

**Three Essays on the Relationship between Social Ties and Mental Health**

by

Michael Fang

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Doctoral Committee:

Associate Professor Elizabeth E. Bruch, Co-Chair  
Professor Sarah A. Burgard, Co-Chair  
Assistant Professor Rachel K. Best  
Professor Paula M. Lantz  
Professor Jeffrey D. Morenoff

Michael Fang

[mikefang@umich.edu](mailto:mikefang@umich.edu)

ORCID iD: [0000-0003-2849-1780](https://orcid.org/0000-0003-2849-1780)

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## **Abstract**

A large body of research has documented a link between social ties and mental well-being. Indeed, hundreds of studies have shown that individuals with robust ties to their family, friends, and communities live longer, happier, and healthier lives compared to individuals without these same relationships. Despite emerging as one of the most robust associations in the literature, existing research has yet to adequately grapple with issues related to effect heterogeneity, mechanisms, and temporality, thereby limiting our understanding of the full impact of social ties. The overarching goal of this dissertation was to begin addressing these knowledge gaps, utilizing three prominent sociological theories as case studies.

In the first empirical chapter, I used data from The National Longitudinal Study of Adolescent to Adult Health (Add Health) to test the social contagion hypothesis, which posits that social phenomena including affective states can spread among people in close contact. Results from school and grade fixed effects models found compelling support for the hypothesis, as classmates' depressive symptoms were positively correlated with respondents' depressive symptoms a year later. This association was especially pronounced for low-SES and Black students and was driven, in part, by the lower level of social support students perceived they had from classmates with depressive symptoms. The second empirical chapter also used the Add Health to reassess the reactivity hypothesis, which suggests that girls may be more vulnerable to the adverse effects of interpersonal stress, contributing to gender differences in depressive symptoms. First-difference models found qualified support for this theory: while the association between peer and school-based interpersonal stress and depressive symptoms did not differ by



gender, familial interpersonal stress was more strongly related with depressive symptoms for girls. These results suggest that girls may be more sensitive to the influence of social ties, though this tendency may be domain specific rather than universal, as is often implied by the reactivity hypothesis. In the third empirical chapter, I used data from the 1979 cohort of the National Longitudinal Survey of Youth to examine whether the impact of marriage on mental well-being becomes stronger over time, as implied by life course theory. Results from marginal structural models showed that increased exposure to marriage during early adulthood was positively associated with mental well-being later in life. These results highlight the need to further consider the length of time social relationships are maintained to more fully understand their impact on health.

## **Chapter 1: Introduction**

Mental and emotional well-being are important components of overall health. According to the US Department of Human and Health Services, positive mental health enables people to “realize their full potential, cope with the stresses of life, work productively, and make meaningful contributions to their communities.” A large body of research supports this claim, showing a strong association between mental disorders and health problems such as physical morbidity and premature mortality (Barth, Schumacher and Herrmann-Lingen 2004, Cuijpers and Smit 2002, Russ et al. 2012, Carney et al. 2002, Moussavi et al. 2007, Pan et al. 2011, Rugulies 2002). Mental well-being also has broad social implications. Depression, for example, costs tax payers and employers an estimated 210 billion dollars annually (Greenberg et al. 2015). Given its broad impact, understanding the determinants of mental well-being remains a critical task for researchers, policy makers, and medical practitioners alike.

Early research on the etiology of mental well-being focused largely on biological causes, such as chemical imbalances in the brain (Coppen 1967, Schildkraut 1965). However, researchers have increasingly recognized the limitations of this approach and have called for greater examination of the psychosocial determinants of mental health (Engel 1977). Within this context, one social factor that has received considerable attention is social relationships. This line of work began with Durkheim’s classic study on social integration and suicide (Durkheim 1951). In the 1970s, a series of influential epidemiological papers built on this research, finding that unmarried men and socially isolated individuals were at greater risk for psychotic disorders (Berkman and Syme 1979, Cassel 1976, Cobb 1976). Since then, hundreds of studies have

shown that individuals with robust ties to their family, friends, and communities live longer, happier, healthier lives compared to individuals without these same relationships (Aneshensel 1992, Cohen 2004, House, Landis and Umberson 1988, Kawachi and Berkman 2001, Smith and Christakis 2008, Umberson, Crosnoe and Reczek 2010).

Despite emerging as one of the most robust associations in the literature, existing research is limited by its implicit use of a binary perspective. That is, studies have generally sought to offer a straight-forward, yes/no answer to the question: do social relationships matter? Of course, this does not mean studies have not examined complexity within social relationships or how they might differentially impact different groups. However, even these more nuanced results have been understood in terms of how they fit within the binary perspective. Moreover, empirical investigations of underlying theoretical mechanisms are largely absent from the literature (Thoits 2011). Thus, as a result of the binary perspective, important substantive questions around how and under what social conditions social ties matter remain underexamined.

The overarching aim of this dissertation was to begin addressing this gap, focusing on three broad questions. First, does the association between social relationships and mental well-being vary across groups? Most studies assume that social ties have a uniform effect on all individuals, ignoring potentially rich forms of effect heterogeneity. Second, what are the mechanisms linking social relationships and mental health? While the underlying mechanisms have been subject to thorough theorization, few studies have empirically tested their existence. Third, are the potential benefits of social relationships on mental well-being cumulative? While theoretical work often suggests that the effects of relationships are strongest when they are sustained, existing research has largely ignored the role of time.

To examine issues of effect heterogeneity, causal mechanisms, and temporality, I conduct three separate studies, each which revisit an influential hypothesis related to social relationships and depressive symptoms. These hypotheses help ground the dissertation in extant theoretical debates and serve as case studies through which to probe the knowledge gaps described above. This information is not only valuable from a theoretical perspective, but may additionally aid policy makers in the development of more effective interventions. For instance, the findings on heterogeneous effects may enable interventions to better target individuals who are most sensitive to the effects of social relationships. This dissertation thus stands to make important substantive and policy contributions related to mental well-being.

Each paper also employs a novel methodological approach to more rigorously test these longstanding ideas. Researchers in the social sciences have increasingly focused on examining the impact an exposure (e.g., social ties) on an outcome (e.g., depressive symptoms) using an ever growing set of methodological tools (Gangl 2010; Morgan and Winship 2014). Existing research on social ties and mental health, however, has been slow to adopt these methodological advances. Indeed, even much of the newest work in the literature still relies on conventional regression analyses, which estimates the association between social ties and mental wellbeing by adjusting for observable confounders (Umberson, Crosnoe and Reczek 2010). While this research is important, it is also limited because it may not account for unobserved confounders and may also underestimate the effect of social relationships by “overcontrolling” for indirect pathways (Morgan and Winship 2014). Though experimental designs most reliably deal with these issues, these new tools can help reduce the influence of estimation issues where experimental data is not available.

In Chapter 2, I used data from The National Longitudinal Study of Adolescent to Adult Health (Add Health) to re-evaluate the social contagion hypothesis, which posits that phenomena including depressive symptoms can spread among people in close contact. Results from school and grade fixed effects models, which leverage small variation in the mental-wellbeing of classmates within the same schools in different grades, indicated that greater exposure to peers with depressive symptoms was positively related to higher levels of depressive symptoms a year later. Estimates, however, were much smaller than prior studies, suggesting that conventional approaches may overstate the magnitude of peer contagion. This paper also found that the estimate for peer contagion was particularly pronounced for low-SES and Black students and was driven, in part, by the lower level of social support students perceived they had from classmates with depressive symptoms.

In Chapter 3, I again used the Add Health to reassess the reactivity hypothesis, which suggests that girls may be more vulnerable to the detrimental effects of interpersonal stress, contributing to gender differences in depressive symptoms. First-difference models, which eliminate the influence of time constant confounders, found qualified support for this theory: while the association between peer and school-based interpersonal stress and depressive symptoms did not differ by gender, familial interpersonal stress was more strongly associated with depressive symptoms for girls. Mediation analysis further showed that this difference may emerge because familial problems may elicit more negative affect among girls. Taken together, these results suggest that girls may be more reactive than boys to certain types of interpersonal stress, though this trend may depend heavily on the specific social ties considered.

In Chapter 4, I examined whether the positive (negative) association between marriage (being unmarried) and mental well-being becomes stronger over time, as implied by the life

course theory. Estimating the potential cumulative effect of marriage, however, is methodologically challenging because standard approaches do not properly address the impact of time-varying confounders (e.g., income) that both influence, and are influenced by, marriage. Drawing on the National Longitudinal Survey of Youth 1979, I utilized marginal structural models with inverse-probability of treatment weights to account for simultaneously confounding and mediating factors. While estimates from conventional regression models were relatively small, results from marginal structural models showed that those who were continuously married had significantly fewer depressive symptoms than those who were continuously unmarried. Secondary analyses also indicated that (1) the cumulative benefits of marriage may be stronger for Blacks than Whites; (2) long-term cohabitation may not offer the same cumulative benefits as marriage; and (3) the benefits of long-term marriage may be more pronounced in the latter parts of early adulthood.

## **Chapter 2: Peer contagion and mental well-being among adolescents**

### **Introduction**

While mental and emotional well-being are fundamental to individuals' overall quality of life, current rates of mental health problems are alarmingly high. For instance, an estimated 7.6% of all Americans over the age of 12 suffered from depression in any two-week period from 2009 to 2012 (Pratt and Brody 2014). From a public health perspective, this is a critical concern, as mental health issues significantly reduces quality of life and is a major risk factor for a variety of health problems, including suicide, illicit drug use, and heart disease (Franklin et al. 2017; Rosenfield and Mouzon 2013, Walker, McGee and Druss 2015). Mental health problems also carry broad social implications; depression, for example, costs tax payers and employers an estimated 210 billion dollars annually (Greenberg et al. 2015). Given its broad health and economic impact, understanding the determinants of mental well-being remains a critical task for researchers and medical practitioners.

Within the social sciences, social contagion theory offers a provocative explanation for how poor mental well-being may emerge. More specifically, it posits that certain social phenomena, including moods and emotions, are “contagious” and can spread among people in close proximity (Christakis and Fowler 2013; Hatfield, Cacioppo and Rapson 1993). Thus, exposure to individuals with poor mental health may lead others to develop mental health problems as well. A number of empirical studies find support for this contagion hypothesis. Most notably, Christakis, Fowler and their colleagues have shown in several well-known articles that the level of depression, happiness, and loneliness among family, friends, and neighbors have a

substantial effect on one's own odds of being depressed, happy, or lonely (Rosenquist, Fowler and Christakis 2011; Cacioppo, Fowler and Christakis 2009; Fowler and Christakis 2008). Given these findings, they conclude in one of these papers that moods such as happiness are “not merely a function of individual experience or individual choice but is also a property of groups of people. Indeed, changes in individual happiness can ripple through social networks...giving rise to clusters of happy and unhappy individuals” (Fowler and Christakis 2008: 7).

Despite this evidence, there are at least three important shortcomings with existing research on the contagion hypothesis. First, much of the empirical research has been criticized for its inability to account for important confounding factors (Manski 1993; Cohen-Cole and Fletcher 2008). Second, while some researchers have theorized that some social groups may be more sensitive to social contagion effects than others, most empirical studies have assumed that contagion has a uniform relationship (Umberson and Montez 2010). Therefore, potentially important forms of *effect heterogeneity* remain largely unexplored. Third, while researchers have posited a variety of ways exposure to individuals with poor mental health may influence one's own mental health, few studies empirically test these (Kawachi and Berkman 2001; Thoits 2011). Because of this, it remains unclear *how* mental well-being might spread between people.

In this study, I use a within-school, between-cohort design to test of the contagion hypothesis (Hoxby 2000). To provide a more intuitive understanding of this approach, consider a hypothetical school where, on average, 10% of all incoming students have depressive symptoms. However, due to chance, some grades occasionally may have a slightly larger (e.g., 11%) or smaller (e.g., 9%) share of students with depressive symptoms. If the social contagion hypothesis holds true, we should expect to find that students who are exposed to grades with a greater share of classmates with depressive symptoms will themselves be more likely to develop these



symptoms compared to those in grades with classmates with fewer depressive symptoms.

Though not a traditional experiment, such a design is better able to account for important sources of bias (e.g., selection) compared to more conventional analyses. To complement this main analysis, this study also examines effect heterogeneity across social demographic characteristics and potential mechanisms that might mediate peer contagion. Taken together, this paper provides a rigorous and comprehensive examination of the social contagion hypothesis.

### **Theoretical Background**

Existing sociological research highlights a variety of social mechanisms through which poor mental health may be “contagious” (Kawachi and Berkman 2001; Thoits 2011). For the purposes of this study, I organize these into one of three inter-related pathways.

