.363)	(0.362)
other	–	0.581	0.581	–	0.801	0.872
		(0.449)	(0.454)		(0.714)	(0.714)
Age: 18-20	–	–	–	–	–	–
20-21	–	-0.104	-0.161	–	-0.285	-0.346
		(0.201)	(0.199)		(0.418)	(0.418)
22-25	–	-0.232	-0.381	–	-0.674	-0.778~
		(0.234)	(0.238)		(0.425)	(0.429)
Educ: less than HS	–	0.483	0.384	–	0.270	0.202
		(0.352)	(0.348)		(0.484)	(0.491)
HS grad	–	–	–	–	–	–
Some college	–	-0.782***	-0.679**	–	-0.91	-0.148
		(0.210)	(0.219)		(0.388)	(0.406)
College grad	–	-0.861**	-0.637*	–	-0.308	-0.247
		(0.276)	(0.290)		(0.601)	(0.650)
Depression score, Wave 1	–	0.218***	0.167***	–	0.265***	0.257***
		(0.022)	(0.023)		(0.037)	(0.038)
Suicidal ideation, Wave 1	–	–	1.336***	–	–	0.698
			(0.341)			(0.486)
Friend or relative committed suicide	–	–	0.417	–	–	0.338
			(0.543)			(0.669)
Felt college is likely, Wave 1	–	–	-0.258*	–	–	-0.010
			(0.114)			(0.154)
Felt accepted by others, Wave 1	–	–	-0.956***	–	–	-0.93
			(0.287)			(0.481)
Constant	5.058	4.036	6.019	5.510	3.691	3.817

Note: "CES-D score" measured on a 10-question modified version of Radloff's (1977) CES-D index, included with Add Health; higher numbers indicate more severe or more frequent depressive symptoms. ˜ = p<0.1, * = p<0.05, ** = p<0.01, *** = p<0.001

groups are consistent for both subpopulations, and are even virtually identical in size with the sex difference found in the previous chapter: about a point and a half out of 30 on the modified CES-D scale. Similarly unsurprising is the finding that CES-D score measured at Wave 1 is highly predictive of CES-D score at Wave 3 for both those who were unexposed (B=0.218, SE=0.022, p<0.001) and those who were exposed (B=0.265, SE=0.037, p<0.001) to Wave 1 violence; the higher one's reported level of depression at Wave 1, the higher one's predicted level of depression at Wave 3, net of all other factors. Age and race appear to play only a minor role in predicting depression score: the average scores across race groups are statistically identical in both panels, and while there is a monotonic trend of lower depression scores among older age categories for both exposure groups, the gap never reaches statistical significance. Interestingly, more education is monotonically associated with lower depression score for both groups, but only to a statistically significant extent among those who were not exposed at Wave 1.

Finally, Model 3 in both panels of Table 3.10 adds several additional pretreatment measures of socio-emotional well-being. Again, however, the introduction of these variables does not change the estimated main effect size for either subgroup by very much: the estimated effect remains substantively small and marginally significant among those who were exposed to violence at Wave 1 (B=0.609, SE=0.344, p<0.1) and neither sizeable nor significant among those who were not (B=0.294, SE=0.343, p>0.1), and there did not appear to be any stratum-specific patterns in outcome for either group. In other words, violence exposure at Wave 2 does not appear to change the average Wave 3 depression score much for either subsample. Consistent with earlier results, women have higher average CES-D scores for both the previously unexposed (B=1.399, SE=0.182, p<0.001) and the previously exposed (B=1.346, SE=0.394, p<0.001) net of other factors, and higher depression score at Wave 1 is highly associated with higher depression

score at Wave 3 among both the previously unexposed (B=0.167, SE=0.023, p<0.001) and the previously exposed (B=0.257, SE=0.038, p<0.001). Both race and age appear to play the same modest to negligible role they did in Model 2, and education is still associated with lower depression score but only to a significant extent among those who were unexposed at Wave 1. Here we also see that Wave 1 suicidal ideation is highly associated with increased Wave 3 depression score among those who were not exposed to violence at Wave 1 (B=1.336, SE=0.341, p<0.001) but not among those who were (B=0.698, SE=0.486, p<0.05); the effect in panel 1, nearly as large as the gender gap, is highly notable and suggests a persistence of emotional health concerns over several years. The other Wave 1 mental health predictors in this model, though, showed significant associations only among those who were previously unexposed: feeling that college is likely, a common measure of future-orientation, reduces average depression score (B=-0.258, SE=0.114, p<0.05), and reporting that one feels accepted by others also exerts a strong protective effect (B=-0.956, SE=0.287, p<0.001). Surprisingly, the association between having a friend or relative who committed suicide as of Wave 1 and depression score at Wave 3 – a prediction supported by theories about the social network contagion of mental health problems (e.g. Eisenberg et al 2013; Prinstein 2007; Rosenquist, Fowler, & Christakis 2011) – appears insignificant for both subgroups.

To sum up the effects reported in Table 3.10, while exposure to violence has a negative impact on depression score, all estimates of this effect are substantively small and none reach a conventional 0.05 level of significance. Moreover, contrary to expectations (and to the patterns from the other analyses), the effect size is higher for those who were previously exposed – that is, if anything, the effect of re-exposure on depression appears worse than the effect of first exposure. These patterns hold across all model specifications. This suggests that the effects of

violence on depression may accumulate rather than diminish over time, though the low significance of the effect size estimates cautions against reading too much into them.

In sum, the results shed new and sometimes unexpected light on the three hypotheses given earlier. Hypothesis 1, that those who suffer early exposure will have worse outcomes than those who suffer only later exposure, is upheld; Table 3.4 shows that compared to those who were unexposed at Wave 1, those who were exposed have tremendously higher risk of exposure at Wave 2, much lower rates of college enrollment, significantly higher average depression score, and higher rates of suicidal ideation (though not achieving significance). Hypothesis 2, that Wave 2 violence exposure for those who were not exposed at Wave 1 (i.e. isolated exposure) will exert a sharp negative effect, is partially upheld; the results conform to this pattern for suicidal ideation and generally conform for college enrollment in the multivariate models, but deviate from this pattern in the case of depression scores. Hypothesis 3, that Wave 2 violence exposure among those who were previously exposed at Wave 1 (i.e. repeated exposure) will not exert a significant effect, is generally upheld: for all outcomes examined, the effect of Wave 2 violence exposure on those who were exposed at Wave 1 is substantively small and never achieves a conventional level of significance, though in the case of depression we see marginally significant increases in score, and in the case of suicidality we see counterintuitive marginally significant reductions in risk.

DISCUSSION & CONCLUSION

Taken as a whole, the findings from these analyses generally support the chapter's three main hypotheses with some obvious caveats. This first hypothesis, that those who are exposed to violence early in life will show consistently worse outcomes later in life than those who were not so exposed, receives probably the most unequivocal support. The results displayed in Table 3.4

show that across the board, those who were exposed to Wave 1 violence manifest worse outcomes at Wave 3 than do those who were not exposed to Wave 1 violence – the former group is significantly less likely to go to college, scores significantly higher on the depression index, and is substantially (though only marginally significantly) more likely to report suicidal ideation. These results are consistent with findings by Shonkoff and colleagues (2012), among others, who suggest that early exposure to toxic stressors – including violence – exerts a more harmful effect. The strongest association by far, though, is the link between Wave 1 violence exposure and Wave 2 violence exposure; those who were exposed at Wave 1 are nearly five times more likely to suffer Wave 2 exposure than those who were not exposed at Wave 1. This too is consistent with previous analyses of re-victimization; early violence initiation seems powerfully associated with a higher likelihood of violence exposure becoming a chronic, or at least repeated, problem. Lauritsen and Quinet (1995), noting a similar pattern, suggest a two-fold explanation for why those who are victimized early are at elevated risk of later victimization: such re-victimizations are due not only to victims' underlying vulnerability, but also to changes to the individual him- or herself that result from the experience of victimization. This calls attention to the life course approach that motivated this study, and the question of whether those early victims are changed by the experience is taken up in the tests of the next two hypotheses. Of course, it is also possible that the students in these two groups are victimized for different reasons – for example, that the isolated victims are vulnerable by circumstance, whereas the repeat victims are those who put themselves at risk by interacting with violent peers, engaging in violent situations, and perhaps even perpetrating violence themselves. This will be investigated directly in the next chapter.

The second hypothesis is that, among students who do not report violence exposure at Wave 1, an isolated exposure at Wave 2 will cause a sharp increase in negative outcomes. This

hypothesis is generally but incompletely supported by the data. Panels 1 of Tables 3.5 and 3.6 show that under various model specifications, exposure to Wave 2 violence reduced the odds of college enrollment by between about 25% and 34% among those who do not have a history of previous violence exposures; while this effect persists at a substantively similar level in all three models, it falls below the 0.05 significance threshold after controlling for Wave 1 academic and demographic characteristics. Similarly, panels 1 of Tables 3.7 and 3.8 show the effects of Wave 2 violence exposure on Wave 3 suicidal ideation among those without a history of previous exposures; in all models, such exposure produces statistically significant increases in the odds of suicidal ideation of between 89% and 99%. Contrary to the prediction from hypothesis 2, however, the results shown in panels 1 of Tables 3.9 and 3.10 do not reveal a substantively or statistically meaningful effect of Wave 2 isolated violence exposure on Wave 3 depression.

The third hypothesis receives broad but qualified support. The causal investigations into all three key outcomes reveal that the effects of Wave 2 exposure to violence on students who had previously suffered Wave 1 exposure were occasionally substantively suggestive but never more than marginally significant; that is, among those with a pattern of past exposures, an additional exposure at Wave 2 produced little appreciable change in their chances of college attendance or on their level of depression, though the odds of suicidal ideation among this group did appear (surprisingly) to drop as a result of Wave 2 violence exposure. In general, though, it seems the damage to these students is already done. Taken together with the results of the first hypothesis, which show that early exposure is associated with dramatically worse outcome rates for college attendance, depression, and suicidality, these results challenge the idea of explaining the effects of violence with a straightforward dose-dependence model and suggest replacing it with something like a desensitization model. The unexpected finding for suicidality may even

indicate a parabolic trend: isolated exposures increase risk, but further exposures have a "toughening up" effect as students come to see themselves as survivors. This finding – that violence is bad for life chances and its effects accumulate but with diminishing severity – is consistent with other research on the effects of early childhood exposures (e.g. Boynton-Jarrett et al 2008). Clearly, early exposures are associated with circumstances that produce harmful life consequences, as evidenced by the results for Hypothesis 1. But it appears that not all violence exposures are equal. A single isolated exposure, as shown in the results for Hypotheses 2 and 3, may do more harm by itself than does any single instance within a pattern of repeated exposure.