The first is social influence or imitation. According to Thoits, this refers to the tendency of individuals to “assess the appropriateness of their own attitudes, beliefs, and behaviors against standards that are avowed and/or modeled by reference group members, usually shifting their own to match those of the group” (Thoits 2011: 147). Adolescents may be particularly susceptible to imitation given the pressure to fit in with peers during this specific point in the life course (Coleman 1961). In schools where there is a high share of students with poor mental health, students may adopt norms and behaviors that ultimately elevate their own risk for mental health issues. For example, individuals with depression commonly avoid social contact and activities (Rubin, Coplan and Bowker 2009). Students may in turn mimic their peers and begin withdrawing from social activities, viewing such behavior as normal and acceptable.

The second is the level of social support received from others. Social support refers to the level of help and care (either perceived or actual) individuals have available from friends, family, and others in their social network (House, Umberson and Landis 1988). Social support can come

in a variety of forms, including emotional, financial, or informational. Regardless of the specific type of help, research has consistently shown a positive link between social support and mental-wellbeing (Umberson and Montez 2010). However, in environments with a high share of students with poor mental well-being, it is possible that the quality and level of social support available for students may be diminished. This is because students with poor mental health may be less willing/able to provide social support to their classmates (Joiner and Katz 1999). Similarly, classmates with poor mental health may be less willing to socialize with others, reducing the number of potential friendships available to other students (Rubin, Coplan and Bowker 2009). In both cases, the level of social support available to students becomes reduced, increasing their risk of mental well-being problems.

The third way poor mental well-being may “spread” is by having to provide social support to classmates with mental health issues. Research shows that while receiving social support positively impacts well-being, providing care to others often does the opposite, especially if the recipients of the support have health problems. For instance, a rich literature demonstrates that caring for an elderly parents often carries a significant psychological toll for family members (Schulz and Martire 2004; Eters, Goodall and Harrison 2008; Chiao, Wu and Hsiao 2015). Providing support for classmates with mental health issues may also be challenging, as some research suggests that interactions with these adolescents may be highly stressful and unpleasant (Coyne 1976). Consequently, individuals may come away from the experience with elevated levels of stress. Furthermore, adolescents may empathize with their distressed classmates and begin internalizing their emotional problems (Hatfield, Cacioppo and Rapson 1993), which may in turn generates negative feelings. In schools with a high share of classmates with poor mental health, students may be more likely to come into contact with and

provide support for their distressed peers, ultimately raising their own risk of mental health problems.

### **Prior Empirical Studies**

A small body of empirical research has tested the contagion hypothesis, finding a positive association between exposure to individuals with poor mental health and a respondents' own likelihood of developing psychological distress. Broadly speaking, studies have employed one of three research designs.

The first is small scale experimental research. In general, these studies examine contagion effects by exposing a small, randomly selected group of respondents to a negative emotional stimulus, most commonly interaction with a depressed individual. Respondents' emotional reaction to this encounter is measured and then compared to a control group that is exposed to a more neutral stimulus, such as interaction with a non-depressed individual. A meta-analysis by Joiner and Katz (1999) conclude that within these short-term experimental conditions, the treatment group typically reports higher levels of depressive symptoms, suggesting that negative emotional affect may be contagious and can spread between individuals (Joiner and Katz 1999). This finding is consistent with related experimental research, which shows that emotions more generally can be passed on from person to person (Hatfield, Cacioppo and Rapson 1993). Nonetheless, while these studies yield compelling causal evidence, they still only provide a limited test of the contagion hypothesis because they temporarily induce negative emotional feelings within a confined setting. As others have noted, this is fundamentally different from spreading depressive symptoms under real conditions over a more sustained amount of time (Eisenberg et al. 2013).

A second type of research is correlational research. These studies typically examine the association between an individual's depressive symptoms and those in their social networks after controlling for observable confounders, in particular prior depression. A number of correlational studies in the literature rely on small, non-representative samples that only contain limited information about respondents' background characteristics (Prinstein 2007; Stevens and Prinstein 2005; Zalk et al. 2010). An important exception to this is the work conducted by Christakis, Fowler, and their colleagues, who offer compelling evidence for contagion effects using the Framingham Heart Study, a representative longitudinal dataset with rich social network information (Christakis and Fowler 2013; Cacioppo, Fowler and Christakis 2009, Rosenquist, Fowler and Christakis 2011; Fowler and Christakis 2008). In one study, they find that living close to a happy friend increases an individual's odds of being happy themselves by 25% (Fowler and Christakis 2008). In a second study, Rosenquist et al (2011), report even larger effect sizes; living near a depressed mutual friend increases the odds that a respondent is depressed by 359% after adjustment for covariates.

While correlational research using representative longitudinal data offer important evidence for the social contagion hypothesis, it is often criticized for its inability to convincingly account for confounding factors (Manski 1993; Cohen-Cole and Fletcher 2008). In particular, the issues of selection and common shocks confront any researchers estimating effects related to social interaction. Individuals with depression may cluster because of selection/homophily, or the tendency for individuals with similar characteristics to come together. Additionally, we might observe clustering because exposure to common environmental factors, such as a dangerous violent neighborhood, may increase all individuals' risk of depression. There is some evidence that conditioning on observables may be insufficient for addressing the influence of these

alternative causal pathways. In one particularly noteworthy example, Cohen-Cole and Fletcher (2008) use the Add Health dataset to replicate a significant obesity contagion effect after controlling for observable confounders, as reported by Christakis and Fowler (2007). However, after they adjust for common shocks through school-fixed effects, they show that the effect of social contagion completely disappears.

A third study design—quasi-experimental research—tries to address issues related to unobserved confounders by relying on naturally occurring variation in a respondent’s social network to identify causal effects. To my knowledge, only one study has used this type of approach. In their study, Eisenberg et al. (2011) investigate mental health contagion effects among 1,641 students with randomly assigned roommates across two colleges. Overall, they report finding little evidence that having a depressed roommate increases one’s own odds of developing depression. The main exception to this is male students with pre-existing depressive symptoms; for this population, exposure to other depressed roommates appeared to have a strong contagion effect. This study, however, is limited in its generalizability since it only focused on two universities.

In sum, a large body of research finds a significant contagion effect for depressive symptoms, though only a handful provide convincing evidence of longer-term effects using rich longitudinal data. These studies fail to adjust for known confounding factors, however, raising questions about the validity of their findings. One existing study has corrected for these, finding much more qualified support for social contagion theory. Nonetheless, this study is also limited because its sample only covered two colleges. In this study, I aim to contribute to this existing literature on social contagion and depressive symptoms by employing a more rigorous research design with a nationally representative sample of American adolescents.

## **Heterogeneous Effects**

There are also important theoretical reasons to expect that certain socio-demographic groups may be more vulnerable to contagion effects compared to others. The majority of this work centers on gender differences, with most work suggesting stronger contagion effects for women. Socialization theories, for example, posit that women may be more relationship oriented than men because of differences in childhood rearing practices (Chodorow 1999; Radloff and Rae 1979). As a result, the influence of peers with poor mental health may be stronger for them than men. Others speculate that girls may be more prone to provide care for others (Gilligan 1982; Kessler and McLeod 1984). Because of this, they may be more likely to be exposed to and internalize the negative feelings from their classmates with depressive symptoms. Existing empirical work testing gender heterogeneity has yielded mixed results. Short-term experimental studies on the subject have generally found that exposure to depressed individuals has a similar effect for both males and females (Joiner and Katz 1999). On the other hand, two correlational studies report a stronger effect for women (Rosenquist, Fowler and Christakis 2011; Cacioppo, Fowler and Christakis 2009). One study finds stronger contagion effects for male versus female college students (Eisenberg et al. 2011). In general, it remains unclear whether the exposure to individuals with poor mental health is more harmful for boys or girls.

There are also theoretical reasons to suspect that the contagion effect varies across racial/ethnic and SES lines. For instance, the well-known “vulnerability hypothesis” posits that disadvantaged groups may have fewer psycho-social resources to handle stressors such as exposure to depressed individuals, making them more susceptible to the negative consequences (Aneshensel 1992). Yet, virtually no empirical study has directly examined contagion

heterogeneity by race and SES, leaving this critical question unanswered. Some existing work finds that social relationships may have a stronger impact on mental health for disadvantaged populations, providing suggestive evidence for differential effects across race and SES. For instance, some studies show that relative to Whites, familial social support may have a stronger impact on African-Americans' and Hispanics' mental-wellbeing (Umberson and Montez 2010; Eschbach et al. 2004). Nonetheless, others have found no differential effect or the exact opposite when examining different types of social relationships, such as marriage (Umberson and Montez 2010). This suggests that the direction of the moderation effect may be contingent on the type of social relationship examined. One of the contributions of this study is to explore how the contagion effect may vary by gender, race, and class.

## **Data and Methods**

### **Data**

In this study, I draw on the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative sample of American adolescents. The study began in the 1994-95 school year with a nationally representative sample of 144 high schools and associated feeder middle schools. During Wave I, a brief questionnaire was administered to all students (grades 7-12) attending these schools (N = 90,118). A more detailed in-home interview was also conducted with a representative sub-sample of students and their parents (N = 20,745). In Wave II, conducted in 1996, researchers conducted follow-up interviews with students and parents from the in-home sample (N = 14,738).

Figure A-1 shows the restrictions I implement to create my analytic sample. First, I limit my sample to students who were followed from Wave I to Wave II because my analysis draws on information from both waves of data collection (N=14,738). A large portion of these dropped cases were students who were 12<sup>th</sup> grade in Wave I, who were intentionally omitted by Add

Health researchers because these students were no longer in high school during Wave II. Next, I drop 705 students who were in 12<sup>th</sup> grade during Wave I but still remained in the study in Wave II. Third, I drop all students at or below 8<sup>th</sup> grade or lower during wave I (N=4,463). Prior research finds systematic differences in depression rates between middle and high school aged students, suggesting that the two may not be directly comparable (NIHM 2015). Fourth, I drop 1306 cases with missing information on study variables, such as school identifiers and sample weights. Fifth, I omit respondents in grades with 20 or fewer sample students in order to ensure that small and potentially noisier grades are not included in the study. Sixth, I drop 172 students in schools that have only one cohort (i.e., only 9<sup>th</sup> graders) because this approach relies on variation within schools between multiple grades. Finally, I drop 29 students because they report being 9<sup>th</sup> grade students in schools for 10-12 graders. These exclusions yield a final analytical sample of 7,769 students in grades 9-11 across 68 schools.

### **Measures**

*Predictor variable: Proportion of students in a cohort with depressive symptoms*

During the first wave of data collection, nearly all 90,000 students in the in-school sample responded to a limited number of questions related to their mental health, including the following: “In the last month, how often did you feel depressed or blue?” Responses for this question ranged from 0 to 4 (0 = never; 1 = rarely; 2 = occasionally; 3 = often; 4 = everyday). Those who answered 3 or 4 were coded as having depressive symptoms, while those who answered 0-2 were coded as not having depressive symptoms. I then aggregated this binary measure to the school-cohort level, creating a variable that captured the share of students who had depressive symptoms within a specific cohort/grade at a particular school during wave one of data collection.

*Dependent Variable: Depressive status*



The main outcome of interest in this study is depressive symptoms, measured at wave 2 of data collection. Unlike the in-school sample, students in the in-home sample were asked a more comprehensive set of questions related to their mental health, including 19 questions capturing depressive symptoms over the past week. These items were modeled after the Center for Epidemiologic Studies' Depression (CES-D) scale, a reliable, well-validated 20 item instrument used to identify individuals at risk of major depression (Radloff 1977). Responses to these 19 questions ranged from 0 to 3, (0 = never or rarely; 1 = sometimes; 2 = a lot of the time, 3 = most or all of the time). These items were first summed to create a single index ( $\alpha=0.86$ ), then converted into a binary indicator (0 = no depressive symptoms, 1 = has depressive symptoms).<sup>1</sup> Prior psychiatric studies recommend using a cutoff score of 24 for girls and 22 for boys to maximize sensitivity and specificity for major depressive disorder, as defined by the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (Roberts, Lewinsohn and Seeley 1991). However, these values were determined using the standard 20 item CES-D scale. Since the Add Health scale only has 19 items, I adjust the cutoffs at 23 for girls and 21 for boys. These modified cut-offs follow several other studies examining depressive symptoms using the Add Health dataset (Lehrer et al. 2006; Shrier, Harris and Beardslee 2002).

### *Control Variables*

I also include a limited set of socio-demographic variables in the analysis as controls. These were all measured at Wave I and included respondents' race (White, Black, Hispanic, and Other), parental education (0 = both parents did not attend college; 1 = at least one parent attended college), gender (0 = male, 1 = female), age, and immigration status (0 = native born; 1 = foreign born), and prior depressive status (0 = no depressive symptoms; 1 = has depressive

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<sup>1</sup> The CES recommends dropping cases with 4 or more items. However, this cutoff is for the standard 20 item CES-D scale. Since the Add health depression scale only has 19 items, I drop students with three or more missing items.

symptoms). Prior depressive status was measured in the same way as the dependent variable: that is, during the Wave I interview, respondents were asked 19 questions related their depressive symptoms over the past week and scores were converted into a binary indicator.