These findings are consistent with the life course framing which initially animated this analysis. As predicted, young people's response to a trauma such as violence is contingent on a number of factors related to their past experience, in particular whether their exposure is isolated or part of a pattern of repeated exposures. These differential responses to life stressors fit with the pattern described by Aneshensel and colleagues (1991), who suggest that different personal and social circumstances can predict different kinds of responses to stressors. However, the results suggest a slight modification to the predictions derived from cumulative inequality theory: while it is clear that exposures add up to worse life circumstances overall, as revealed in the results from Hypotheses 1 and 2, it does not appear that the contributions of individual exposures to later outcomes are additive; rather than leading to less adaptive responses, it may be the case that those who suffer repeat victimization grow accustomed or desensitized over time.

One unexpected substantive result of this investigation is the suggestion that suicidality and depression respond differently to the stressor event of violence exposure within each of the subgroups. Among those with no history of past exposures, an isolated incident of violence exposure increases likelihood of suicidal ideation but does not affect depression score; among

those with a pattern of past exposures, a single exposure may slightly increase depression score and may even slightly decrease the odds of suicidal ideation. This leads us to ask: what accounts for the difference between the observed effects on these two outcomes? One possibility may be differences in what is considered "suicidal ideation." Suicidal thoughts are not the same as suicidal planning or attempt. Add Health asks subjects "During the past 12 months, have you ever seriously thought about committing suicide?", but the two violence exposure subgroups may interpret the specification "seriously thought" in different ways. For example, adjustment to a lifestyle more characterized by stress or learned helplessness (Peterson & Seligman 1983) may lead those who experience repeated violence exposure to overlook passive thinking about suicide and only acknowledge serious suicidal planning or attempt; whereas those who experience only isolated exposure may be more attuned to, and therefore treat as more serious, even passive self-destructive thoughts. This would bias the results and potentially obscure effects.

Another possibility is that the differences are due to differences in the etiologies of depression and suicidality. Various studies of suicidality among school-age populations have shown a complex developmental pathway, frequently including depression as a risk factor. For example, Thompson and colleagues (2005) identify hopelessness – itself a plausible outcome of violence exposure (Osofsky 1995) – as a direct risk factor in depression, but only as an indirect risk for suicidality, mediated by depressive symptoms. Yang and Clum (1994) draw a similar conclusion, and also find that perceived lack of social support – another possible secondary outcome of violence exposure, particularly chronic exposure – worsens depression directly and suicidality indirectly; the role of social support will be explored in more detail in the following chapter. Arria and colleagues (2009) found that while about 6% of first-year college students presented with suicidal ideation, only about 40% of those students met the standard criteria for

depression; and that while depression, alcohol use disorders, and social support predicted suicidal ideation, forms of interpersonal conflict (e.g. with parents) did not. The upshot seems to be that depression is much more responsive to situational influences, and therefore more susceptible to short-term change; suicidality, by contrast, is the outcome of a longer and more complex process, and is therefore more deep-seated and harder to move with any single environmental stimulus. As noted above, it may also be the case that repeat victims' self-concept changes; they may see themselves as "survivors" if they interpret their hardships as challenges which they have overcome, thus making suicidal ideation a less internally consistent position. In any case, further investigation into this point is warranted.

Finally, of course, it is possible that the findings here represent a Type II error. Page et al (2013)'s recent study of suicidal ideation prevalence in 49 countries using Global School-based Health Surveys suggests an overall prevalence of suicide ideation among school-age adolescents of 15.3%; only about 6.2% of overall retained respondents in this sample report suicidal ideation in the last year, and even in the high-risk subgroup the rate was only 7.3%. Crosby et al (2011) report that about 5.7% of American adults ages 18-29 had suicidal thoughts in 2008-09, but draw on data from 92,264 respondents to the National Survey on Drug Use and Health; here, although the Add Health sample ranges in age only between 18 and 25 (a riskier subset), we are limited to a much smaller sample. Because suicide ideation has a relatively low prevalence in this relatively small sample, we may lack the statistical power necessary to detect smaller effect sizes.

To be sure, this study is limited in several other respects. Importantly, although it follows violence exposure at several different points in time, the operationalization of violence exposure as a binary condition at each wave potentially glosses over differences in the severity of violence exposure at each time. The choice to measure violence as "exposed" or "unexposed" means that,

within each wave, someone who reports a single instance of witnessing violence is given the same score as someone who reports multiple incidents of direct victimization. However, it is reasonable to suspect – on the basis of common sense and existing evidence (e.g. Boynton-Jarrett et al 2008) – that more severe exposures are both rarer and more harmful, and these results still suggest that violence exposure of any sort is harmful overall.

Another inevitable limitation comes from the need to restrict observations to the region of common propensity score support. Excluding subjects from both ends of the propensity score distributions means that the subsamples analyzed may imperfectly represent the subpopulations to which I generalize. However, this is less of a concern if violence's effects are similar across the risk spectrum; since the results did not show stratum-specific patterns in either group, there is evidence that this may be the case. If anything, this limitation may impose a slight conservative bias on the effect size estimates: since many more observations were dropped from the bottom than from the top of the two distributions, and since the results suggest acute effects of violence exposure among those who are less likely to be exposed, the effects of violence exposure on those dropped may be even worse than on those who fall within the common support regions.

Despite these and other limitations, a number of policy suggestions present themselves on the basis of these findings. First, the evidence helps answer the earlier question of whether policy ought to focus on the intensive or the extensive margin of prevention – that is, whether to target students who are subject to large numbers of repeat victimizations or to seek broadly to minimize the share of youth who are exposed to violence overall. These results make it clear that early exposure to violence is associated with much poorer long-term outcomes than later exposure. Younger minds may be more susceptible to the corrosive effects of violence exposure than are more developed adolescents or young adults (cf. Sharkey 2010, Shonkoff et al 2012). We also

see a diminishing effect of exposures; later repeat exposures are not as damaging in themselves as are the initial shocks. In addition to the fact that isolated exposure to violence exerts acute effects on mental health and development trajectories, early exposure also may be problematic because it sets young people up for a persistent condition of repeated violence exposures. This suggests that policies designed to prevent early violence exposure to the greatest degree possible could mitigate negative outcomes. In other words, the results suggest a focus on the extensive rather than the intensive. The more we can prevent initial victimizations, it seems, the more good we will do. Of course, this is easier said than done; schools, however, are uniquely positioned institutions for effective violence prevention efforts (Gottfredson 2001) and may be able to get better leverage on this problem than other institutions such as neighborhoods and families; these results suggest that any reduction in the risk of exposure would be beneficial.

To be sure, more good could be done if schools and communities, whenever possible, also offer more substantial support for those who suffer chronic exposure or even aim interventions at mitigating the negative outcomes of initial violence exposure. Ideally, targeted interventions could both alleviate the harmful effects of acute exposure observed here, and also potentially prevent a single exposure from turning into a pattern of re-victimization. While additional exposures seem to produce diminishing effect sizes on the undesirable outcomes I examine, it remains the case that those who suffer repeated exposure manifest significantly worse results across the board in terms of depression, suicidality, educational attainment, and further patterns of violence exposure. If those students who are chronic violence victims can be identified early, it is possible that interventions could be tailored specifically to help them overcome these problems. However, in light of present findings, it seems that efforts to prevent violence exposure in the first place ought to be the priority.

CHAPTER FOUR

Victimization in Context:
Using Risk Profiles and School Efficacy to Understand School Violence Patterns

This chapter is primarily motivated by three practical questions: What kinds of students are likely to be exposed to violence, and how can we identify them? To what extent can school practices reduce students' likelihood of being exposed to violence? And does the possibility of such protection vary as a function of those student-level characteristics that put students at risk of exposure in the first place? Finding answers to these questions is complicated not only because, like the analyses in previous chapters, it requires a multilevel approach, but also because theory suggests that several heterogeneous features contribute to students' chances of victimization. The answers to these questions are important from a policy as well as a theory perspective: if violent victimization is concentrated among one kind of student, then one kind of intervention ought to suffice; but if heterogeneous types of students are victimized for different reasons, then educators and policymakers need to consider several intervention strategies. This chapter will investigate the existence and consequences of diverse risk factors which predict students' chances of violent victimization, and will additionally seek to determine whether the relationship between these factors and victimization risk changes across schools with different levels of supportive climate.

FACTORS PREDICTING VICTIMIZATION

Individual Behaviors and Network Characteristics

Criminology since at least the mid-20th century has addressed the problem known as the "victim-offender overlap" (von Hentig 1948): namely, that the victims of violence are often people who commit violence themselves. This pattern's consistent empirical robustness has led some (e.g. Lauritsen, Sampson & Laub 1991) to postulate that perpetration of and victimization

by violence are so intertwined that it may be inappropriate to treat them as separate domains. This conceptualization has consequences for the way that "violence exposure" is operationalized in school research, with many (e.g. Acosta, Albus, Reynolds, Spriggs & Weist 2001) arguing that witnessing, perpetrating, and being victimized by violence should all be considered "exposure."

Many argue that victim-offender overlap is driven by human ecology (Felson & Cohen 1980). Since the 1970s, the most fully developed account of this has come from routine activities theory. In this model, violence occurs when likely perpetrators encounter suitable targets in the absence of capable guardians (Cohen & Felson 1979), and victim-offender overlap happens because many potential perpetrators of violence follow behavioral patterns that also make them suitable victims – in particular, spending unsupervised time with other people who share similar normative views on violence (Berg et al 2012). Jensen and Brownfield (1986) have gone so far as to postulate that "criminal…routines are the most victimogenic of all routines" (87).

This leads me to postulate the first type of student likely to suffer violent victimization: students who habitually participate in violence themselves. Those who perpetrate violence, in general, tend to choose to spend time around others who also perpetrate violence, and are more likely to find themselves in situations where their own risk of victimization is high. These students, we may say, are at risk of violence exposure as a result of their *availability*.

A separate line of research calls attention to a separate but important type of victim: those who are targeted due to low social status or relative social isolation. Dan Olweus's pioneering work (e.g. Olweus 1993) makes clear that for bullying in particular, victims are selected on the basis of an imbalance in power which the victim experiences as favoring the perpetrating student (Olweus 2013). This power imbalance need not be physical; in many cases, social status and friendship group position is a crucial concern. Perpetrators choose victims who appear weak,

have few friends, or for some other reason seem less capable of offering resistance (Coloroso 2003). Relative social isolation, then, may act as a risk factor to the extent that it separates students from others who would stand up for them, defend them, support them, or otherwise present a deterrent. In the terms of routine activities theory above, these students are suitable targets who lack capable guardians. Contemporary work shows that many forms of nonphysical aggression are also organized by social hierarchy position (Faris 2012, Faris & Felmlee 2011).

Research linking social isolation to violence exposure is complex, largely because the effects of isolation are presumed to be a simultaneous function of the characteristics of the individual, the type of isolation they face, and the characteristics of those from whom they are isolated. To the extent that social isolation removes students from peers or adults who might act as capable guardians, it may increase risk of victimization (e.g. Spano & Nagy 2005). However, given that adolescents in general are a high-risk group for both offending and victimization (Lauritsen, Sampson & Laub 1991), social isolation may serve as a protective factor if it keeps individuals away from situations where they would be victimized. Moreover, different types of isolation – such as unwilling marginalization versus elective social disinterest – appear to be linked to different forms of violence exposure (Niño, Ignatow, & Cai 2016).