## Methods

This paper uses two complementary analyses to test the contagion hypothesis. In the main analysis, I use variation in the share of students with depressive symptoms across grades within a given school in an attempt to limit issues related to selection or common shock. This is a common approach used within other social sciences (especially economics) to examine the impact of peer effects (Hoxby 2000; Bifulco, Fletcher and Ross 2011). To implement this strategy, I estimate the following logistic regression model:

$$Y(\text{Depression status, Wave II})_{ijk} = \beta_0 + \beta_1 (\% \text{ of cohort with depressive symptoms})_{jk} + \beta_2 (\text{Depression status, Wave I})_{ijk} + \beta_3 (\text{Individual controls})_{ijk} + \beta_4 (\text{School FE})_j + \beta_5 (\text{Grade FE})_k + \varepsilon_{ijk}$$

where  $i, j, k$  are indices for students, schools, and grades, respectively;  $Y_{ijk}$  is a binary indicator of student's depression status at wave 2;  $\beta_4$  is a school-fixed effect;  $\beta_5$  is a cohort or grade fixed effect;  $\beta_2$  is a binary indicator of a student's depression status at wave 1;  $\beta_1$  is the main parameter of interest and represents the percent of students in school  $j$  and cohort  $k$  that have depressive symptoms;  $\beta_3$  is a vector of student-level covariates;  $\varepsilon_{ijk}$  is an error term that may correlated across observations within the same school. To account for this, I cluster standard errors at the school level.

School fixed-effects help absorb average observed and unobserved difference between schools. Similarly, grade fixed effects absorb average differences between grades. This may be particularly important for the Add Health data because students in different cohorts are in different grades during the data collection, making it necessary to capture bias that may be

related to age.<sup>2</sup> Together, these controls provide strong protection against selection and common shocks. Variation in the main predictor thus comes from small changes in the share with depressive symptoms between grades within the same school. I also include a set of individual-level covariates to account any other factors that may not be captured by fixed effects.

Figure A-2 illustrates more concretely the underlying logic of this approach. In a randomly selected school from the analytic sample, about 18% of all grade students reported having depressive symptoms. This overall rate is represented by the black line. Each grade also has its own specific rate of depressive symptoms, represented by white dots, which may differ slightly from the overall mean. For instance, around 16% of all students in the class of 1997 (11<sup>th</sup> graders) reported having depressive symptoms, compared to 19% of students in the class of 1998 (10<sup>th</sup> graders) and 18% of students in the class of 1999 (9<sup>th</sup> graders). The variation in this approach comes from the small deviation of these grade-specific means from the overall school mean. If the contagion hypothesis holds true, then students in grades with a higher proportion of classmates with mental health issues should be more likely to develop depressive symptoms themselves relative to peers in grades with fewer classmates with mental health issues.

While this within-school, between grade approach offers important protection against estimation problems, it is limited because it does not examine whether students are actually interacting with classmates with depressive symptoms. Exploring this is important because contagion effects are thought to operate through engagement with persons with poor mental health. For instance, research often finds that the influence of peers stops at “three degrees of separation” (Christakis and Fowler 2013). Here, I use Add Health’s friendship data to address this issue. More specifically, I estimate conventional logistic regression models, regressing

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<sup>2</sup> Most studies that use this within-school between cohort design rely on repeated cross-sections where different cohorts of students are observed passing through the same grade over time. In the Add Health, we instead have one large cross-section that observe different grades of students simultaneously.

respondents' depressive status at follow-up on their friends' depressive status at baseline. These models also adjust for a vector of individual level characteristics, including socio-demographic measures and respondents' depressive status at baseline. While these models are less rigorous from a causal inference perspective, they are nonetheless instructive because they examine whether depressive symptoms might “spread” across close ties, as postulated by social contagion theory.

## **Results**

### **Descriptives**

Table A-1 shows the descriptive statistics of the variables used in the main analysis. Overall, around 10% of the students in the analytic sample had depressive symptoms during the second wave of data collection, while 11% had depressive symptoms during Wave I. On average, around 17% of students in every cohort had depressive symptoms. 49% of the sample is male, 36% have at least one college educated parent, and 11% were born outside of the United States. In terms of the racial/ethnic makeup, 48% of the sample is White, 21% is Black, 19% is Hispanic, and 11% identify themselves as part of another group.

### **Peer contagion and fixed-effects models**

Table A-2 examines the association between the share of a cohort with depressive symptoms and respondents' own depressive symptoms at follow-up. I begin with an unconditional model and progressively include more controls, including fixed effects and individual level covariates, to account for various sources of bias.

Model 1 confirms a significant, positive relationship exists between cohort and respondent depressive symptoms, as predicted by social contagion theory. More specifically, a one percentage point increase in the share of students with depressive symptoms is associated

with a 3% increase in the odds of having depressive symptoms (e.g.,  $\exp(.030) = 1.03$ ). In model 2, I add school and grade fixed effects into the model. This specification indicates that a one percentage point increase in the share of students with depressive symptoms is now associated with a 7% increase in the odds of having depressive symptoms (e.g.,  $\exp(.070) = 1.07$ ). In model 3, I add respondents' depressive status during baseline. Not surprisingly, the inclusion of this variable reduces the cohort estimate significantly (0.070 to 0.052), since past mental health is generally the stronger predictor future mental health. Nevertheless, the coefficient remains positive and highly significant. In model 4, I add several individual-level control variables that may not be accounted for by fixed effects and prior depression status. This final specification indicates that a one percentage point increase in the share of students with depressive symptoms is associated with a 5% increase in the odds of being depressed (e.g.,  $\exp(0.051) = 1.05$ ). Overall, across all model specification we find that the exposure to peers with depressive symptoms increases student's own odds of developing depressive symptoms, providing compelling support for the contagion hypothesis.

To provide a more intuitive understanding, Figure A-3 plots the predicted probability of depressive symptoms based on the estimates from model 4, with all covariates set at their means. This figure can be interpreted as the expected change in an "average" respondent's probability of depressive symptoms given exposure to different classmates. While the exact effect size varies by level of depression within a cohort, we find that a one SD increase (4 percentage points) in the share of classmates with depressive symptoms is roughly associated with a 1 percentage point increase in the probability of developing depressive symptoms.

### **Peer contagion and conventional models**

Results from logistic regression analyses indicate that having at least one friend with depressive symptoms was positively associated with having depressive symptoms for respondents a year later (Table A-3, Row 1, Model 1:  $\beta=0.464$ ). This relationship persisted after adjusting for socio-demographic characteristics, though it became only marginally significant after controlling for respondents' depressive status at baseline. Analyses that considered the mental well-being of respondents' best friends found an even more pronounced association: even after accounting for baseline depressive status, the odds that respondents would have depressive symptoms increased significantly if their best friends had these same symptoms at baseline (Table A-3, Row 3, Model 3:  $\beta=0.663$ ). Interestingly, this relationship was strongest for same-sex, rather than opposite sex, best friends. After adjustment, the odds of having depressive symptoms at follow-up increased by 282% if a same-sex best friend also had depressive symptoms at baseline (e.g.,  $\exp(1.035) = 2.82$ ). These results, along with those from the fixed effects models, thus offer support for the contagion hypothesis, as respondents' mental well-being was strongly related to their friends' well-being.

### **Peer contagion across sub-groups**

As discussed above, there are also important theoretical reasons to believe that the impact of exposure to peers with depressive symptoms may vary across socio-demographic groups. To examine this further, I re-estimate the main model but also include an interaction term between the share of students with depressive symptoms and different socio-demographic characteristic. It is important to view these results as only suggestive, as sub-groups analyses may be prone to type I error because of multiple hypothesis tests. Nevertheless, these may provide useful information on a subject that has been neglected within the social contagion literature.

For the gender specific models (Table A-4, model 1), we find that the interaction term is essentially 0 and non-significant, indicating that exposure to peers with depressive symptoms has the same association for both boys and girls. These results are somewhat surprising given that some existing research report heterogeneity across gender (Rosenquist, Fowler and Christakis 2011; Eisenberg et al. 2013). On the other hand, they are consistent with some experimental studies that find a relatively uniform effect across gender (Joiner and Katz 1999).

For the race specific models (Table A-4, models 2-4), we find no evidence that the association between classmate and respondent depressive symptoms differs for Whites (compared to non-Whites) and Hispanics (compared to non-Hispanics). On the other hand, there is suggestive evidence that peer contagion may be stronger for Blacks relative to non-Blacks. While this point estimate is only marginally significant ( $p=.09$ ), it is still relatively large and may have important substantive implications. To illustrate these more concretely, I estimate the predicted change associated with going from a cohort with an average level of classmates with depressive symptoms to one that is one SD above average (Figure A-4). Here I find that for black students, this change in classmates is associated with a 3.0 percentage point increase in depressive symptoms, compared to 0.9 percentage points for non-Blacks. This suggests Blacks in particular may be more sensitive to exposure to classmates with depressive symptoms.

For the SES specific model (Table A-4, model 5), exposure to classmates with depressive symptoms is moderated by parental education. To further examine this relationship, I again estimate the predicted change associated with going from a cohort with average depression to one that is one SD above average (Figure A-4). This analysis indicates that a one-SD increase in classmates with depressive symptoms is associated with a 2.2 percentage points in the probability of having depressive symptoms, compared to no change for students from college-

educated households. Thus, while the greater exposure to classmates with depressive symptoms may have no influence advantaged students, it may have a particularly strong influence on those from less educated homes.

### **Exploratory mediational analyses**

In the final part of the analysis, I examine the potential mechanisms that may facilitate the “spread” of depressive symptoms between students (Table A-5). As described above, the three primary mechanisms posited in the literature is (1) social influence, (2) social support received, and (3) social support provided. Separately testing these three factors is challenging because they are highly inter-connected and difficult to measure accurately. Moreover, from a causal inference perspective, this analysis is less rigorous because the temporal relationship between depressive symptoms and these factors is unclear. Nevertheless, it is an important task for researchers and healthcare practitioners to understand the relative importance of the different processes potentially driving contagion effects. With these limitations in mind, I use different proxy measures to explore the role of these three mechanisms.<sup>3</sup>

#### *Social Influence*

To test the social influence mechanism, I first examine the statistical interaction between cohort depressive status and a student’s own sense of independence.<sup>4</sup> The logic here is that students with low levels of independence are more likely to conform and imitate those around them. Thus, if social influence is a mechanism, we should find that it has a particularly pronounced effect on the least independent students. Results in model 1 do not find evidence for

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<sup>3</sup> Many of these proxy measures had a small number of incomplete missing. In order to keep the sample size the same and allow for a comparison of effect sizes, I use imputation with chained equations to fill in missing data for mediators.

<sup>4</sup> The specific item used for this analysis is a question which asked students to rate their own sense of independence from a scale of 1-5, with 1 being the highest and 5 being the lowest.



this hypothesis, as the interaction term between student independence and cohort depressive status is non-significant.

As a second test of the social influence mechanism, I examine students' level of engagement with their friends and classmates. Prior research indicates that depressed adolescents are more likely to withdraw from social activities from friends and classmates (Rubin, Coplan and Bowker 2009). The social influence mechanism predicts that students may see and adopt this behavior, thereby elevating their risk of depression. If this is true, then we should expect to find a diminished contagion estimate if we control for students' level of interaction with friends. To test this, I create binary measures of social interaction with friends and classmates and add these as mediators into the main model. Compared to the base model (model 2a), I find that these factors accounted for essentially none of the social contagion coefficient (model 2b). Taken together, these analyses suggest that the social influence may not be the specific channel through which depressive symptoms are spread.

#### *Social support received*

In models 3a and 3b, I consider the mediating role of social support that students receive from others. In environments with a high share of classmates with depressive symptoms, students may have (or perceive to have) fewer friends and classmates who they can turn to for help and support. If this is true, then controlling for perceived support should theoretically reduce the contagion estimate. To examine this, I create several measures of perceived social support and add them into the main model (model 3b). Consistent with existing studies, I find that all of these estimates are negative and many are statistically significant, indicating that receiving social support is an important protective factor against depressive symptoms. I also find that these variables are important mediators, reducing the contagion estimate by roughly 15% ( $\beta = 0.051$  to

$\beta=0.043$ ). Finally, the contagion coefficient becomes marginally significant with the inclusion of these measures ( $p=0.09$ ). This suggests that exposure to classmates with depressive symptoms may increase the risk of depressive symptoms for respondents by reducing the level of support individuals have (or perceive to have) available to them.

### *Social support provided*

Finally, I consider the level of social support provided to other classmates in model 4 by interacting the cohort measure with a self-rated measure of empathy.<sup>5</sup> While supporting classmates with depressive symptoms may negatively influence a student's own mental well-being, this association should be stronger for the most empathetic students, since they may be the most inclined to provide classmates with help. If this is true, then we should expect to find a significant interaction between cohort depressive status and student empathy. Overall, results do not support this hypothesis, as the interaction term is non-significant.

### **Sensitivity analysis**

The validity of the within school, between grade approach requires that cohort-to-cohort variation in the share of students with depressive symptoms within schools be roughly random. Unfortunately, there is no definitive way to ensure that this requirement is met. Nevertheless, several recent studies have implemented diagnostic tests to assess the plausibility of this randomness assumption (Legewie and DiPrete 2012; Bifulco, Fletcher and Ross 2011). In this section, I report the results from two such tests.

To begin, I conduct a series of balancing tests that examine whether within school variation in the proportion of students with depressive symptoms is correlated with student background characteristics. Null results here provide evidence that the school and cohort fixed

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<sup>5</sup> The specific item used for this analysis is a question which asked students to rate their own sensitivity to other people's feelings from a scale of 1-5, with 1 being the highest and 5 being the lowest.

effect help address systematic sorting along observable characteristics. This is important because some argue that the level of selection on observables provides a useful approximation of the level of selection on unobservables (Altonji, Elder and Taber 2005). To perform these tests, I regress different observable student characteristics on the main predictor variable (i.e., the cohort measure of depressive symptoms) while adjusting for school and grade fixed effects. Overall, Appendix A-1 shows that none of these coefficients here are statistically significant, indicating that, conditional on school and grade fixed effects, variation in the share of students with depressive symptoms is uncorrelated to observed student background characteristics.

Second, following Legewie and DiPrete (2012), I examine whether the within school, across cohort variation in students with depressive symptoms is consistent with a random process using simulations (see Appendix A-2 for details). To do this, I restrict the in-school sample to 9<sup>th</sup> to 11<sup>th</sup> grade students from the 68 schools in the analytic sample. I then randomly assign students to a cohort within the same school and compute the average within-school variation of students with depressive symptoms, keeping the number and size of grades constant. I then repeat this process 1000 times to create a distribution of within-school variation generated exclusively through a random allocation process. I then compare this hypothetical distribution to the observed within-school variation in depression students. The results of this exercise (Appendix A-3) indicate that the observed within-school variation in students with depressive symptoms is consistent a random process. Taken together, these analyses provide suggestive evidence that the within school, between grade/cohort variation in student depressive symptoms is plausibly random.

## **Discussion and Conclusion**

Overall, this paper had three main results. First, I find that exposure to classmates with depressive symptoms is positively related to the odds that a student will have depressive symptoms him/herself, as postulated by the contagion hypothesis. Effect sizes from varied depending on the methods used to test the hypothesis. Fixed-effect models, which leveraged small variation in the share of classmates depressive symptoms in a cohort within the same school, yielded relatively small estimates; a one SD increase in classmates with depressive symptoms at baseline was predicted to increase the probability of depressive symptoms by about 1 percentage point at year later. Standard regression models, on the other hand, found that friends' mental-wellbeing was strongly associated with their own mental well-being a year later. For instance, having a same-sex best friend with depressive symptoms at baseline was correlated with a 282% increase in the odds of having depressive symptoms at follow-up. Given that respondents are likely to be influenced emotionally by close friends compared to random classmates, it is not surprising that other papers report much larger estimates. Nonetheless, the fact that both set of analyses converge around the same substantive conclusion offer compelling support for the contagion hypothesis.

Second, this study finds that the extent to which depressive symptoms are contagious varies by a student's socio-demographic background. In gender specific models, I find that the contagion estimate is roughly the same for both adolescent boys and girls. This result is somewhat surprising given that much existing research on contagion and depression report differential gender effects. Nevertheless, this is consistent with many small scale experimental studies which find that men and women are equally susceptible to the effects of emotional contagion (Joiner and Katz 1999). In contrast, I find that black students and low-SES students

may be more vulnerable to exposure to students with depressive symptoms than their peers. It is unclear why social contagion effects appear larger for socially disadvantaged groups. One possibility is that disadvantaged adolescents rely more heavily on social relationships than their counterparts as a response to the additional stressors they encounter because of their marginal status. This is an open question that I encourage future researchers consider. Overall, these results highlight the importance of moving beyond average effects and considering variation across groups.

Third, this study sheds light on the social mechanisms that may drive the spread of depressive symptoms. The existing literature suggests that social influence (or the adoption of attitudes and behaviors), a lack of social support, and providing social support to others may be three reasons why exposure to classmates with depressive symptoms negatively impact other students' mental health. A mediational analysis confirmed that a lack of social support was indeed an important mechanism, accounting for a sizable portion of the contagion estimate. On the other hand, social influence and providing social support mediated virtually none of the social contagion estimate. These results suggest that exposure to classmates with depressive symptoms result in worse mental health because it makes others feel disconnected or unsupported by those around them. Prior work suggests that this may emerge because depressed people are unable to provide the same level of social support as non-depressed individuals. Others also argue that classmates with depressive symptoms may simply be more unpleasant to interact with than non-depressed peers. Ultimately, this suggests that lower quality social relationships partially explain how depression can spread among adolescents.

This study has several important limitations. The most important one relates to the cohort measure of depressive symptoms. Unlike the in-home sub-sample, which uses a well-validated

instrument (CES-D), the in-school sample uses a single question to measure mental well-being. Thus, it is likely that depression is measured with less precision among this larger population. Ideally, we would want to use the same 19 item measure to construct the cohort-level depressive symptom measure, but this information is unfortunately not available. Still, there is some work that suggests that single-item questions can still measure depression relatively well (Young et al. 2015). Furthermore, measurement error is reduced because the in-school sample surveyed almost all students within a school. Therefore, the measure used in this study, while imperfect, arguably produces a reasonable approximation of the share of students who had depressive symptoms within a cohort. Second, the mediational analysis uses admittedly crude and indirect measures to test for mechanisms. For example, it was assumed that students adopted social withdrawal behavior because of exposure to classmates with depressive symptoms. A more accurate approach might have asked students whether they were withdrawing from social activities because their friends were doing so. Unfortunately, these types of measures were not available in the dataset. Thus, it is important to view those results as suggestive rather than directly causal. A third limitation relates to the generalizability of these results. While this study uses a nationally representative sample that is much larger than most prior studies, I only include students in grades 9 to 11 in the analysis. Because of this, these results should be viewed as only applying to older adolescents rather than all youth.

In future work, I plan to continue using the rich friendship data in the Add Health to explore the contagion hypothesis. One line of analysis will descriptively examine the characteristics of friendship networks in the grades with a higher share of students with depressive symptoms. For instance, do these additional students with depressive symptoms have connections with non-depressed classmates or are they socially disconnected from their peers?

The former is consistent with the contagion theory, as it suggests that these “random” classmates interact with others and potentially “spread” their symptoms. The latter, on the other hand, implies a more indirect link; these classmates with depressive symptoms may, for instance, may potentially create a classroom environment that negatively impacts classmates’ well-being. A second analysis will explore whether the role of respondents’ friendship networks more formally. Here, the plan is to construct individual-level network characteristics, such as percentage of friends with depressive symptoms and total number of friends with depressive symptoms, and enter these as mediators into the main models (i.e., the within school between cohort analysis).

In conclusion, this paper provides some of the strongest evidence that exposure to individuals with depressive symptoms worsens one’s own mental well-being. This association may be particularly pronounced for low-SES and black students and results, in part, because of the lower level of social support students perceive they have from classmates with depressive symptoms. Therefore, I argue that researchers should continue examining the linkage between social relationships and mental well-being, particularly focusing on mechanisms and effect heterogeneity by sub-groups. Developing this more nuanced understanding of social ties will assist policy makers in developing more effective interventions for individuals with or at risk for depression.

Table A-1: Descriptives

	Mean	SD
<b>Outcome Variable</b>		
Depression at Wave II	0.10	0.30
<b>Predictor Variable</b>		
% cohort w/ depressive symptoms	17.16	3.67
<b>Controls</b>		
Prior depression	0.11	0.31
Male	0.49	
Age	16.51	1.00
Non-Hispanic White	0.48	
Non-Hispanic Black	0.21	
Non-Hispanic other race	0.11	
Hispanic	0.19	
Foreign born	0.11	
Parents completed college	0.36	
9 <sup>th</sup> grade	0.30	
10 <sup>th</sup> grade	0.37	
11 <sup>th</sup> grade	0.34	

Note: N = 7,669. Figures are unweighted



Table A-2: Fixed-effect models results

	(1)	(2)	(3)	(4)
	Unadjusted	+School and grade FE	+Prior depression	+Covariates
% cohort w/ depressive symptoms	0.030*** (0.010)	0.070*** (0.018)	0.052** (0.021)	0.051** (0.022)
Prior depression status			2.461*** (0.108)	2.409*** (0.112)
Male				-0.258** (0.113)
Age				0.213** (0.090)
Born in US				-0.094 (0.231)
Parents completed college				-0.231* (0.130)
Race (ref=White)				
Black				-0.007 (0.199)
Hispanic				0.205 (0.216)
Other race				0.395* (0.240)

Note: Model 1 is unconditional and includes no additional controls, while models 2-4 include school and grade/cohort fixed effects. Robust standard errors clustered at the school level in parentheses. \*\*\* p<0.01, \*\* p<0.05, \* p<0.10

Table A-3: Conventional regression model results

	(1) Unconditional	(2) +Socio- demographics	(3) +Baseline depressive status
At least one friend at risk of depression at baseline (N=4,097)	0.464** (0.184)	0.416** (0.197)	0.381* (0.200)
At last one best friend (either male or female) at risk of depression at baseline (N=3,064)	0.750*** (0.285)	0.725** (0.290)	0.663** (0.321)
Best same-sex friend at risk of depression at baseline (N=2,267)	1.016*** (0.303)	0.948*** (0.315)	1.035*** (0.353)
Best opposite-sex friend at risk of depression at baseline (N=1,246)	0.108 (0.477)	0.134 (0.462)	-0.130 (0.491)

Each row presents log-odds from separate logistic regression models examining the association between friends' depressive status at baseline and respondents' depressive status at follow-up.. Friends who were nominated first friend nominated first. Robust standard errors clustered at the school level in parentheses.  
\*\*\* p<0.01, \*\* p<0.05, \* p<0.10

Table A-4: Sub-Group Analysis

	(1)	(2)	(3)	(4)	(5)
% cohort w/ depressive symptoms	0.053** (0.027)	0.037* (0.022)	0.048** (0.024)	0.062* (0.034)	0.069*** (0.023)
Male	-0.173 (0.596)				
Male * cohort	-0.005 (0.033)				
Black		-1.467* (0.866)			
Black * cohort		0.078* (0.046)			
Hispanic			-0.465 (1.300)		
Hispanic * cohort			0.031 (0.076)		
White				0.142 (0.786)	
White * cohort				-0.016 (0.042)	
Parents college educated					0.969** (0.454)
Parents * cohort					-0.067** (0.027)

Note: Each model includes a school and grade fixed effect, along with a respondents' prior depression status and the socio-demographic covariates listed in Table 1. Robust standard errors clustered at the school level in parentheses. \*\*\* p<0.01, \*\* p<0.05, \* p<0.10

Table A-5: Mediation Analysis

	(1) Social Influ- ence	(2a)  Social Influence	(2b)  Social Influence	(3a)  Support Received	(3b)  Support Received	(4) Support t provid ed
% cohort w/ depressive symptoms	0.086 (0.066)	0.051** (0.022)	0.051** (0.022)	0.051** (0.022)	0.043* (0.026)	0.116 (0.076)
Student independence	-0.025 (0.308)					
Cohort * independence	-0.009 (0.018)					
Went to a friend's home			-0.162 (0.158)			
Met a friend afterschool			0.040 (0.191)			
Met a friend on the weekend			-0.050 (0.156)			
Discussed a problem with a friend			0.526** * (0.168)			
Talked on the phone with a friend			-0.295 (0.212)			
Has at least one friend			0.067 (0.212)			
My friends care about me					-0.129* (0.069)	
I feel socially accepted					- 0.297** * (0.106)	
I feel loved and wanted					- 0.830** * (0.115)	
I feel close to the people in my school					-0.114 (0.070)	

I feel like I am part of the school	-	
	0.219**	
	*	
	(0.081)	
Student empathy		0.214
		(0.340)
Cohort * empathy		-0.016
		(0.017)

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Note: All coefficients and standard errors are from logistic regression models that include school and grade fixed effects, along with the covariates listed in Table 1. Missing values on meditating variables were imputed using chained equations. Robust standard errors clustered at the school level in parentheses.  
\*\*\* p<0.01, \*\* p<0.05, \* p<0.10

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Figure A-1: Add Health data structure and sample exclusions