Schools, however, draw from diverse constituencies and require all students to spend long stretches of time around their cohort-mates. The fact that schools' populations are likely to have heterogeneous potential for violence – in the sense that they are composed of some students who may become perpetrators, and others who are not likely ever to be perpetrators but who might become suitable targets – plausibly attenuates the protective component of isolation. During school hours, students with few friends are not being kept away from potential tormentors; they merely lack access to peers who would protect them.

This compels researchers to pay attention to social network factors in attempting to explain victimization, and leads me to postulate a second type of likely victim: students who are socially isolated or considered low-status in their school social networks. These students may be at risk of violence exposure as a result of their *vulnerability*.

If this distinction is empirically verified, the policy implications become clear. Should we discover that victimization is isolated among those students who are also actively involved in perpetrating violence themselves, we could target them with a single kind of intervention. Should we find that only isolated students are at risk, it would suggest a very different but no less targeted kind of intervention strategy. If both groups are at risk, then different kinds of simultaneous interventions would be called for.

Institutional Characteristics

A sociological perspective urges attention to the school organizational context as well as the social network contexts of violent victimization. The processes that lead to school violence involve a complex interplay between schools' organizational functioning and students' behavior in school. School characteristics like organizational design (Rowan, Raudenbush, & Kang 1991), level of supervision and student social control policies (Devine 1996), teacher collective efficacy (Goddard 2001), and level of trust among staff and students or parents (Coleman 1987, Bryk & Schneider 2002) – as well as community (Crowder & South 2003; Newman, Fox, Harding, Mehta, & Roth 2004) and social/cultural context (Benbenishty & Astor 2005) – all plausibly influence the expression of violence and process of victim selection in the school setting. In short, the way the school is run has implications for students' chances of victimization.

The school context as such likely exacerbates both problems: the victim-offender overlap and the risky circumstances for isolated students. Schools, especially high schools, are

particularly vulnerable to the ecological circumstances that produce victim-offender overlap because they face the simple demographic problem that their job is to aggregate people who are at the peak age of offending (Gottfredson 2001). This is a challenging task in light of routine activities theory, which predicts that greater spatio-temporal co-occurrence of motivated offenders and suitable targets without capable guardians will lead to more victimization. Much evidence indicates that these are real risks. While it is true that with respect to some kinds of victimization – especially serious assaults and fatalities – schools are the safest place for students (Snyder, Dinkes, Kemp, & Baum 2009), students actually face elevated risks of nonfatal victimization while in school or on their way to or from school (Cook, Gottfredson, & Na 2010; Snyder & Sickmund 1995). This is especially true for property victimization (Dinkes et al 2009); students make especially suitable targets for other motivated offenders when they carry and display desirable objects, like electronics, designer sneakers or jackets, etc. Snyder & Sickmund (1995) find that as much as 56% of all juvenile victimizations occur in or around the school, and claim that "there is no comparable place where crimes against adults were so concentrated" (16). This problem is likely to be especially pronounced in schools that are less effective at providing effective supervision and guardianship (Gottfredson & DiPietro 2011).

In theory, schools could leverage routine activities to reduce victimization in a number of ways: by reducing potential offenders' motivation, by reducing potential targets' suitability, by providing more competent guardianship, or by preventing the co-occurrence of these three ingredients which are criminogenic when mixed. In practice, probably the second and third of these are the most viable options. The argument could be made that schools' developmental tasks include teaching students the kinds of interpersonal and moral skills which would reduce their inclination to perpetrate (e.g. Devine 1996, Lickona 1996), but this is a long-term strategy. In the

immediate term, schools may have more success by reducing their students' suitability as targets – for example, by banning electronics – and by improving the level of guardianship they provide. This latter strategy strongly implicates the importance of school climate.

A broad and rich research tradition across social science disciplines finds that supportive and caring school climates – ones in which students experience strong feelings of belonging and personal safety, enjoy caring relationships with peers and adults, and have limited exposure to risky peer behavior – can promote positive academic, social, emotional, and psychological outcomes among student populations (Brand, Felner, Shim, Seitsinger, & Dumas 2003; Thapa, Cohen, Guffey & Higgins-D'Alessandro 2013; Wilson 2004; Zullig, Koopman, Patton, & Ubbes 2010). Favorable school climate has been shown to exert protective effects on outcomes including psychological distress (Eccles, Midgley, Wigfield, Buchanan, Reuman, Flanagan, & Mac Iver 1993), psychosomatic health (Modin & Östberg 2009), risky and violent behavior (Khoury-Kassabri, Benbenishty & Astor 2005), and more.

Importantly, there is reason to believe that this effect will differ for different types of students. In particular, certain kinds of students whose personal characteristics would predict a relatively high risk of adverse school experiences may reap additional protective benefits from a safe, healthy, and supportive school environment. For example, children from military families are known to suffer disproportionately from certain mental health problems such as depression and suicidality as a result of stress related to parents' deployment or to frequent relocations (e.g. Gorman, Eide, & Hisle-Gorman, 2010). However, school climate factors such as support, inclusion, and caring have been shown to exert a particularly large protective effect on students with such backgrounds (e.g. Astor, De Pedro, Gilreath, Esqueda, & Benbenishty 2013; De Pedro, Astor, Gilreath, Benbenishty, & Berkowitz 2015). It also appears to be true that school climate

can partially mitigate the risk factors in students' out-of-school environments, such that in communities with high rates of violence, especially effective and supportive schools can be "atypically safe" environments (Astor, Benbenishty, & Estrada 2009).

This leads to a modification of the earlier predictions. Students who put themselves in risky positions – by perpetrating violence themselves and spending time with others who perpetrate – can be thought of as having "opted in" to violent situations, and therefore may be at risk regardless of school efficacy. However, for those who are put at risk by others rather than by any behavioral decisions of their own, improving supervision and guardianship may reduce the chances of victimization. Thus, I predict that positive school climate characteristics will reduce the risk of violence exposure for students who are at risk because of "vulnerability," but will have no such protective effect on students who are at risk because of "availability." Obviously this distinction has policy implications as well: if we discover that schools only have the leverage to help one kind of student, certain policy and intervention avenues will be foreclosed.

In light of all this, the chapter will focus on three main hypotheses: 1. Students who perpetrate violence are more likely to be victims (due to "availability"); 2. Socially isolated or low-status students are more likely to be victims (due to "vulnerability"); and 3. Strong social control and organizational functioning in schools reduces chances of victimization for "vulnerable" students, but not for "available" students. To preview the results, I find strong support for hypotheses 1 and 2 and qualified but sufficient support for hypothesis 3.

METHOD

The central questions of this study involve the simultaneous interplay of factors operating at two different levels. Specifically, because students are clustered together in schools, their chances of being exposed to violence are a function of both school-level and individual-level

characteristics, such that students in the same school have associated chances of victimization regardless of their individual traits. This violates the independence-of-units assumption, a key principle of standard regression modeling (Burstein 1980, Raudenbush & Bryk 1986) which requires that each subject's error term in a model be independent of every other subject's.

I solve this problem by using hierarchical models (Raudenbush & Bryk 2002) to estimate students' outcomes. In this case, because the outcome will be binary (victimized or not victimized by violence) I use hierarchical binary logistic regression, a form of hierarchical generalized linear models (HGLM). Students' odds of victimization may vary both within and across schools, so I use a 2-level HGLM with students at level 1 and schools at level 2 to predict the binary outcome at level 1 while using level-1 covariates to control for facts about the individual subjects and level-2 covariates to control for characteristics of the schools in which they are nested. The calculated coefficients give students' likelihood of victimization as a function of both level-1 and level-2 factors.

An additional problem is the methodological specification of temporal ordering. While I am not directly investigating causal effects here, the hypotheses all postulate the influence of some factors (perpetration, isolation, school safety) on victimization. Using cross-sectional data, it would not be possible to determine whether this conceptual ordering of events actually aligns with their empirical ordering. For example, cross-sectional data showing that students with fewer friends are more likely to be victimized would face a fundamental ambiguity: are these students being victimized because they are isolated, or does the experience of victimization lead to a sense of fear, anxiety, and social withdrawal? Likewise, if the data showed that students who perpetrate are more likely to be victimized, we would not know if they are being victimized because they are in risky situations or if they perpetrate in revenge for an earlier victimization.

In such cases, the solution is to use longitudinal data. Specifically, it makes sense here to look at two waves of measurement: a first wave, in which we capture individual behaviors and traits (in particular, perpetration of violence and social isolation) and school characteristics; and a second wave, in which we observe victimization by violence. By doing so, we ensure that the violence exposure in question followed, rather than preceded, the relevant predictors.

To sum up: I will use data from two waves of a longitudinal study to build cross-lagged 2-level HGLMs, where the Wave 2 outcome of violence victimization is predicted with Wave 1 individual- and school-level characteristics.

DATA

Answering questions that seek simultaneously to examine the role of individual and contextual factors requires the use of multi-level data. Since the context under investigation here is the school, a school-based survey – one which records individual-level data from students and aggregates them into the schools they attend, also recording information about the school itself – is most appropriate. Additionally, longitudinal data is needed to make sense of the ordering of relevant events and traits. For these reasons, I draw on data from the Public Use subsample of the National Longitudinal Study of Adolescent Health (Add Health). Add Health, one of the largest and most comprehensive surveys of adolescents ever, follows a nationally representative sample of adolescents in the United States. The study began during the 1994-95 school year with respondents in grades 7-12. A sample of 80 high schools and 52 middle schools from the US was chosen with unequal probability of selection, using systematic sampling methods and implicit stratification to ensure that the sample is representative of US schools with respect to region of country, urbanicity, school size, school type, and ethnicity. Study subjects were given in-school as well as in-home surveys; 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 study, I drew on individual and social network data from Add Health Waves 1 and 2. The Public Use subsample at Wave 1 includes information on 6504 individuals and 134 schools; 4834 of these students are measured again at Wave 2; I retained only those respondents who provided valid data at both waves. I reduced the sample further according to several criteria. First, I excluded any respondent who indicated during Wave 2 that he or she was not presently attending school, or had not attended during the reference year, for any reason; this eliminated virtually all students who were in Grade 12 at Wave 1, the remaining few of whom were dropped as well. I also restricted the sample to observations with non-missing values on primary variables of interest (described below). Because of low sampling density in some schools leading to inability to calculate certain network measures, I retained a final sample consisting of 2583 students in 113 schools. To reduce bias from differential probability of censoring, I also calculated weights based on inverse probability of retention at Wave 2 and used them to adjust estimates below as noted.

Key Variables

The primary outcome variable for this study is victimization by school violence at Wave 2. I focus on victimization – that is, directly inflicted physically violent action – rather than including more general notions of "exposure" which might include witnessing or even hearing about violence happening to others (e.g. Benbenishty & Astor 2005, Selner-O'Hagan et al 1998). This decision stems from the theories that inform the hypothesis, particularly routine activities theory (Cohen & Felson 1979). One of the key predictor variables for this analysis is perpetration of violence; we ought not to find it particularly interesting if students who commit violence also

see violence being committed (indeed, so long as they have their eyes open at the time, we can expect that they necessarily will). Our interest is in whether those same students will themselves be victims of violence. Likewise, we are less interested in knowing whether socially isolated students see or hear about violence being done to others than we are in knowing whether they are at greater risk of being targeted themselves.