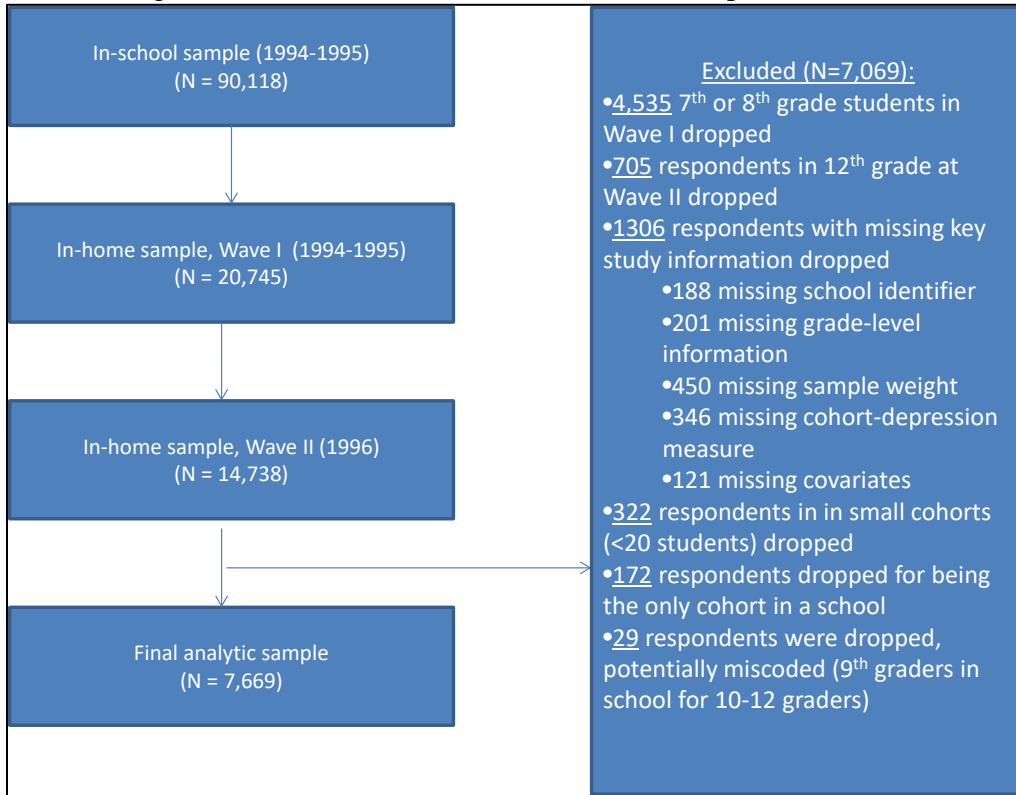


Figure A-2: Variation in the share of students with depressive symptoms within a school during Wave I, by cohort/grade

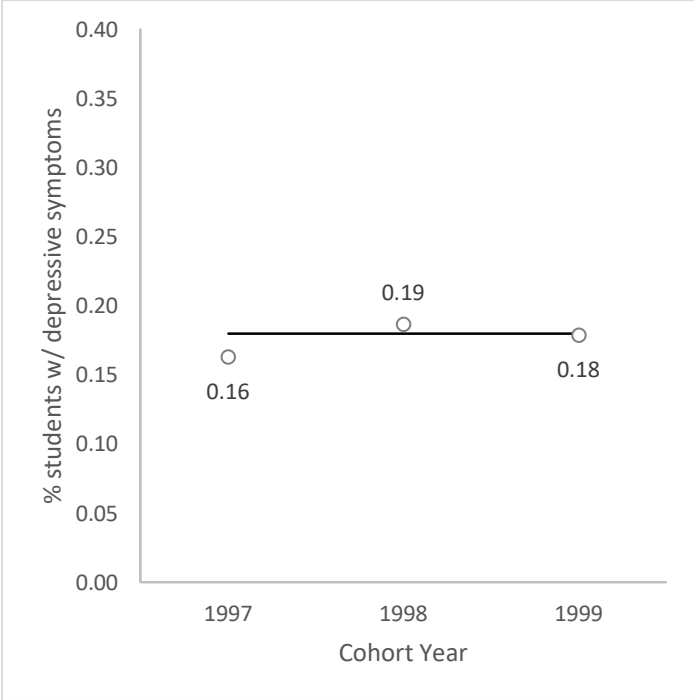
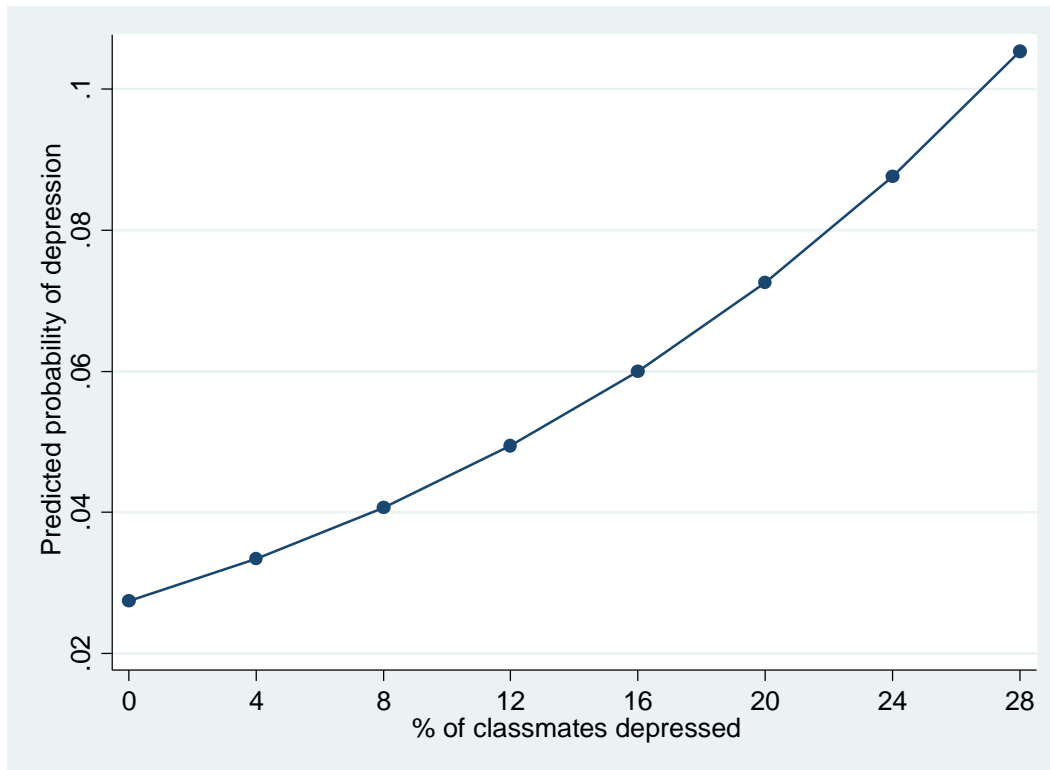


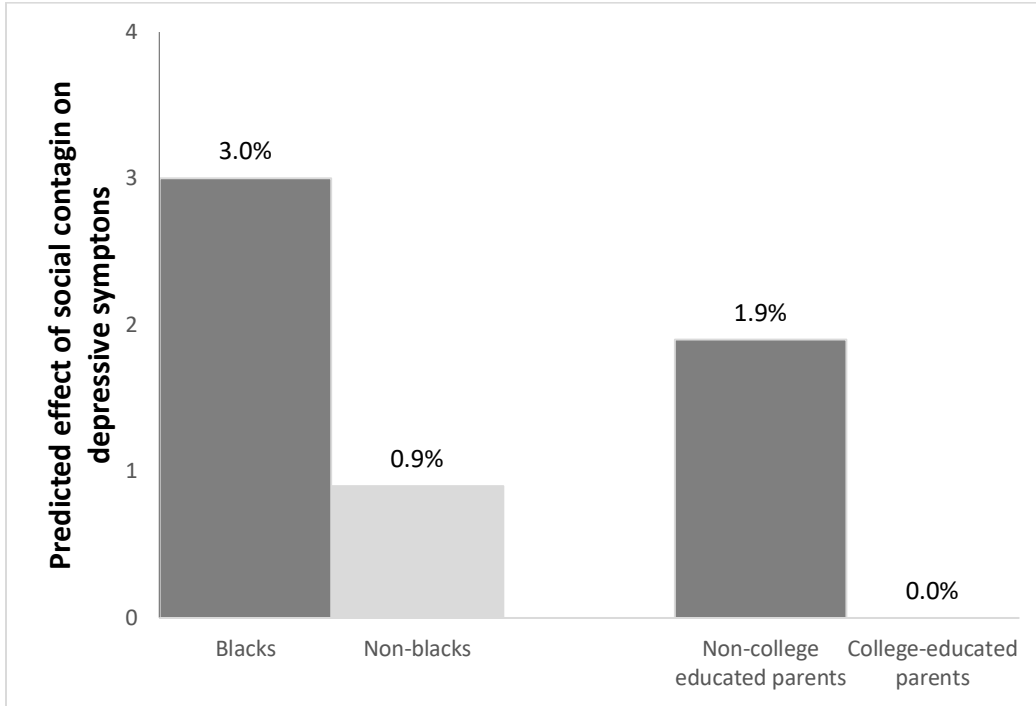
Figure A-3: Predicted probability of depression across different levels of exposure to classmates with depressive symptoms



Note: These predicted probabilities come from Table 2, model 4. All covariates are set at their means.



Figure A-4: Predicted effect of a one-SD increase in classmates with depressive symptoms, by race and SES



Note: These predicted probabilities come from table 4, models 2 and 5, respectively. All covariates are set at their means.

Appendix Table A-1: Balancing Test

Dependent Variable	% cohort with depressive symptoms	Obs
Male	-0.30 (0.240)	7,669
PPVT score	-0.19 (0.547)	7,345
Age	0.61 (0.435)	7,669
Family income	-0.52 (0.562)	5,790
Born in US	-0.05 (0.130)	7,669
Number of siblings	-0.71 (0.675)	7,619
White	-0.08 (0.190)	7,669
Black	0.11 (0.141)	7,669
Hispanic	0.12 (0.136)	7,669
Other race	-0.15 (0.136)	7,669
Parents college educated	-0.12 (0.285)	7,669
Parents born in US	-0.07 (0.151)	6,653
Single parent household	-0.00 (0.241)	6,652

Note: The figures in the second column are coefficients from separate linear regression models that include the share of students with depressive symptoms within a cohort, along with school and grade fixed effects and the dependent variables listed in the first column. The PPVT is a common standardized test by researchers to assess a student's scholastic ability. All variables are measured at wave 1 of the Add Health study. Figures in parentheses are standard errors robust to clustering at the school level. \*\*\* p<0.01, \*\* p<0.05, \* p<0.10

## Appendix A-2: Simulation of Random Assignment

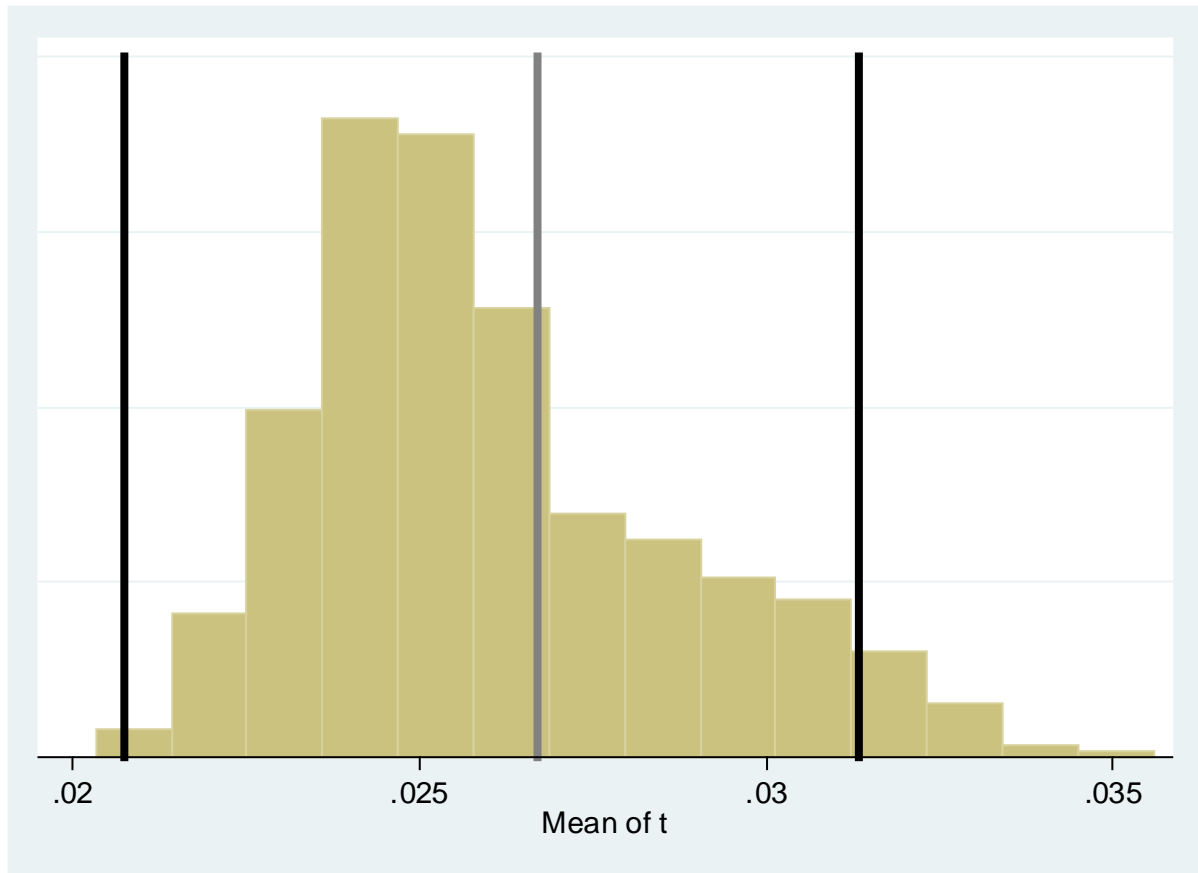
Following the procedure outlined in Legewie and DiPrete (2012), I examination whether the within-school variation in students with depressive symptoms is consistent with a random allocation process. To do this, I randomly assign studies to grades within their schools, keeping the size of the school and grades constant. I then computed the following statistic

$$t_j = \sqrt{\frac{1}{n_j} \sum_{k=1}^{n_j} (\bar{x}_{jk} - \bar{x}_j)^2}$$

where  $j$  and  $k$  are indices for schools and grades;  $\bar{x}_j$  is the average share of students with depressive symptoms within school  $j$ ;  $\bar{x}_{jk}$  is the average share of students with depressive symptoms in cohort  $k$  and school  $j$ ;  $n_j$  is the of grades in school  $j$ . This statistic thus captures the average standard deviation of cohort means from the school mean.

This process is repeated 1000 times, resulting in a sampling distribution of the t-statistic that is the result of a purely random allocation. The observed t-static is then compared to this distribution as an informal way to test whether it is consistent with a random process.

Appendix Figure A-3: Observed within-school variation compared with variation generated through random simulations



Note: This graph shows the average within-school variation in the share of students with depressive symptoms in the within school sample (vertical gray line) along with the sampling distribution of this statistic generated through 1,000 random simulations (histogram). Vertical black lines indicate 95% confidence intervals from these simulations.

### **Chapter 3 : Gender, social relationships, and depressive symptoms: Revisiting the reactivity hypothesis**

#### **Introduction**

Gender differences in depression are well documented. Beginning in early adolescence, girls are roughly twice as likely as boys to develop depression (Nolen-Hoeksema and Girgus 1994). In the United States, for instance, 4.0% of boys aged 12-17 were depressed in 2009-2012, compared to 7.4% of girls (US Centers for Disease Control and Prevention 2014). This gender gap has broad public health implications, as depression is a major risk factor for a variety of health problems, including suicide, illicit drug use, chronic conditions, and even premature death (Franklin et al. 2017, Moussavi et al. 2007, Stone et al. 2012, Walker, McGee and Druss 2015).

Within health and social science research, one prominent theory posits that gender differences arise in part because girls may be more sensitive to the depressogenic effects of stressful life events. This may be particularly salient for events involving close social ties, such as family members, as girls are believed to place greater importance on interpersonal relationships (Chodorow 1999, Cyranowski et al. 2000, Gilligan 1982, Hankin and Abramson 2001, Hyde, Mezulis and Abramson 2008, Nolen-Hoeksema 2001, Rudolph 2002). As a result, when confronted with similar levels of interpersonal stress, girls may experience higher levels of distress compared to boys, leading to higher levels of depression.

Numerous studies have tested this so-called reactivity hypothesis, but results remain mixed. While many report that social ties have a stronger association with mental well-being for girls, others have failed to replicate this finding (see Rueger et al. 2016 and Rosenfield and Mouzon 2013 for recent reviews of the literature). Much of the existing research, however, is

limited in at least two ways. First, most prior studies rely on small, non-representative, cross-sectional data, limited measures of social relationships. More importantly, nearly all employ conventional regression approaches, which account for confounders by conditioning on observable characteristics. While this approach is useful for examining the conditional association between variables, the results are highly sensitive to the variables included in the models (Greenland 1989). Because of this, traditional regression models may be inappropriate for drawing conclusions about the reactivity hypothesis.

Beyond methodological issues, few studies using representative data have actually tested the underlying mechanism implied in the reactivity hypothesis—that is, girls’ greater responsiveness to interpersonal stress compared to men. In almost all existing studies, researchers have focused on the statistical interaction between gender and social relationship, with a significant finding here being viewed as confirmation of the hypothesis. Yet, the interaction term only assesses whether social relationships have a stronger association with mental health for girls than boys; it does not directly examine whether girls’ greater responsiveness accounts for these trends. To empirically test the latter claim, researchers must perform a mediational analyses to assess how much of the gender \* social relationship interaction can be accounted for by gender differences in reactivity. Relatively few studies have conducted such analyses, leaving the critical question of *why* unanswered.

The overarching goal of this study was to revisit the reactivity hypothesis, focusing specifically on these two limitations. To address methodological shortcomings, we estimated first-difference models (Allison 2009), examining how changes in the quantity and quality of social relationships related to changes in depressive symptoms within a person. From an empirical standpoint, this approach is an important improvement from prior studies because the

bias stemming from time-invariant confounders are eliminated. To address the gap in mechanisms, we conducted a mediational analysis, examining whether differences in girls' responsiveness to changes in social ties explained why the association between social relationships and depressive symptoms might be stronger for them than boys. By using a more appropriate analytic approach and exploring underlying mechanisms, this paper provided a more rigorous and comprehensive assessment of the reactivity hypothesis.

In the following sections, I begin by outlining theoretical arguments for why girls may be more reactive to interpersonal stress compared to boys. I then review existing empirical studies that investigate the relationship between social ties, gender, and adolescent mental health, focusing specifically on familial, peer, and school relationships. Following this, I estimate a series of individual fixed-effect models that examine whether the association between various social ties and depressive symptoms differs by gender. I then conduct a mediational analysis to explore whether differences in responsiveness explain why social ties may be more strongly related to mental health for girls than boys. Finally, I conclude with a discussion about the implications about these findings.

### **Theoretical background**

Social theorists from a broad range of disciplines have argued that compared to males, females place greater value on intimate social ties, emotional expression, inter-connectedness, and nurturance. As a result, events that strain social ties are believed to illicit a stronger negative response for girls than boys. Broadly speaking, theorists attribute this alleged gender difference to two overlapping but distinct forces: socialization and social structures. It is important to note that differences in how families raise children and society organizes opportunities do not reflect “natural” differences between boys and girls. Instead, gender scholars argue that they are a direct

result of an institutionalized system that ultimately legitimizes and perpetuates gender-based inequality (Ridgeway and Correll 2004).

### *Socialization theories*

Socialization theories posit that girls' greater responsiveness to social ties stem from differences in child-rearing practices during early childhood. From an early age, parents raise boys in ways that are consistent with traditional notions of masculinity, including teaching them to be aggressive and competitive. In contrast, girls are taught to constrain these traits and instead focus on empathy and nurturance, which enable them to form and maintain close relationships with others (Gilligan 1982; Chodorow 1999). These gender norms are subsequently reinforced throughout the life course by a number of sources, including family, friends, significant others, mass media, and broader social environments, such as schools and neighborhood contexts (Ridgeway and Correll 2004). These norms often become so deeply engrained that they ultimately come to define individuals' self-identity and outlook on life. Gillian (1982), for example, claims that differences in socialization lead women to develop a sense of morality centered on caring for others.

### *Structural theories*

Structural accounts stress differences in the social positions women and men generally hold, which require different skill sets and reward different type of behaviors. For instance, while women are more likely to enter occupations where caring for others and "emotional labor" are needed (e.g., nursing), men enter into "thing-oriented" fields (e.g., engineering) where interpersonal skills are thought to be secondary (Xie and Shauman 2003). Within the family, women are also more likely than men to serve as the primary caregiver to children, even if they are employed in the labor market (Bianchi 2000), helping them meet their emotional needs by



providing care and affection. Men, on the other hand, have traditionally served as the breadwinner in the family, though this dynamic has changed dramatically over the past several decades (Bianchi 2000). Thus, because of the gendered division of labor in the home and labor market, women often occupy social positions that are more likely to require emotional and interpersonal skills. Some argue that these structural constraints also reinforce gender differences in behavior through gendered role expectations and gendered skill development (Eagly, Wood and Diekmann 2000). For instance, young girls may see women entering “people-oriented” occupations at disproportionate rates, decide that this is the only appropriate career for them, and seek to develop their interpersonal skills.

### *Social ties and adolescence*

The effects of these social and structural processes vary by age and may be especially pronounced during adolescence. The pressure to adhere to gender norms becomes amplified during adolescence, as youth begin to mature physically and become romantically involved with one another (Hill and Lynch 1983). There is some evidence that this process may be particularly true for social relationships. For example, while girls already spend more time with friends than boys prior to puberty, some work shows that the gap grows even wider during adolescence (Raffaelli and Duckett 1989). Moreover, some studies suggest that gender differences in social positions emerge and become most salient during adolescence. For instance, the science gender gap is largest during the middle and high school years, as girls begin losing interest in “thing-oriented” fields and gain interest in “people oriented” fields at a rate much faster than boys (Legewie and DiPrete 2014). Taken together, this suggests that gender differences in the importance of social relationships may be especially salient during adolescence.

## **Prior empirical studies**

Existing research on youth and the association between social ties and mental health has focused on adolescents' relationships with their parents, peers, and school environment, respectively. Below I review these three areas.

### *Parental relationships*

Adolescence is often described as a period during which the importance of the family diminishes, as young people begin assuming greater control over important life choices. Nonetheless, a large body of research still finds the parent-child relationship to be one of the most important determinants of an adolescent's psychological functioning (Steinberg and Morris 2001). A recent meta-analysis shows that two mechanisms in particular drive this relationship (Yap et al. 2014). The first is parental warmth and support. Studies suggest that parents who excessively criticize their children and disapprove of their actions undermine the development of their self-esteem and sense of independence, elevating their risk for depression. In contrast, parental social support—characterized by love, care, and affection—has been shown to protect adolescents from adverse mental conditions such as depression (McLeod, Weisz and Wood 2007). The second is parental control. Research demonstrates that adolescents whose parents excessively regulate their attitudes and behaviors have a decreased sense of autonomy and control over their lives, which may contribute to a higher risk of depression (Yap et al. 2014).

Fewer studies examine whether the association between parents and children and depressive symptoms differs by gender, though existing work supports the reactivity hypothesis. Longitudinal studies have generally found girls to be more reactive to parental support than boys (Landman-Peeters et al. 2005; Ge et al. 1994; Meadows, McLanahan and Brooks-Gunn 2007; Meadows 2007; Windle 1992; Vaughan, Foshee and Ennett 2010; Rueger et al. 2014). Some

speculate that parental support may matter more when parents and children are the same gender, as adolescents more commonly identify with a same-gender parent. That is, a father's support is believed to be more strongly associated with well-being for boys, while a mother's support is predicted to be more strongly related to girls' well-being. Empirical studies exploring these predictions typically only find support for this same-gender hypothesis for the mother-daughter dyad (Meadows 2007; Rueger et al. 2014; Vaughan, Foshee and Ennett 2010; see Branje et al 2010 for an exception), again suggesting that girls may be more sensitive to the effects of parental support.

### *Peer relationships*

Along with parental relationships, a significant body of research has examined the association between peer ties and adolescent mental health. The bulk of these studies focus on self-reported perceptions, and nearly all find that adolescents who perceive higher levels of support and affection from friends report lower levels of depression (see Rose and Rudolph 2006 for a review). This association is not surprising, as peer relationships take on increasing importance during adolescence and becomes arguably the most important predictor of mental well-being during this time. Research on the association between social network characteristics (e.g., popularity) and mental health, though less common, similarly highlight the importance of peer relationships. Ueno (2005), for instance, finds that the total number of friends an adolescent has is the single best social network predictor of depression. Others also find that popularity, network centrality, and status within a network also predict mental health, though the size of the association was generally small (Ueno 2005; Oldenburg 1997). Overall, existing work on adolescents generally shows that the quality and quantity of peer relationships is positively related to psychological well-being.

An important exception to this trend is romantic relationships. Existing work here finds that adolescents in romantic relationships are actually at greater risk of depression than their comparable peers who remained single (Joyner and Udry 2000; Meier 2007). Part of this may be due to the volatile nature of adolescent romantic relationships: dating during adolescence is often characterized by conflict and dramatic emotional swings. These relationships also tend to be relatively short, forcing adolescents in romantic relationships to cope with the psychological stress of a break up. In fact, much work speculates that it is the process of breaking up--rather than being in a relationship per se--that drives the link between dating and adolescent depression (see Collins, Welsh and Furman 2009 for a review).

Few studies explore heterogeneous effects, but existing work also suggests that the association between peer relationships and mental well-being is stronger for girls than boys. Several studies find that perceived peer support, friendship quality, and social network factors (e.g., popularity) protect girls more than boys against mental health problems such as depression (see Rose and Rudolph 2006 for a review). Studies on differential effects of adolescent romantic relationships echo these findings. In their influential study, Joyner and Udry (2000), find that the negative association between adolescent dating and depressive symptoms was significantly stronger for girls than boys. Similarly, Meier (2007) finds that adolescent sexual intercourse was more strongly related to mental health for girls. Overall there is limited but suggestive evidence that girls rather than boys are more sensitive to the effect of peer relationships.