In the Add Health In-Home survey at Wave 2, students are asked the following questions: "During the past 12 months, how often did each of the following things happen?: Someone pulled a knife or gun on you; Someone shot you; Someone cut or stabbed you; You got into a physical fight; You were jumped." Students were also asked "During the past 12 months, how many times were you in a physical fight in which you were injured and had to be treated by a doctor or nurse?" The answers to these questions were coded as "never," "once," or "more than once." While we should be interested in understanding the frequency of victimization, this operationalization makes counting specific numbers of occurrences impossible. We might also be interested in knowing the number of different kinds of violence to which a student had been exposed, but since it is not clear that the different exposures suggested here are commensurate in terms of their severity, it would be inappropriate to count them up as equivalent units. Therefore, in order to simplify analyses and avoid making dubious assumptions, this outcome is operationalized as binary, with students who indicated anything other than "never" to any of the questions getting coded as "1" and all other students coded "0".

All individual-level predictors are measured in Add Health Wave 1. The two key level-1 predictors are 1. self-reported perpetration of violence and 2. number of friendship nominations received from other students in one's school, commonly referred to as "in-degree" nominations. Perpetration was measured by subjects' responses to questions about their own violent behavior,

including “During the past 12 months, how often did each of the following things happen?: You pulled a knife or gun on someone; You shot or stabbed someone;” and “In the last 12 months, how often did you: Hurt someone badly enough to need bandages or care from a doctor or nurse?; Use or threaten to use a weapon to get something from someone?; Take part in a fight where a group of your friends was against another group?” Students who reported perpetrating any of the specific violent acts included in the questionnaire were coded with a value of “1” and students who reported no such behaviors were coded as “0”.

In-degree friendship nominations represent the number of times each student was named as a friend by another student in his/her school. This predictor is an important measure of relative social isolation, insofar as it represents how close other students feel to the student in question. It is also commonly used as a measure of student popularity, connectivity, and integration within networks (e.g. Faris 2012, Kreager 2007, Niño et al 2016). The school-clustered design of Add Health was implemented, in large part, to allow extensive investigation of friendship networks within schools. At Wave 1, students were asked to nominate up to 5 male and 5 female friends. Each of these nominations, or “sent ties,” links to the named student by a unique identification number, allowing the researchers to count the number of different times each student received a nomination from another student. The Add Health Wave 1 datasets come preloaded with such scores calculated for each student, along with a number of other network characteristics which are less relevant to this investigation. Schools in which fewer than 50 percent of students completed the questionnaire were dropped from the analysis to present more accurate depictions of schools’ social structures (Harris, Halpern, Whitsel, Hussey, Tabor, Entzel, & Udry 2009).

At the school level, we are primarily concerned with measuring overall sense of safety and school efficacy. A wealth of existing literature indicates that more effective schools – those

with higher levels of mutual trust among students and teachers (Bryk & Schneider 2002), with greater sense of shared norms for conduct (Coleman 1987, Gottfredson & DiPietro 2011), with better student ratings of subjective sense of safety (Benbenishty & Astor 2005), and with more efficacy among staff and faculty (Goddard 2001) – produce better results for student safety and violence prevention. This chapter's third hypothesis predicts that this will hold for students who are at risk of victimization as a function of their vulnerable social location (i.e. relative social isolation), but not those who are at risk because of their availability (i.e. their own participation in violent activities). For these reasons, the aspect of school efficacy that I focus on is students' average subjective feeling of safety in school. Add Health participants were asked to respond to the statement "You feel safe in your school" by indicating whether they strongly agree, agree, neither agree nor disagree, disagree, or strongly disagree. Students' responses were averaged within each school to produce a school-specific index score, with higher values indicating a lower average feeling of safety. This measure will be used as the primary level-2 covariate.

In addition to these key variables – the outcome measure of violent victimization, the individual-level predictor measures of violent perpetration and in-degree popularity, and the school-level predictor measure of average subjective sense of school safety – the analysis incorporates a number of other control variables at the individual level. Summary statistics for relevant variables are presented in Table 4.1. I account for a variety of student demographic characteristics, including sex (coded as an indicator variable for "female"), race (using whites as the reference category, since they are the numerical majority in the sample, and including indicator variables for "black" and "other"), and grade level (using 7^{th} grade as the reference category, operationalized with a vector of indicator variables for grades 8 through 11; 12^{th} graders at Wave 1 are excluded by Wave 2). In later models, I account for additional Wave 1

TABLE 4.1: Descriptive Statistics

Variable	Mean	S.D.	Min-Max
Level-1			
Victimization (outcome)	0.158	0.366	0 – 1
Perpetration	0.303	0.459	0 – 1
Friendship nominations	4.816	3.815	0 – 30
Female	0.538	0.499	0 – 1
White	0.668	0.472	0 – 1
Black	0.237	0.427	0 – 1
Other race	0.095	0.294	0 – 1
7^{th} grade	0.199	0.399	0 – 1
8^{th} grade	0.215	0.412	0 – 1
9^{th} grade	0.196	0.397	0 – 1
10^{th} grade	0.200	0.398	0 – 1
11^{th} grade	0.177	0.382	0 – 1
Ever suspended	0.225	0.419	0 – 1
Approximate GPA	2.890	0.723	1 – 4
Self-esteem index*	0	1	-1.557 – 4.340
Protective Factors index**	0	1	-3.984 – 1.853
Neighborhood risk index†	0	1	-2.130 – 1.463
Feeling disliked††	0.336	0.336	0 – 1
Sexually active	0.299	0.295	0 – 1
Marijuana use	0.298	0.289	0 – 1
Level-2			
Safe at school***	2.158	0.350	1.351 – 2.857

* "Self-esteem index" is calculated from students' answers to a battery of questions from Add Health's Section 10, "Feelings Scale," that related to self-image; raw values ranging from 0 to 28 were standardized as z-scores, with higher scores indicating more positive self-concept.
** "Protective Factors index" is calculated from students' answers to questions from Add Health's Section 35, "Protective Factors." Answers ranged from 1 = "not at all" to 5 = "very much." Answers were reverse coded as necessary summed such that higher scores indicate higher level of protective influence, and raw values were standardized as z-scores.
† "Neighborhood risk index" is calculated from students' answers to questions from Add Health's Section 36, "Neighborhood." Higher numbers indicate less positive feeling about the neighborhood; raw values were standardized as z-scores.
†† "Feeling disliked" is calculated from students' answer to the question "How often was … the following … true during the past week? You felt that people disliked you." Answers were coded such that 0 = "never or rarely" or "sometimes" and 1 = "a lot of the time" or "most of the time or all of the time."
*** "Safe at school" is a school-level average of students' agreement with the statement "You feel safe in your school" from 1 = "strongly agree" to 5 = "strongly disagree"; higher scores indicate lower average feelings of safety.

individual-level protective or risk factors for violence exposure, including approximate GPA (calculated on the basis of students' responses to questions about their most recent grades earned in English, mathematics, science, and social studies or history classes); an index of protective factors offered on the Add Health questionnaire (including factors related to relationship with family, friends, teachers, neighbors, and peers); an index of neighborhood risk characteristics (captured in Section 36, "Neighborhood", of the Add Health questionnaire), an index of self-esteem related questions included in Add Health; students' self-reports of ever being suspended from school; students' self-reports of feeling disliked and rejected by others; and students' self-reports of engaging in other risky behaviors such as early sexual initiation and drug use.

I proceed by estimating a series of HGLM models with different specifications. I begin by estimating two different versions of a naïve model, Models 1 and 2. Each of these models estimates the outcome – Wave 2 violence victimization – using only the two key predictors (Wave 1 perpetration and in-degree popularity) at level 1, but I differentiate Model 2 from the previous by changing how the key variables are allowed to vary at level-2 and by introducing level-2 covariates to the model. I additionally add level-1 demographic variables (sex, race, and grade level) in Model 3, and I deploy the full range of level-1 covariates in Model 4.

RESULTS

Naïve models

Model 1, the purely naïve model, predicts the likelihood of violent victimization using only perpetration and popularity as covariates, and allows only the intercept from level 1 to vary at level 2. The equations for Model 1 are thus:

Level-1 Model

$$\text{Prob}(Y_{ij}=1|\beta_j) = \phi_{ij}$$
$$\log[\phi_{ij}/(1 - \phi_{ij})] = \eta_{ij}$$
$$\eta_{ij} = \beta_{0j} + \beta_{1j}*(\text{perpetration})_{ij} + \beta_{2j}*(\text{in-degree})_{ij}$$

Level-2 Model

$\beta_{0j} = \gamma_{00} + u_{0j}$

$\beta_{1j} = \gamma_{10}$

$\beta_{2j} = \gamma_{20}$

Mixed Model

$\eta_{ij} = \gamma_{00} + \gamma_{10}$*(perpetration)$_{ij} + \gamma_{20}$*(in-degree)$_{ij} + u_{0j}$

where Y, the outcome, is the indicator for students' self-reports of being victimized by violence at Wave 2; "in-degree" is a student's in-degree friendship score at Wave 1, and "perpetration" represents students' self-reports of perpetrating violence at Wave 1. This model specifies that students' likelihood of victimization within their school contexts varies as a function of their own violent behavior and the number of people who nominate them as a friend, and that schools vary randomly in the average rates at which their students are victimized by violence.

Results from Model 1 are presented in Table 4.2. The key findings relate to the estimated associations for "perpetration," γ_{10}, and "in-degree," γ_{20}. The results from this preliminary model strongly support hypotheses 1 and 2. First, and most disturbing, is the size of the association between perpetrating violence and being victimized by violence. The odds ratio for γ_{10} is 4.696, suggesting that students who report perpetrating violence themselves at Wave 1 are more than four and a half times more likely to be victimized by violence at Wave 2 than are students who do not perpetrate violence (γ_{10} = 1.547, OR = 4.696, 95% CI = 3.691–5.975, p<0.001). This is a huge, and hugely significant, association. Importantly, it provides support for hypothesis 1 and is consistent with the victim-offender overlap theories from which hypothesis 1 is derived. Second, the odds ratio for γ_{20}, the coefficient of the variable representing the number in-degree friendship nominations, is 0.954 (γ_{20} = -0.047, OR = 0.954, 95% CI = 0.928–0.980, p<0.001); this suggests that for each additional person in a school who nominates a student as a friend at Wave 1, the nominated student's odds of being victimized by violence at Wave 2 decline by about 4.6%. This provides support for hypothesis 2, and comports with theories (e.g. Olweus 1993, Spano & Nagy

2005) that social isolation increases students' likelihood of victimization. These results clearly suggest that the more friends a student has – that is, the more other students who nominate the target student when asked to name their friends – the less likely that student is to be victimized.