### *School relationships*

It is widely recognized that identifying with and being connected to a broader community can have beneficial effects on an individual's mental health (Kawachi and Berkman 2001). In the case of adolescents, schools are often one of the most important institutions in their lives, as they

spend most of their time in these settings. Prior studies most commonly focus on the concept of school connectedness—or the extent to which a student feels accepted, valued, and respected in school—finding a positive relationship between it and student mental well-being (Bond et al. 2007; McNeely and Falci 2004; Shochet et al. 2006). For instance, a recent meta-analysis finds that higher levels of school connectedness is associated with lower levels of suicidal thoughts and attempts among adolescents (Marraccini and Brier 2017). Some work suggests that support and encouragement from teachers may be an important aspect of adolescents’ relationship with their school environment, potentially protecting them against mental health problems and risky behaviors, such as drug use (Resnick et al. 1997; Colarossi and Eccles 2003; Suldo et al. 2009).

Unlike the research on family and peer relationships, existing studies on differential effects of school ties seem to suggest that girls and boys are equally responsive. Four studies report that the association between school connectedness and mental well-being was similar for boys and girls (Shochet et al. 2006; Millings et al. 2012; Loukas, Ripperger-Suhler and Horton 2009; Wit et al. 2011). Similarly, at least three studies find that students’ relationship with important school actors, such as teachers, has a similar impact on girls and boys (Colarossi and Eccles 2003; Wit et al. 2011; Suldo et al. 2009). Thus, the few studies on this topic suggest that school relationships may be an important exception and affect boys and girls in similar ways.

### *Summary and limitations*

In sum, a large body of research has investigated the association between adolescents’ relationship with parents, peers, and school environment and their mental health, generally finding a positive relationship between the two. Fewer studies have examined heterogeneous effects across gender, though existing work suggests that girls are more sensitive to the effects of parental and peer ties.

However, most of the studies investigating the reactivity hypothesis fail to address important confounders because they rely on cross-sectional research designs. Some studies attempt to address this issue by using longitudinal data and including a lagged measure of depressive symptoms, which is assumed to capture some of the unmeasured heterogeneity between respondents (Wooldridge 2015). While this approach is an improvement compared to traditional cross-sectional designs, it still may result in biased estimates if there are unmeasured time-invariant factors that are related to social relationships and depressive symptoms. To address these shortcomings, I use fixed effect models to examine the association between changes in social relationships and changes in depressive symptoms within the same person. Because variation is based on changes, all the factors that remain constant within a person do not impact estimates (Halaby 2004).

Further, none of the studies reviewed above directly investigate the mechanism implied by the reactivity hypothesis—that is, girls’ greater responsiveness compared to boys. Instead, they focus on the statistical interaction between gender and social relationships. However, a significant interaction here only indicates that social ties are strongly related to well-being for girls than boys; it does not directly signify that girls are more responsive. As part of this study, I examine the interaction between gender and social relationships and conduct a mediational analysis to investigate why girls might be more sensitive to social ties.

## **Methods**

### *Analytic sample*

This study examined data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative, prospective study of American adolescents. Respondents were selected from 80 high schools and 52 middle schools and

interviewed in 1994-1995 (Wave I) and 1996 (Wave II). Details about survey design and data collection have been discussed elsewhere and were approved by the Institutional Review Board at the University of North Carolina at Chapel Hill (Chen and Chantala 2014). Written informed consent was obtained for all participants.

To create the analytic sample, we retained respondents who were (a) interviewed in both Wave I to Wave II; (b) had survey weights available; (c) had depressive symptoms and social relationship data available at both waves of data collection. As shown in Table B-1, the percentage of respondents lost due to missing information for social relationships varied from 0% to 34%. Respondents who were males, racial/ethnic minorities, low-SES, and older in age were more likely to have missing information related to social relationships (Appendix B-1). Differences were generally small, though low-SES and racial minorities had significantly more missing information related to their fathers.

### *Measures*

All measures used in the analysis were change scores. These were computed by subtracting values at Wave II from values at Wave I, capturing the change in a particular item over a one year period. All measures were also standardized with a mean of 0 and standard deviation (SD) of 1 to facilitate interpretation. A more detailed description of all measures is available in Appendix B-2.

### *Change in depressive symptoms*

The main outcome of interest was change in depressive symptoms. The Add-Health study administered a modified, 19-item version of the Center for Epidemiologic Studies' Depression (CES-D) scale, a reliable, well-validated instrument used to measure depressive symptoms Radloff 1977. Responses to these 19 questions ranged from 0 to 3 (0 = never or rarely; 3 = most

or all of the time). After reverse coding appropriate items, responses to all 19 questions were summed at each wave to form a depressive symptoms index ( $\alpha=0.86$  at Wave I,  $\alpha=0.87$  at Wave II).

#### *Changes in family relationships*

Three types of family ties were considered in this study. The first was respondents' relationship with their mothers. Respondents were asked to rate the relationship with their mothers along four dimensions: closeness, warmth, communication, and overall quality. These four items were summed at each wave ( $\alpha=0.85$  at Wave I,  $\alpha=0.86$  at Wave II) to form an index capturing the overall quality of a respondent's relationship with of his/her mother. The second was respondents' relationship with their fathers. Respondents were asked to rate their relationships with their fathers along the same four dimensions as their mothers. These four items were summed at each wave ( $\alpha=0.89$  at Wave I,  $\alpha=0.89$  at Wave II) to form a father relationship index. The third was family relations more generally. Respondents were asked to rate the extent to which their families understood, paid attention, cared, and had fun with them. These four items were summed at each wave ( $\alpha=0.76$  at Wave I,  $\alpha=0.77$  at Wave II) to form a family relationship index.

#### *Changes in peer relationships*

Four types of peer relationships were also examined. The first was the total number of friends. Respondents were asked to name their best five male and female friends, respectively, during both waves of data collection. The total number of nominations was summed at each wave and had a minimum value of 0 (for adolescents who had no friends) and a maximum value of 10. The second was support from friends. Respondents were asked how much they thought their friends cared about them on a scale of 1 to 5 (1 = not at all; 5 = very much). The third was



engagement with friends. Respondents were asked to describe how often they hung out with their friends in the past week on a scale of 1 to 4 (1 = not at all; 4 = 5 or more times). The fourth was romantic relationships. Adolescents were asked whether they were romantically involved with someone over the past 18 months (0 = No, 1 = Yes). Unlike the previous measures, dating was a binary item, resulting in three possible change values (-1 = went from dating to being single; 0 = remained single or remained dating; 1 = went from single to dating).

#### *Changes in school relationships*

Finally, two types of relationships within schools were examined. The first was school connectedness. Respondents were asked to describe how close they felt to people at school, how happy they were to be at school, and whether they felt like they were part of the school. These three items were summed at each wave to form a school connectedness index ( $\alpha=0.77$  at Wave I,  $\alpha=0.78$  at Wave II). The second was respondents' relationship with their teachers. Respondents were asked to rate how fairly teachers treated students, how well teachers got along with them, and how much teachers cared about them. These three items were summed at each wave to form a teacher relationship index ( $\alpha=0.61$  at Wave I,  $\alpha=0.61$  at Wave II).

#### *Responsiveness*

During both data waves, respondents were asked to rate the extent to which they felt "loved and wanted" on a 1 to 5 scale, with 1 representing the lowest level of perceived love and affection and 5 representing the highest level. Scores from Wave II were subtracted from Wave I, resulting in a change score that sought to capture respondents' responsiveness to changes in social relationships. The underlying logic of this measure was that improvements in social ties would bolster mental health by increasing the extent to which adolescents felt loved and wanted. However, because girls are hypothesized to be more reactive to their social ties, this increased

feeling of perceived affection should have a stronger protective effect for girls. Similarly, interpersonal stress may result in poorer mental health by decreasing the degree to which adolescents feel loved, but this adverse effect was predicted to be stronger for girls.

*Time-varying control variables*

The analysis also adjusted for several time-varying factors that may confound the association between changes in social ties and changes in depressive symptoms (See Appendix B-2 for information about how these measures were created). Within the school context, this study controlled for changes in school attendance and changes in academic performance. Within the home context, this study controlled for changes in household size, changes in home location, and changes in parental employment. We used multiple imputation to handle missing data on control variables, generating 5 complete datasets using chained equations to simulate missing values (White, Royston and Wood 2011). Analyses that imputed for missing information on social ties (not shown here) also yielded similar results.

*Statistical analyses*

The main analysis proceeded in three general. First, we estimated a series of first-difference models that examined the association between changes in the number and quality of social relationships and changes in depressive symptoms for the same adolescent over time. In essence, these examine how changes in the number and quality of social relationships relate to changes in depressive symptoms for the same adolescent over time. By focusing on variation within each respondent, this approach eliminates the influence of time invariant confounders (e.g., race), both observed and unobserved. To estimate these models, I begin by first subtracting all variables at time 2 by all their values at time 1:

$$Y_{iT2} - Y_{iT1} = \beta_1(SR_{iT2} - SR_{iT1}) + (Cov_{T2} - Cov_{T1}) + (\varepsilon_{T2} - \varepsilon_{T1}) \quad (1)$$

which can re-written as

$$\Delta Y_i = \beta_1 (\Delta SR_i) + (\Delta Cov_i) + (\Delta \varepsilon_i) \quad (2)$$

where  $\Delta Y$  is the change in CES-D score for student  $i$ ,  $\Delta SR_i$  is the change in some type of social relationship (e.g., change in relationship with mother),  $\Delta Cov_i$  is the change in a vector of time-varying control variables, and  $\Delta \varepsilon_i$  is the change in the error term over time.

Second, we assessed the reactivity hypothesis by allowing the social relationships estimate to vary across gender. This was accomplished by adding an interaction term (change in social relationship \* gender) into the model:

$$\Delta Y_i = \beta_1 (\Delta SR_i) + \beta_2 (\Delta SR_i * Girl_i) + (\Delta Cov_i) + (\Delta \varepsilon_i) \quad (3)$$

Note that the main effect estimate for girl is omitted from equation 3 because it is a time invariant measure and is “differenced” out. Typically, estimating an interaction term without the corresponding main effect estimate results in model mis-specification. In this case, however, the interaction between social relationships and gender ( $\beta_2$ ) can still be properly estimated without the inclusion of this time-invariant measure (Allison 2009).

Third, we conducted a mediational analysis to test whether gender differences in responsiveness accounted for the differential effect of social relationships, as postulated by the reactivity hypothesis. This was accomplished by including an additional interaction term (changes in feeling loved and wanted \* gender) into the model:

$$\Delta Y_i = \beta_1 (\Delta SR_i) + \beta_2 (\Delta SR * Girl) + \beta_3 (\Delta LW_i) + \beta_4 (\Delta LW_i * Girl_i) + \beta_5 (\Delta Cov_i) + (\Delta \varepsilon_i) \quad (4)$$

Linear regression was used to estimate all multivariable models. Following Add Health analytic guidelines, all analyses were weighted using recommended survey weights (Chen and Chantala 2014). These accounted for the unequal probability of being chosen for the sample (e.g., oversampling of certain subgroups) and attrition across survey waves. Moreover, sample weights make the results here representative of American adolescents. Unweighted analyses (not

shown here) yielded substantively similar results. All standard errors were clustered at the student level. All analyses were conducted using Stata 14.0 (StataCorp).

## **Results**

### *Descriptive results*

Table B-1 presents the means and SD of the variables used in the analysis, stratified by gender. Between Wave I and Wave II of data collection, girls had an average decline of 0.16 points in their depressive symptoms, compared to 0.04 for boys. Both are relatively small changes within this context, as the CES-D ranges from 0 to 57 and thus had a maximum potential change score of 57. However, this is not entirely surprising, given that only a year passes by between waves. Nevertheless, the relatively large standard deviation for this measure indicates that some students experience sharp fluctuations in depressive symptoms. Differences in the average change in CES-D scores, however, did not differ by gender, indicating that shifts in number and intensity of depressive symptoms are roughly the same for both boys and girls. Respondents' relationship with their families also remained relatively stable, though boys experienced larger declines in their overall relationship with their family. Boys were also more likely than girls to lose friends and less likely to become romantically involved. School relationships were relatively stable across waves and did not vary across gender. Overall, these descriptives indicate that, to the extent differences exist, boys tend to experience more negative (or smaller positive) changes in their social relationships than girls.