TABLE 4.2: Predicted Log Odds of Wave 2 Victimization, Results from Model 1 (Unit-specific model with robust standard errors)

Fixed Effect	Coefficient	SE	OR	95% CI	Appx. *d.f.*
For Intercept-1, β_0					
Intercept-2, γ_{00}	-2.116***	0.114	0.121	0.096 – 0.151	112
For perpetration slope, β_1					
Intercept-2, γ_{10}	1.547***	0.123	4.696	3.691 – 5.975	2440
For in-degree slope, β_2					
Intercept-2, γ_{20}	-0.047***	0.014	0.954	0.928 – 0.980	2440

Note: ˜ = p<0.1, * = p<0.05, ** = p<0.01, *** = p<0.001

Model 2 expands on Model 1 by allowing the coefficients for perpetration and in-degree friendship nomination to vary across schools, and by introducing for each of these two main effects a level-2 predictor for schools' average level of students' agreement or disagreement with the claim that they feel safe in school. This latter operationalization allows us to test hypothesis 3 by seeing whether the effect sizes of perpetration and number of friends on victimization vary from school to school as a function of school-level efficacy in terms of providing a safe and supportive environment. Thus, the equations for Model 2 are:

Level-1 Model

$\text{Prob}(Y_{ij}=1|\beta_j) = \phi_{ij}$

$\log[\phi_{ij}/(1 - \phi_{ij})] = \eta_{ij}$

$\eta_{ij} = \beta_{0j} + \beta_{1j}*(\text{perpetration}_{ij}) + \beta_{2j}*(\text{in-degree}_{ij})$

Level-2 Model

$\beta_{0j} = \gamma_{00} + u_{0j}$

$\beta_{1j} = \gamma_{10} + \gamma_{11}*(\text{school-safe}_j) + u_{1j}$

$\beta_{2j} = \gamma_{20} + \gamma_{21}*(\text{school-safe}_j) + u_{2j}$

Mixed Model

$$\eta_{ij} = \gamma_{00} + \gamma_{10}*\text{perpetration}_{ij} + \gamma_{11}*\text{school-safe}_j*\text{perpetration}_{ij} + \gamma_{20}*\text{in-degree}_{ij} + \gamma_{21}*\text{school-safe}_j*\text{in-degree}_{ij} + u_{0j} + u_{1j}*\text{perpetration}_{ij} + u_{2j}*\text{in-degree}_{ij}$$

where Y, "perpetration", and "in-degree" are the same as specified above; and "school-safe" is a grand-mean-centered school-level average of students' agreement with the statement "You feel safe in your school," from 1 = "strongly agree" to 5 = "strongly disagree" so that higher scores indicate lower average feelings of safety.

Results from Model 2 are presented in Table 4.3. The school-level coefficients for perpetration and in-degree friendship nominations tell essentially the same story as they did in Model 1. The odds ratio for γ_{10}, the intercept for the coefficient for students' own violence perpetration, remains virtually constant at 4.659 (γ_{10} = 1.539, OR = 4.659, 95% CI = 3.667–5.918, $p<0.001$), again suggesting a large increase in students' risk of Wave 2 victimization associated with their own Wave 1 participation in violent behavior. Likewise, the impact of γ_{20}, the intercept for the coefficient for students' number of in-degree friendship nominations, increases slightly to an odds ratio of 0.951 (γ_{20} = -0.050, OR = 0.951, 95% CI = 0.922–0.981, $p<0.01$), suggesting that each additional Wave 1 friendship nomination is associated with a 4.9% decrease in a student's Wave 2 odds of being victimized. These results, as before, respectively support hypotheses 1 and 2. The additional results for the effects of school efficacy additionally provide strong support for hypothesis 3. As average subjective level of disagreement with the statement "You feel safe at school" increases, there is no meaningful associated change in the level-2 slope for effect of perpetration on victimization (γ_{11} = -0.189, OR = 0.828, 95% CI = 0.470–1.458, $p>0.05$); thus, the extent to which students' victimization is associated with their own participation in violent behavior does not vary as a function of how effective schools are at providing a safe and supportive environment. By contrast, γ_{21}, the level-2 slope for the effect of

in-degree nominations on victimization, shows a meaningful increase as the average subjective level of disagreement with the statement "You feel safe at school" increases (γ_{21} = 0.130, OR = 1.139, 95% CI = 1.045–1.242, p<0.01). Thus, in schools that provide less safe and supportive environments, number of friendships offers less effective protection against victimization, and in schools with greater average sense of relative safety, having more friends is a more effective preventive against violence; the risk of victimization associated with one's own violent behavior, though, is invariant as a function of school safety and positive climate.

TABLE 4.3: Predicted Log Odds of Wave 2 Victimization, Results from Model 2 (Unit-specific model with robust standard errors)

Fixed Effect	Coefficient	SE	OR	95% CI	Appx. *d.f.*
For Intercept-1, β_0					
Intercept-2, γ_{00}	-2.098***	0.114	0.123	0.098 – 0.154	112
For perpetration slope, β_1					
Intercept-2, γ_{10}	1.539***	0.121	4.659	3.667 – 5.918	111
Safe at school, γ_{11}	-0.189	0.286	0.828	0.470 – 1.458	111
For in-degree slope, β_2					
Intercept-2, γ_{20}	-0.050**	0.016	0.951	0.922 – 0.981	111
Safe at school, γ_{21}	0.130**	0.044	1.139	1.045 – 1.242	111

Note: ~ = p<0.1, * = p<0.05, ** = p<0.01, *** = p<0.001. "Safe at school" is a school-level average of students' agreement with the statement "You feel safe in your school," from 1 = "strongly agree" to 5 = "strongly disagree"; higher scores indicate lower average feelings of safety.

Summarizing the naïve model results: "available" students who commit violence at Wave 1 are more likely to be victimized at Wave 2 (supporting hypothesis 1) and "vulnerable" students who have fewer friendship nominations at Wave 1 are more likely to be victimized at Wave 2 (supporting hypothesis 2); as well, favorable school climate is protective for "vulnerable" students but provides no such protection for "available" students (supporting hypothesis 3).

Demographic model

Model 3 maintains the operationalization of the key variables from Model 2, but introduces a set of demographic variables as additional controls. The equations for Model 3 are:

Level-1 Model

$\text{Prob}(Y_{ij}=1|\beta_j) = \phi_{ij}$

$\log[\phi_{ij}/(1 - \phi_{ij})] = \eta_{ij}$

$\eta_{ij} = \beta_{0j} + \beta_{1j}*(\text{perpetration}_{ij}) + \beta_{2j}*(\text{in-degree}) + \beta_{3j}*(\text{female}_{ij}) + \beta_{4j}*(\text{black}_{ij}) + \beta_{5j}*(\text{other}_{ij}) + \beta_{6j}*(\text{grade8}_{ij}) + \beta_{7j}*(\text{grade9}_{ij}) + \beta_{8j}*(\text{grade10}_{ij}) + \beta_{9j}*(\text{grade11}_{ij})$

Level-2 Model

$\beta_{0j} = \gamma_{00} + u_{0j}$

$\beta_{1j} = \gamma_{10} + \gamma_{11}*(\text{school-safe}_j) + u_{1j}$

$\beta_{2j} = \gamma_{20} + \gamma_{21}*(\text{school-safe}_j) + u_{2j}$

$\beta_{3j} = \gamma_{30}$

$\beta_{4j} = \gamma_{40}$

$\beta_{5j} = \gamma_{50}$

$\beta_{6j} = \gamma_{60}$

$\beta_{7j} = \gamma_{70}$

$\beta_{8j} = \gamma_{80}$

$\beta_{9j} = \gamma_{90}$

Mixed Model

$\eta_{ij} = \gamma_{00} + \gamma_{10}*\text{perpetration}_j + \gamma_{11}*\text{school-safe}_j*\text{perpetration}_{ij} + \gamma_{20}*\text{in-degree}_{ij} + \gamma_{21}*\text{school-safe}_j*\text{in-degree}_{ij} + \gamma_{30}*\text{female}_{ij} + \gamma_{40}*\text{black}_{ij} + \gamma_{50}*\text{other}_{ij} + \gamma_{60}*\text{grade8}_{ij} + \gamma_{70}*\text{grade9}_{ij} + \gamma_{80}*\text{grade10}_{ij} + \gamma_{90}*\text{grade11}_{ij} + u_{0j} + u_{1j}*\text{perpetration}_{ij} + u_{2j}*\text{in-degree}_{ij}$

where Y, "perpetration", "in-degree", and "school-safe" are the same as specified above; "female" is an indicator for female sex; "black" and "other" are indicators for self-identified racial group (with "white" as the reference category), and "grade8" through "grade11" are indicators for students' grade in school as of Wave 1 (with 7th grade as the reference category).

Results from Model 3 are summarized in Table 4.4. Although the estimated impacts of the two key predictors both decline slightly, the picture presented by these results is largely the same as in the previous model. In particular, the odds ratio for γ_{10}, the intercept for the coefficient for violence perpetration, drops slightly but remains high at 4.264, suggesting again a hugely elevated risk of Wave 2 violent victimization associated with students' own participation

in violent behavior at Wave 1 (γ_{10} = 1.446, OR = 4.246, 95% CI = 3.305–5.454, p<0.001). The estimate for γ_{11}, the level-2 slope for how the effect of perpetration on victimization changes in response to school efficacy, remains substantively and statistically insignificant (γ_{11} = -0.279, OR = 0.757, 95% CI = 0.432–1.326, p>0.05), reinforcing the previous finding that the association between violent perpetration and violent victimization does not vary as a function of school safety. The odds ratio for γ_{20}, the intercept for the coefficient for friendship nominations, moves only slightly to 0.958 (γ_{20} = -0.043, OR = 0.958, 95% CI = 0.929–0.988, p<0.01), suggesting again that having more Wave 1 friendship nominations is associated with lower risk of Wave 2 victimization net of other factors. And the odds ratio for γ_{21}, the level-2 slope for how the effect of in-degree popularity on victimization changes in response to school efficacy, is virtually unchanged at 1.138 (γ_{21} = 0.130, OR = 1.138, 95% CI = 1.045–1.240, p<0.01), reinforcing the prediction from Hypothesis 3 that the protective influence of having more friendship nominations is magnified in schools with safer climates.

The effects of the demographic variables, though not directly relevant to the hypotheses, are nevertheless worthy of explanation. The results suggest that, all else constant, girls' odds of victimization are approximately half of boys' (γ_{30} = -0.629, OR = 0.533, 95% CI = 0.423–0.673, p<0.001); black students and other non-white students face higher odds of victimization than do white students, though the difference is significant only for black students (γ_{40} = 0.365, OR = 1.441, 95% CI = 1.117–1.857, p<0.01 ; γ_{50} = 0.322, OR = 1.380, 95% CI = 0.878–2.170, p>0.05); and students in 7th grade appear to have a medium risk of victimization relative to other grades, with 8th and 9th graders showing slightly higher risk and 10th and 11th graders showing slightly lower – though given small effect sizes and low significance, reading too much into this pattern may be a mistake.

TABLE 4.4: Predicted Log Odds of Wave 2 Victimization, Results from Model 3 (Unit-specific model with robust standard errors)

Fixed Effect	Coefficient	SE	OR	95% CI	Appx. *d.f.*
For Intercept-1, β_0					
Intercept-2, γ_{00}	-1.925***	0.156	0.146	0.107 – 0.199	112
For perpetration slope, β_1					
Intercept-2, γ_{10}	1.446***	0.126	4.246	3.305 – 5.454	111
Safe at school, γ_{11}	-0.279	0.283	0.757	0.432 – 1.326	111
For in-degree slope, β_2					
Intercept-2, γ_{20}	-0.043**	0.016	0.958	0.929 – 0.988	111
Safe at school, γ_{21}	0.130**	0.043	1.138	1.045 – 1.240	111
For female slope, β_3					
Intercept-2, γ_{30}	-0.629***	0.118	0.533	0.423 – 0.673	2208
For black slope, β_4					
Intercept-2, γ_{40}	0.365**	0.130	1.441	1.117 – 1.857	2208
For other race slope, β_5					
Intercept-2, γ_{50}	0.322	0.231	1.380	0.878 – 2.170	2208
For 8th grade slope, β_6					
Intercept-2, γ_{60}	0.257~	0.143	1.294	0.977 – 1.712	2208
For 9th grade slope, β_7					
Intercept-2, γ_{70}	0.039	0.182	1.040	0.728 – 1.486	2208
For 10th grade slope, β_8					
Intercept-2, γ_{80}	-0.145	0.195	0.865	0.590 – 1.268	2208
For 11th grade slope, β_9					
Intercept-2, γ_{90}	-0.353~	0.195	0.703	0.480 – 1.030	2208

Note: ~ = p<0.1, * = p<0.05, ** = p<0.01, *** = p<0.001. "Safe at school" is a school-level average of students' agreement with the statement "You feel safe in your school," from 1 = "strongly agree" to 5 = "strongly disagree"; high scores indicate lower average feelings of safety.

The critical conclusions here reaffirm the findings from the previous models with respect to our three hypotheses. Net of other factors, Wave 1 violent perpetration tremendously increases the risk of Wave 2 victimization (supporting Hypothesis 1), and the size of this association does not vary as a function of school safety (supporting Hypothesis 3); having more friendship nominations at Wave 1 is associated with lower odds of victimization at Wave 2 (supporting

Hypothesis 2), and this effect is more pronounced in schools with greater average subjective sense of safety (also supporting Hypothesis 3).

Full model

Model 4, the full model, maintains the operationalization of the key explanatory and demographic variables from Model 3, and introduces a set of variables for several individual-level risk and protective factors. The equations for Model 4 are:

Level-1 Model

$\text{Prob}(VICTIM1_{ij}=1|\beta_j) = \phi_{ij}$

$\log[\phi_{ij}/(1 - \phi_{ij})] = \eta_{ij}$

$\eta_{ij} = \beta_{0j} + \beta_{1j}*(\text{perpetration}_{ij}) + \beta_{2j}*(\text{in-degree}_{ij}) + \beta_{3j}*(\text{female}_{ij}) + \beta_{4j}*(\text{black}_{ij}) + \beta_{5j}*(\text{other}_{ij}) + \beta_{6j}*(\text{grade8}_{ij}) + \beta_{7j}*(\text{grade9}_{ij}) + \beta_{8j}*(\text{grade10}_{ij}) + \beta_{9j}*(\text{grade11}_{ij}) + \beta_{10j}*(\text{suspended}_{ij}) + \beta_{11j}*(\text{appxGPA}_{ij}) + \beta_{12j}*(\text{self-esteem}_{ij}) + \beta_{13j}*(\text{PF-index}_{ij}) + \beta_{14j}*(\text{neighborhood}_{ij}) + \beta_{15j}*(\text{disliked}_{ij}) + \beta_{16j}*(\text{ever-sex}_{ij}) + \beta_{17j}*(\text{marijuana}_{ij})$

Level-2 Model

$\beta_{0j} = \gamma_{00} + u_{0j}$

$\beta_{1j} = \gamma_{10} + \gamma_{11}*(\text{school-safe}_j) + u_{1j}$

$\beta_{2j} = \gamma_{20} + \gamma_{21}*(\text{school-safe}_j) + u_{2j}$

$\beta_{3j} = \gamma_{30}$

$\beta_{4j} = \gamma_{40}$

$\beta_{5j} = \gamma_{50}$

$\beta_{6j} = \gamma_{60}$

$\beta_{7j} = \gamma_{70}$

$\beta_{8j} = \gamma_{80}$

$\beta_{9j} = \gamma_{90}$

$\beta_{10j} = \gamma_{10\,0}$

$\beta_{11j} = \gamma_{11\,0}$

$\beta_{12j} = \gamma_{12\,0}$

$\beta_{13j} = \gamma_{13\,0}$

$\beta_{14j} = \gamma_{14\,0}$

$\beta_{15j} = \gamma_{15\,0}$

$\beta_{16j} = \gamma_{16\,0}$

$\beta_{17j} = \gamma_{17\,0}$

Mixed Model

$\eta_{ij} = \gamma_{00} + \gamma_{10}*\text{perpetration}_j + \gamma_{11}*\text{school-safe}_j*\text{perpetration}_{ij} + \gamma_{20}*\text{in-degree}_{ij} + \gamma_{21}*\text{school-safe}_j*\text{in-degree}_{ij} + \gamma_{30}*\text{female}_{ij} + \gamma_{40}*\text{black}_{ij} + \gamma_{50}*\text{other}_{ij} + \gamma_{60}*\text{grade8}_{ij} + \gamma_{70}*\text{grade9}_{ij} + \gamma_{80}*\text{grade10}_{ij} + \gamma_{90}*\text{grade11}_{ij} + \gamma_{10\,0}*\text{suspended}_{ij} + \gamma_{11\,0}*\text{appxGPA}_{ij} + \gamma_{12\,0}*\text{self-esteem}_{ij} + \gamma_{13\,0}*\text{PF-index}_{ij} + \gamma_{14\,0}*\text{neighborhood}_{ij} + \gamma_{15\,0}*\text{disliked}_{ij} + \gamma_{16\,0}*\text{ever-sex}_{ij} + \gamma_{17\,0}*\text{marijuana}_{ij} + u_{0j} + u_{1j}*\text{perpetration}_{ij} + u_{2j}*\text{in-degree}_{ij}$

where Y, “perpetration”, “in-degree”, “school-safe”, “female”, “black”, “other”, and “grade8” through “grade11” are the same as specified above; “suspended” is an indicator variable for whether the student reports ever having been suspended from school; “appxGPA” is a measure of students’ most recent GPA, calculated by taking the average of students’ responses to questions about their most recent grades earned in English, mathematics, science, and social studies or history classes; “self-esteem” captures students’ answers to a battery of questions from Add Health about their self-concept, standardized to a z score; “PF-index” is a composite score calculated based on students’ answers to a battery of questions about protective factors (such as how much they feel like adults care about them, how well they get along with their families, and more), where higher scores indicate greater levels of protective influence, standardized to a z-score; “neighborhood” captures students’ responses to several questions about their sense of belonging and safety in their neighborhoods, standardized to a z-score; “disliked” represents students’ responses to a question about whether they feel disliked and rejected by others; “ever-sex” is an indicator for whether students report that they are sexually active; and “marijuana” is an indicator for students’ self-reports of whether they or any of their best friends use marijuana.

Results from Model 4 are presented in Table 4.5. The substantive results for the key variables are largely similar to what was found in previous models, with a few key differences. First, it is again apparent that perpetration of violence is associated with a tremendous elevation in the risk of violent victimization, and that the size of this association does not depend on school efficacy. The estimate for γ_{10}, the intercept for the coefficient for violence perpetration, drops somewhat from the previous estimate, but remains very high (γ_{10} = 1.054, OR = 2.870, 95% CI = 2.190–3.762, p<0.001). The estimate for γ_{11}, the level-2 slope for how the effect of perpetration on victimization changes with higher school efficacy, is actually slightly negative (γ_{11} = -0.519,

OR = 0.595, 95% CI = 0.329–1.076, p<0.1), suggesting that safer school may be associated with an increase in the risk of Wave 2 victimization associated with Wave 1 perpetration, but the estimate only achieves marginal statistical significance. The link between friendship nominations and victimization, however, remains substantively identical. The odds ratio for γ_{20}, the intercept for the coefficient for friendship nominations, changes only slightly to 0.967 (γ_{20} = -0.034, OR = 0.967, 95% CI = 0.938–0.997, p<0.05), still suggesting that each additional Wave 1 friendship nomination is associated with a 3.3% lower risk of Wave 2 victimization. And γ_{21}, the rate at which the association between friendship nominations and victimization changes as a function of subjective school safety, drops only slightly from the previous model (γ_{21} = 0.105, OR = 1.111, 95% CI = 1.026–1.203, p<0.01), meaning that safer schools mitigate the odds of violence victimization associated with student social isolation. The results, then, fully support hypotheses 1 and 2, and provide qualified support for hypothesis 3.

The other factors in the model offer interesting results as well. As in Model 3, female students have significantly lower odds of victimization than male students (γ_{30} = -0.568, OR = 0.566, 95% CI = 0.434–0.739, p<0.001) and nonwhite students have higher odds of victimization than white students, though the differences are not significant. The differences between grade levels also persist, with those who were in 8th grade at Wave 1 showing slightly higher odds of Wave 2 victimization than those who were in 7th grade, and all other grades showing slightly lower odds than 7th graders. Additionally, most of the added level-1 risk and protective factors introduced in Model 4 behave as expected. For the Wave 1 risk factors, ever having been suspended from school ($\gamma_{10\,0}$ = 0.408, OR = 1.504, 95% CI = 1.186–1.908, p<0.001), reported feeling disliked by peers ($\gamma_{15\,0}$ = 0.271, OR = 1.311, 95% CI = 1.101–1.561, p<0.01), being sexually active ($\gamma_{16\,0}$ = 0.473, OR = 1.605, 95% CI = 1.236–2.086, p<0.001), and using or having

TABLE 4.5: Predicted Log Odds of Wave 2 Victimization, Results from Model 4 (Unit-specific model with robust standard errors)

Fixed Effect	Coefficient	SE	OR	95% CI	Appx. *d.f.*
For Intercept-1, β_0					
Intercept-2, γ_{00}	-2.097***	0.163	0.123	0.089 – 0.170	112
For perpetration slope, β_1					
Intercept-2, γ_{10}	1.054***	0.137	2.870	2.190 – 3.762	111
Safe at school, γ_{11}	-0.519~	0.299	0.595	0.329 – 1.076	111
For in-degree slope, β_2					
Intercept-2, γ_{20}	-0.034*	0.015	0.967	0.938 – 0.997	111
Safe at school, γ_{21}	0.105**	0.040	1.111	1.026 – 1.203	111
For female slope, β_3					
Intercept-2, γ_{30}	-0.568***	0.135	0.566	0.434 – 0.739	2201
For black slope, β_4					
Intercept-2, γ_{40}	0.143	0.140	1.153	0.876 – 1.519	2201
For other race slope, β_5					
Intercept-2, γ_{50}	0.313	0.224	1.367	0.881 – 2.122	2201
For 8th grade slope, β_6					
Intercept-2, γ_{60}	0.077	0.144	1.080	0.814 – 1.433	2201
For 9th grade slope, β_7					
Intercept-2, γ_{70}	-0.340~	0.200	0.712	0.481 – 1.055	2201
For 10th grade slope, β_8					
Intercept-2, γ_{80}	-0.623**	0.211	0.537	0.354 – 0.812	2201
For 11th grade slope, β_9					
Intercept-2, γ_{90}	-0.860***	0.223	0.423	0.273 – 0.656	2201
For suspended slope, β_{10}					
Intercept-2, $\gamma_{10\,0}$	0.408***	0.121	1.504	1.186 – 1.908	2201
For appx. GPA slope, β_{11}					
Intercept-2, $\gamma_{11\,0}$	-0.242**	0.090	0.785	0.658 – 0.936	2201
For self-esteem index slope, β_{12}					
Intercept-2, $\gamma_{12\,0}$	-0.027	0.018	0.974	0.940 – 1.009	2201
For protective factors index slope, β_{13}					
Intercept-2, $\gamma_{13\,0}$	-0.265***	0.075	0.767	0.663 – 0.888	2201
For neighborhood index slope, β_{14}					
Intercept-2, $\gamma_{14\,0}$	0.090~	0.053	1.095	0.987 – 1.214	2201

Table 4.5 continues below

Table 4.5 continued

For feeling disliked slope, β_{15}					
Intercept-2, $\gamma_{15\,0}$	0.271**	0.089	1.311	1.101 – 1.561	2201
For sexually active slope, β_{16}					
Intercept-2, $\gamma_{16\,0}$	0.473***	0.133	1.605	1.236 – 2.086	2201
For marijuana use slope, β_{17}					
Intercept-2, $\gamma_{17\,0}$	0.244***	0.055	1.277	1.147 – 1.422	2201

Note: ˜ = p<0.1, * = p<0.05, ** = p<0.01, *** = p<0.001. "Safe at school" is a school-level average of students' agreement with the statement "You feel safe in your school," from 1 = "strongly agree" to 5 = "strongly disagree"; high scores indicate lower average feelings of safety.

friends who use marijuana ($\gamma_{17\,0}$ = 0.244, OR = 1.277, 95% CI = 1.147–1.422, p<0.001) are all associated with a significantly elevated risk of Wave 2 victimization. For the Wave 1 protective factors, higher approximate GPA ($\gamma_{11\,0}$ = -0.242, OR = 0.785, 95% CI = 0.658–0.936, p<0.01) and higher scores on the protective factors index ($\gamma_{13\,0}$ = -0.265, OR = 0.767, 95% CI = 0.663–0.888, p<0.001) are associated with reductions in risk of Wave 2 victimization; self-esteem index score is also associated with a reduction in risk, but the effect is not statistically significant ($\gamma_{12\,0}$ = -0.027, OR = 0.974, 95% CI = 0.940–1.009, p>0.05), and neighborhood protective factors appear to be associated with a marginally significant increase in risk ($\gamma_{14\,0}$ = 0.090, OR = 1.095, 95% CI = 0.987–1.214, p<0.1), which is difficult to explain substantively.

DISCUSSION & CONCLUSION

Overall, the results obtained from these analyses accord with predictions from the chapter's three main hypotheses, with full support for hypotheses 1 and 2 and qualified support for hypothesis 3. The clearest results are obtained regarding hypotheses 1 and 2, which are strongly upheld in all models. Students who perpetrate violence at Wave 1 are overwhelmingly more likely to become victims of violence at Wave 2, with estimated odds ratios for the increase in risk ranging from 4.696 to 2.870. These estimates are highly significant across all model

specifications. Clearly, the victim-offender overlap is alive and well in these results. This affirms the predictions from routine activities theory outlined earlier. Students whose own activities put them in violent situations – activities such as perpetrating violence or spending time with violent peers – are eventually more likely to become victims themselves. In short, "availability" is a major source of risk.

The results also broadly support hypothesis 2. Across all models, the association between number of friendship nominations and risk of victimization persists and runs in the direction predicted. The largest estimated effect is in Model 2, where each Wave 1 friendship nomination is associated with a 4.6% reduction in the risk of Wave 2 victimization. The smallest effect is in Model 4, where the estimated risk reduction for each friendship nomination is only about 3.3%. Substantively, these results tell a consistent story that supports hypothesis 2 – students with more friends are at lower risk of victimization, and socially isolated "vulnerable" students are at higher risk. This also affirms the routine activities theory prediction, as well as from Olweus (1993, 2013) and others, that social isolation is a risk factor for victimization – either because, in Cohen and Felson's (1979) terms, these are students who are more "suitable" targets, or because they are targets who lack "capable guardians."

The conclusions from the model estimates with respect to hypothesis 3 are qualified but broadly supportive. On the one hand, in Models 2 through 4, the association between Wave 1 social isolation and Wave 2 violent victimization clearly seems to vary as a function of school-level safety and support. In all models, having more friends at Wave 1 is associated with lower risk of victimization at Wave 2, but lower average assessments of school safety are clearly associated with a weakening of the protective association between friendship nominations and victimization, and the extent to which a safer school environment is protective of "vulnerable"

students remains fairly stable across the models. In other words, a student with few friends is at higher risk of victimization in an unsafe school than he/she is in a safer school. This provides strong and consistent support for the first part of hypothesis 3.

On the other hand, the extent to which the link between violent perpetration and violent victimization varies as a function of school-level safety and support changes slightly across models, though the estimated associations never achieve conventional statistical significance. Models 2 and 3 produce substantively negligible estimates of how much the association between Wave 1 perpetration and Wave 2 victimization changes as a function of school-level safety. However, a slight elevation of risk is seen in Model 4, marginally significant at the 0.1 level. Still, the results overall corroborate the second prediction from hypothesis 3, which is that school safety and support will have no strong association with the chances of victimization for "available" victims – that is to say, for those who put themselves at risk of victimization by participating in violent situations, the efficacy of the school environment exercises no particular protective effect. Considering all the results together, it seems fair to conclude that better school safety and support does not reduce the chances of victimization for "available" students.

The finding that perpetrators of violence are at increased risk of later victimization, taken together with other research which shows that victimization is a risk factor for later violent perpetration (e.g. Bingenheimer, Brennan, & Earls 2005; Song, Singer, & Anglin 1998), may be interpreted as support for "cycle of violence" theories. Victimization increases the risk for later perpetration; perpetration increases the risk for later victimization. Violence begets violence. This empirical pattern may clarify other related phenomena. For example, Benbenishty and Astor (2005) observe that students who have been victimized are much more likely to carry weapons to school later in order to "protect themselves", which may end up producing the very violence that

students are seeking to avoid. Dodge (1980) finds a "feedback loop" between hostile attributions and aggressive behavior: students who are exposed to violence learn to interpret ambiguous situations as potentially hostile; these students attribute hostile intentions to ambiguous social cues, confirming their generally negative view of their peers, and react to the perceived hostility in such situations; peers see those aggressive reactions as evidence of the target student's underlying hostility and disagreeableness; those peers become more likely to interpret other future behaviors from the target student as hostile, and begin to treat him/her as a "bad kid"; peers treat the target student with the hostility which they feel his/her behavior warrants, and the cycle starts over again. And findings about the persistence of victimization patterns for some students (e.g. Finkelhor et al 2007, Kochenderfer-Ladd 2003) might be partially explainable as a function of students' oscillating between perpetration and victimization.

These results suggest a number of practical implications. This chapter began by asking three practical questions: What kinds of students are likely to be exposed to violence, and how can we identify them? To what extent can school practices reduce students' likelihood of being exposed to violence? And does the possibility of such protection vary as a function of those student-level characteristics which put students at risk of exposure in the first place? We have now arrived at some conclusive answers.

First, corroborating predictions derived from routine activities theory, at least two kinds of students are at higher risk of violent victimization – "available" students who perpetrate violence themselves, and "vulnerable" students who are relatively socially isolated. Based on this knowledge, teachers and administrators who have relationships with students may be able to assess students' relative risk of victimization in light of these qualities and thereby identify possible intervention strategies. This also has policy implications for the number and type of

interventions needed to prevent victimization. I suggested early in this chapter that by identifying whether victimization is concentrated among a single type of student or spread among several types, these results might help determine whether one or several sorts of interventions should be proposed. Based on these findings, it is clear that different subpopulations are at risk based on heterogeneous characteristics. This implies that more than one kind of intervention is needed.

Our assessment of the potential efficacy of interventions themselves, however, is slightly complicated by these results. It may be the case that improving school-level support and sense of safety, or other relevant elements of school efficacy, can provide additional protection and reduce the risk of victimization for socially isolated students. For one thing, encouraging denser or qualitatively closer social networks should help protect students. If schools cannot directly influence students' friendship networks, they may at least be able to offer an environment in which isolated students are safer – either by reducing their apparent suitability as targets, or by improving capable guardianship over them. However, when it comes to those students who "opt in" to participate in violence themselves, changes to the school environment seem unlikely to produce any direct change in their risk of victimization. For such students, results imply that teachers and administrators (as well as parents, community leaders, and relevant others) might have more success with individual-level behavioral interventions; this might be accomplished by attempting to dissuade students from participation in violent behavior directly, by attempting to limit their involvement in other risky behaviors (such as drug use, school misbehaviors leading to suspensions, etc.), or by increasing the salience of other protective influences in their lives.

These conclusions, of course, are tempered by a number of important limitations. For one, despite the high quality of the data, the characteristics of the Add Health public use sample present some restrictions. Network data is only available for schools in which at least 50 percent

of students completed the questionnaire and provided friendship nominations; if schools with lower response rates and sparser friendship matrices are substantively different from those who are included here, the results may be influenced by unknown bias. Additionally, some of the network identifying variables – including identification numbers for students' sent friendship and romantic partner ties – are not included in the public use subsample; ideally, we would like to match students to the characteristics of those whom they nominate as friends and investigate whether friends' risky behaviors influence students' chances of victimization. It is also possible that operational decisions for the key variables influence the results to an extent. The decision to treat both the outcome (victimization) and one of the key predictors (perpetration) as binary was a simplification that I believe is justified on the basis of the way the relevant questions were asked, but the argument can be made that students' subjective experiences of violence victimization and perpetration are likely to approximate a spectrum of intensity rather than a categorical yes/no; however, additional analyses (not presented here) generally suggest that other operational definitions of those two variables produce substantively similar results, and I believe that the results from the binary operationalization are easier to explain and understand.

Although some of the conclusions are mixed, the results from these analyses may be able to help put a practical handle on the problem of school violence, not only by identifying those types of students likeliest to be victimized, but also by proposing possibilities for how different intervention methods can be tailored in order to give these students the help they need.

CHAPTER FIVE

Conclusion

Throughout this dissertation, a number of questions have emerged as underlying themes. These include: Is exposure to school violence actually harmful for students? If so, what kind(s) of harm does it do? For what kind(s) of student is it most harmful? What kind(s) of student are at risk of exposure? And what can schools do to reduce the risk of violence and the harms it causes? At the end of the dissertation, it makes sense to revisit these questions and see what answers we can give in light of the findings from the previous chapters, as well as to review the contributions of this dissertation more generally to understanding the process of school violence.

Luckily (or perhaps unluckily), the first question is the one with probably the most unequivocal answer: School violence exposure appears to exert a clear harmful effect on students. This result persists, in some form or another, across all three main chapters.

To examine this more closely we may expand into the second question: if school violence is harmful, what kind of harm does it do? In Chapter 2 we see that exposure to violence while in school results in an increase in students' average level of depression, measured with the CES-D scale, during their young adulthood. Because of the propensity score modeling strategy used, we may interpret these results as causal effects. While poor mental health or socio-emotional functioning may put students at higher risk for being victimized, the effects of violence exposure on depression are not confounded by this; violence exposure itself exerts a corrosive effect on students' later mental health. Chapter 2 also suggests, though does not examine directly, another domain in which violence exposure causes harm: early violence exposure appears associated, to a large extent, with an increase in students' chances of suffering later violence exposure.

Chapter 3 looks at three outcomes on which violence exposure may cause harm: as well as depression, it examines academic attainment and suicidal ideation. Patterns of results are largely consistent with Chapter 2. For one, those who are exposed to violence early in life have worse patterns on all three outcomes variables later in life, as well as much higher chances of being exposed to later violence, when compared to those without such early exposure. Moreover, even isolated later-life exposure produces negative causal effects on academic attainment and suicidality – though curiously not on depression, an oddity in the results which may implicate differences in the etiologies of the two problems (Arria et al 2009, Thompson et al 2005). And while later-life repeat exposures do not in and of themselves appear to produce much change in any of the three outcomes, this is likely because those who were exposed early in life are already on a more negative trajectory and thus further exposure has little room to cause additional harm.

Chapter 4 picks up a thread suggested in Chapter 2 and addressed directly in Chapter 3 by examining a different sort of harmful outcome: that participation in violence is associated with later victimization by violence. The results strongly indicate that students who commit violence are at much higher risk of being victimized later, and also suggest that this association persists independent of school protective effects. As noted in the chapter, this finding suggests that one of the harmful outcomes of violent victimization is the possible establishment and perpetuation of a "cycle of violence," in which victimization begets later perpetration and that perpetration begets further victimization. Persistent student victimization may be a result of this pattern.

This leads to the next question: on what kinds of student is violence exposure most harmful? Chapter 2 suggests, contrary to expectations, that there may not be much difference between male and female students in the size of the effect of school violence exposure on later depression; violence is approximately equally bad for members of both sexes, and if anything the

effects of the kind of violence examined here are worse for men than for women. Chapter 3 further establishes that violence's effects vary, if not much by sex, then certainly by life course stage and history of exposures. Specifically, students who are exposed to violence early in life suffer distinctly worse patterns of outcomes than students whose exposure comes later. This also has implications for causal estimates of violence's effects on students: students with isolated, later violence exposure suffer distinct negative effects as a result, whereas students with early patterns of violence do not seem to suffer many additional ill effects as a result of later specific instances of repeat exposure. Violence, by this reckoning, is most harmful on those who experience it early, though it still causes harm to those who experience even isolated exposures later in life. This "concave" association suggests that violence's effects may be cumulative, but they are not strictly additive; from a policy perspective, this implies that prevention of early, initial exposures should produce more aggregate benefit than mitigation of the effects of later exposures among those who are already set on a more negative trajectory.

Chapter 4 does not address the effects of violence directly, but it does imply difference in outcomes for different kinds of student. A certain kind of student – one who voluntarily participates in violence via self-reported perpetration – may become trapped in an ongoing cycle of violent actions and violent victimizations. Another kind of student – one who is socially isolated and relatively disadvantaged in terms of friendship and social support – is also at elevated risk of victimization; combined with what is known about the effects of social support on mental health (e.g. Cacioppo et al 2006), these results plausibly imply that negative socio-emotional consequences could be magnified for students of this type.

Findings throughout the main chapters also help answer the question of what places students at risk of exposure. Previous research has provided a litany of possible answers to this

question, including male gender; obesity, physical disability, and other body issues; non-gender-conforming behavior, especially relative to heterosexuality and especially for males; learning disability; neighborhood disadvantage; cultural valuation placed on violence as a mode of conflict resolution; weak bond to school; and many more (Benbenishty & Astor 2005, Berg et al 2012, Olweus 1993, Pascoe 2011, Saner & Ellickson 1996, Swearer et al 2010). The findings here additionally confirm predictions derived from several relevant sociological theories. Consistent with life course theory, Chapter 2 suggested – and Chapter 3 confirmed – that one of the key risk factors for violence exposure is violence exposure itself; that is, people who have been exposed before are at a much higher risk of being exposed again later. As noted, Chapter 4 draws on routine activities theory to predict, and demonstrate the empirical salience of, two other distinct types of student at risk of victimization – the first type, which is based on what I called "availability," includes those who put themselves in risky situations by perpetrating violence; the second, which is based on what I called "vulnerability," consists of those who are socially isolated and so lack the protection of a peer group which might dissuade potential perpetrators.

And what steps can schools take to protect students and reduce the risk of violence? The answers here are unfortunately mixed. Chapter 4 finds that more favorable school climate, in the form of higher levels of average subjective sense of safety among students, can exert a protective effect on "vulnerable" students – those who are socially isolated, with few friends. In the terms of routine activities theory, this is presumably a result of safer and better-functioning schools' ability to provide capable guardianship over students who are potentially suitable targets for victimization, and perhaps also to reduce opportunities for motivated offenders to encounter these suitable targets in the absence of such guardianship. However, Chapter 4 also finds that "available" students who opt in to risky situations are not amenable to such protection: their risk

of victimization does not decrease as a function of higher levels of school safety. Other key ideas from routine activities theory, though, imply that schools may be able to reach these students by other means – for example, by attempting interventions that aim directly to reduce their motivation to offend. Such efforts have met with mixed success in the past (cf. Devine 1996), but remain a theoretically salient possibility.

Overall, the findings of this research suggest important theoretical, methodological, and practical conclusions. Besides supporting predictions derived from life course theory and routine activities theory, these results throw into relief the limited but fundamentally important role of the school in the process of violence. As has been noted throughout, school violence has unique characteristics, largely due to its being shaped by the school context. Schools determine the peers with whom students will interact; schools provide the structure for those interactions, opening up some opportunities for violence and closing off others; and schools as institutions may have the resources to protect certain kinds of students, but appear to have limited leverage on others. Schools cannot control the past violence experiences of their students, nor can they directly influence the kinds of violence that take place outside of the school itself. However, the results from all three chapters highlight the ways in which schools do matter. Even if they cannot prevent violence exposure for all types of student, because they provide as much oversight as they do, schools may be in a unique position to intervene after violence exposure has happened and potentially mitigate its negative consequences.

Methodologically, this dissertation contributes to a growing body of literature which attempts to address effects of violence exposure on students' lives using rigorous statistical and analytical tools for drawing causal inferences from observational data (e.g. Bingenheimer, Brennan, & Earls 2005; Sampson, Sharkey, & Raudenbush 2008; Sharkey 2010; Sharkey et al.

2012). Most research investigating the links between violence exposure and later harm faces issues of endogeneity and selection bias, but consensus has begun to build behind the conclusion that violence exposure is a cause of later harmful effects – not just a result of pre-existing dispositions towards poor outcomes or a concurrent result of underlying risk factors. The results here, especially those obtained in Chapters 2 and 3, further support this conclusion.

As for practical upshots, these findings warrant a number of salient suggestions. For one, the acknowledgment that violence itself is actually harmful might give additional weight to policy decisions seeking to address the problem. Responses to school violence often feature a dismissive "kids will be kids" attitude; these and other related findings should call attention to the seriousness of the problem. Additionally, the recognition that violence's effects vary based on life circumstances offers several possibilities for preventative or mitigating interventions. Knowing that early exposures set students on a more generally negative trajectory underscores the importance of early interventions to nip potential problems in the bud; doing so may help prevent later academic and mental health problems and also reduce the likelihood of later re-victimization. Schools and other institutions could also provide additional support to students who suffer chronic patterns of repeated victimization – such students are at a greatly elevated risk of various negative outcomes, and interventions to assess and respond to their situations may help set them on a healthier track – but under conditions of limited resources, early prevention from initial exposure seems to be the more fruitful of the two options. For both of these concerns, results imply that early detection and intervention would be helpful; schools are likely in a relatively advantaged position for carrying this out. And although the results from Chapter 4 paint a pessimistic picture of schools' potential to help some kinds of students, this is by no

means to suggest that improving school climate is a waste of time. By protecting even a limited subset of students, schools can greatly reduce aggregate harm in the long term.

Still open are questions regarding what kind of interventions are most effective at reducing school violence and its negative effects. Some options are suggested by the routine activities orientation in Chapter 4. As noted, the theory offers four possible points of leverage on violence prevention: reducing the motivation of potential offenders, reducing the apparent suitability of potential targets, increasing the extent or capability of guardianship, and reorganizing "routine activities" in school so as to minimize possible situations in which the three key elements can co-occur. The first two strategies can be thought of as operating on individual-level factors; while there is certainly no shortage of ideas for how to do this, some of the more interesting ones suggest channeling physical energies into nonviolent applications. For example, Becoming a Man – Sports Edition, implemented in Chicago Public Schools, found dramatic reductions in violent behavior among students who were treated with a program involving participation in non-traditional sports (such as archery, boxing, handball, martial arts, weightlifting, and wrestling) and counseling based on cognitive behavioral therapy (CBT) designed to reduce hostile attribution biases in attitudes and behaviors (Heller, Pollack, Ander, & Ludwig 2013). Other studies show that traditional martial arts training – physical exercise with accompanying philosophical emphasis on respect, responsibility, control, patience, and honor – can reduce violent tendencies in delinquent youth (e.g. Coakley 2001).

The second two strategies – improving guardianship and modifying routine activities – operate at the school level, and therefore may be easier for schools to implement. Some studies have found that schools can leverage these factors simply by increasing well-lit hallways and highly supervised areas, and closing off concealed corners and nooks (Warner, Weist & Krulak

1999). Another option which seems plausible is to change school culture. By making schools a more nurturing place, where students' human rights are treated as primary and shared norms explicitly designate violence as unacceptable, schools may be able to reduce the prevalence of problem behaviors. A number of anti-bullying programs – in particular, the Olweus Bully Prevention Program (Olweus, Limber, & Mihalic 1999) – seek to do exactly this, though they have met with mixed success in the United States (e.g. Bauer, Lozano, and Rivara 2007), and not all violence problems in schools are examples of bullying so defined.

Despite the limits identified throughout this research, schools have a uniquely fruitful potential as sites for violence prevention efforts. Schools may not have the unfettered ability to intervene in all aspects of students' lives, but they retain the capacity to do a great deal of good.

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