### *Main effect analysis*

Table B-2 presents results from models examining the association between changes in respondents' relationship and changes in depressive symptoms. Overall, 5 out of 9 social ties (mother-child relationship; father-child relationship; general family relationship; support from

friends; school connectedness; teacher-student relationships) were significantly associated with depressive symptoms (all  $p$ -values $<0.05$ ), indicating that as these relationships improved, depressive symptoms were predicted to decrease. Changes in family ties were predicted to have the largest impact on mental well-being ( $\beta$  ranged from 0.16 to 0.18), followed by changes in school relationships ( $\beta=0.13$ ), and changes in support from friends ( $\beta = 0.06$ ). In contrast, becoming romantically involved was associated with higher levels of depressive symptoms, though the coefficient was relatively small ( $\beta =0.04$ ,  $p<0.01$ ). Changes in two social relationships (number of friends, engagement with friends) were not significantly associated with changes in depressive symptoms.

#### *Interactive analysis*

The interactive models in Table B-2 presents results from models examining whether the association between changes in respondents' relationships and changes in depressive symptoms varied across gender. Model 1 shows that a one SD increase in the mother-child relationship was associated with a 0.14 SD decrease in depressive symptoms for boys, compared to a 0.21 SD decrease for girls ( $p<0.05$  for interaction term). Model 2 indicates that a one SD increase in the father-child relationship was associated with a 0.12 SD decrease in depressive symptoms for boys, compared to a 0.20 SD decrease for girls, though this difference was only marginally significant ( $p = 0.06$  for interaction term). Model 3 shows that a one SD increase in the general family relations was associated with a 0.15 SD decrease in depressive symptoms for boys, compared to a 0.22 SD decrease for girls ( $p<0.05$  for interaction term). However, Models 4-9 show that the association between changes in peer and school relationships on changes in depressive symptoms did not differ between girls and boys ( $p >0.05$  for all interaction terms).

Overall, these models provide qualified support for the reactivity hypothesis, as only changes in family ties appeared to have a stronger association with depressive symptoms for girls.

#### *Meditational analysis*

Table B-3 presents results from models examining the differential association between changes in family relationships and changes in depressive symptoms before (interactive models) and after (mediational models) accounting for gender differences in emotional responsiveness. Consistent with the reactivity hypothesis, changes in perceived love was more strongly associated with changes in depressive symptoms for girls than boys ( $p < 0.05$  for all gender\*loved and wanted interaction terms in mediational models). More importantly, the introduction of this new interaction term completely attenuated the differential estimate of mother, father, and family ties. Overall, this provides suggestive evidence that girls' greater emotional responsiveness to social ties may underlie the stronger association between family relationships on depressive symptoms.

#### *Sensitivity analysis*

This paper conducted two sensitivity analyses to explore the implications of methodological and measurement choices made in this study. First, we examined the significance of using first-difference models by re-evaluating the reactivity hypothesis with more conventional analysis. More specifically, we estimated linear regression models using family relations measured at Wave I to predict depressive symptoms at Wave II. The estimate for ties were allowed to vary across boys and girls via an interaction term. Results in unadjusted models (Appendix B-3: Models 1a, 2a, and 3a) indicated that only the estimate of general family ties had a stronger impact on depressive symptoms for girls than boys, and after adjusting for baseline depressive symptoms (Model 3b), this difference became statistically insignificant. However, as

noted above, these estimates may be biased because conventional analyses do not take into account all time-invariant confounders. Thus, these results highlight the potential limitations of standard approaches and the value of using first-difference models to test the reactivity hypothesis.

Second, this paper examined the implications of using depressive symptoms as the primary dependent variable. Some research suggests that boys and girls may respond to interpersonal stress in gender-specific ways. For instance, while girls may be more likely to experience depressive symptoms, boys may be more likely to cope by engaging in physical violence or substance abuse Aneshensel 1992. Thus, focusing solely on psychological distress may overstate the extent to which girls are more reactive to interpersonal stress than boys. Appendix B-4 explores this argument by examining whether changes in social ties were more strongly associated to three outcomes for boys than girls: (1) changes in the number of physical fights, (2) changes in alcohol usage, and (3) changes in marijuana usage. The interaction term between gender and family, peer, and school social relationships were insignificant for all outcomes, providing little evidence for this hypothesis.

## **Discussion and conclusion**

In this paper, we used data from a nationally representative study of American adolescents to test the reactivity hypothesis, which posits that girls are more reactive to the adverse impact of interpersonal stress, contributing to gender differences in mental well-being. Our analyses found qualified support for this theory. While changes in peer and school relationships were associated with changes in depressive symptoms, these estimates did not differ by gender. In contrast, changes to family ties were more strongly associated with changes in depressive symptoms for girls than boys. The size of the differential estimates was generally

small in absolute terms. For instance, a one SD decline in the mother-child relationship was associated with a 0.14 SD increase in depressive symptoms for boys and 0.21 SD increase for girls (a 0.07 SD differential estimate). Nonetheless, within the context of this study, where variation in social ties and depressive symptoms was limited because of the fixed-effects, a 0.07 SD differential estimate may be considered meaningful. Furthermore, in relative terms, the difference between a 0.21 and 0.14 estimate (40 percent) is also sizable.

These results are consistent with much of the research on adolescent social ties and depressive symptoms. Existing studies have shown girls to be more responsive to family-related interpersonal stress and equally responsive to school-based interpersonal stress (Meadows 2007, Vaughan, Foshee and Ennett 2010). For instance, Meadows (2007) showed that a one-SD increase in maternal social support was associated with a 0.08 and 0.13 SD decrease in depressive symptoms for boys and girls, respectively. On the other hand, this paper found that the relationship between peer-related interpersonal stress and changes in depressive symptoms did not vary across gender, while prior research has often documented heterogeneous effects (e.g., Joyner and Udry 2000). This discrepancy likely stems from the use of first difference models in this study, which limited variation in peer-related social ties to within-person changes.

Results from a mediational analysis lend further support for the reactivity hypothesis. Here I find evidence that changes in family relationships were positively associated with changes in the extent to which adolescents feel loved and cared for, but more so for girls than boys. Moreover, accounting for this gender difference in responsiveness completely attenuated the differential association between family ties and depressive symptoms. These findings are consistent with the longstanding argument that girls are more reactive to social relationships and thus are more sensitive to their effect on mental health. They also align with a recent study of



315 adolescents, which also found that gender differences in emotionality partially mediated the differential association between interpersonal stress and depressive symptoms (Charbonneau, Mezulis and Hyde 2009). To my knowledge, however, this is one of the first studies to explicitly test this proposed underlying mechanism using a nationally representative sample.

These findings have important implications for the research on gender, social relationships, and psychological functioning. Prior theoretical discussions of gender differences in responsiveness are often extreme, with some implying that females are more reactive to social ties across the board (e.g., Gilligan 1982) and others describing such descriptions as outright myths (e.g., Fischer 1993). The results from this study, I argue, support a more moderate position: while girls may be more responsive than boys, this may only apply to certain types of social ties. For instance, I show here that family ties elicited a stronger response from girls, while peer and school ties did not. This suggests it may be premature to completely dismiss girls' stronger reactivity as a possible mechanism that may drive their greater sensitivity to social relationships. At the same time, it is also important for researchers to move away from conceptualizing girls as universally more reactive and to begin exploring why some types of relationships matter more to girls than boys. Such work will move us away from broad claims about females and males and bring greater clarity around the specific social conditions that give rise to stronger responses.

The results here also suggest that policies aimed at building stronger ties between girls and their family may help mitigate some of the gender differences in adolescent well-being. Strengthening these relationships, however, may be especially difficult during adolescence, as conflicts between young people and their families generally increase during this period (Steinberg and Morris 2001). One potentially practical way to facilitate this process may be to

create opportunities for both parties to consistently spend time together. For instance, some studies suggest that eating family meals together on a regular basis may be an ideal way to foster greater connectedness between parents and children (Franko et al. 2008, Fulkerson, Neumark-Sztainer and Story 2006). It also may be useful to engage a wide range of family members, including parents and extended family, in these efforts, as girls in the study benefitted more than boys from family relationships, regardless of the specific tie.

This study had a number of strengths, in particular the use of a large, nationally representative sample and first-difference analytic models, which eliminated the bias from time-constant confounders. Nonetheless, there were also several important limitations. First, because of limitations in the Add Health, the mediational analysis could only use a crude and overly simplified measure (i.e., perceived love) to capture responsiveness. The construct is likely multi-dimensional, and the single variable used here likely only captures parts of this complexity. Second, this study used an adolescent sample and so the trends may not apply to individuals throughout the life course. Third, only a year elapsed between Wave I and Wave II of data collection. Thus, the trends reported here only hold true in the short run. Fourth, while first difference models help address all observed and unobserved time invariant confounders, estimates rely solely on changes over time. The impact of having stable, persistent social ties, on the other hand, is left unexplored in this analysis. Finally, following most of the existing research, this paper implies that changes in social ties impacts changes in mental health. However, it is possible that the reverse is also true (that mental health shapes the quality of one's social ties). Therefore, it is important to view these results as suggestive rather than causal.

Table B-1 : Study variables and measurements, by gender

	Girls		Boys		Diff	Min	Max	Miss
	Mean	SD	Mean	SD				
<b>Dependent variable</b>								
CES-D score	-0.16	7.41	-0.03	6.23	0.27	-50	47	-
<b>Predictor variables</b>								
<i>Family relationships</i>								
Mother-child relationship	-0.30	2.85	-0.35	2.36	-0.06	-15	14	9.10
Father-child relationship	-0.42	2.93	-0.46	2.66	-0.04	-16	16	34.37
General family relationship	-0.01	2.66	-0.18	2.57	0.17** *	-16	-16	2.00
<i>Peer relationships</i>								
Number of friends	-0.39	2.57	-0.50	2.60	0.11**	-10	10	0.00
Support from friends	0.08	0.90	0.08	0.91	0.00	-4	4	1.04
Engagement with friends	0.05	1.18	0.05	1.12	0.00 0.04**	-4	4	0.02
Romantic relationship	0.06	0.51	0.01	0.56	*	-1	1	0.62
<i>School relationships</i>								
School connectedness	-0.20	2.60	-0.12	2.42	-0.08	-15	15	9.58
Teacher-student relationship	0.02	2.13	0.06	2.23	-0.04	-12	14	10.82
<b>Time-varying control variables</b>								
<i>School factors</i>								
Grade point average	-0.07	0.68	-0.03	0.71	-0.04** - 0.02**	-3 0	3 1	11.32 0.00
School attendance	0.03	0.16	0.04	0.20	*			
<i>Family factors</i>								
Household size	-0.16	1.25	-0.16	1.17	0.00	-10	9	0.06
Relocated to different home	0.12	0.32	0.10	0.3	0.02**	0	1	0.13
Parental employment	0.01	0.14	0.00	0.13	0.00	-1	1	13.03

Note: Figures may not add up due to rounding. All variables listed here are change scores that were created by subtracting the value of a measure at Wave II from the value at Wave I. All measures are unstandardized. Gender differences between were assessed using t-tests.

\*\*\* p<0.01, \*\* p<0.05, \* p<0.10

Table B-2: First-difference models examining the association between changes in social relationships and changes in depressive symptoms, standardized coefficients

	Main effect models	Interactive models
	$\beta$ (SE)	$\beta$ (SE)
(1) Mother-child relationship	-0.18*** (0.01)	-0.14*** (0.02)
x Girl	- -	-0.07** (0.03)
(2) Father-child relationship	-0.16*** (0.02)	-0.12*** (0.03)
x Girl	- -	-0.08* (0.04)
(3) General family	-0.18*** (0.01)	-0.15*** (0.02)
x Girl	- -	-0.07** (0.03)
(4) Number of friends	0.01 (0.01)	0.02 (0.01)
x Girl	- -	-0.02 (0.02)
(5) Support from friends	-0.06*** (0.01)	-0.05*** (0.01)
x Girl	- -	-0.02 (0.02)
(6) Engagement with friends	0.00 (0.01)	0.00 (0.01)
x Girl	- -	0.01 (0.02)
(7) Romantic relationship	0.04*** (0.01)	0.03*** (0.01)
x Girl	- -	0.02 (0.02)
(8) Teacher-student relationship	-0.13*** (0.01)	-0.13*** (0.02)
x Girl	- -	0.00 (0.03)
(9) School connectedness	-0.13*** (0.01)	-0.13*** (0.02)
x Girl	- -	0.01 (0.02)

Note: All models control for changes in respondents' school attendance status, grade point average, household size, home location, and parental employment. Missing values on control variables imputed using multiple imputation with 5 replications. Robust standard errors are in parentheses and clustered at the respondent -level. All models are weighted using recommended sample weight. \*\*\*  $p < 0.01$ , \*\*  $p < 0.05$ , \*  $p < 0.10$

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Table B-3: First-difference models examining the association between changes in family relationships, changes in perceived love, and changes in depressive symptoms, standardized coefficients

	Interactive models $\beta$ (SE)	Mediational models $\beta$ (SE)
(1) Mother-child relationship	-0.14*** (0.02)	-0.10*** (0.02)
x Girl	-0.07** (0.03)	-0.04 (0.03)
Loved and wanted	-	0.13*** (0.02)
x Girl	-	0.05** (0.02)
(2) Father-child relationship	-0.12*** (0.03)	-0.07** (0.03)
x Girl	-0.08* (0.04)	-0.05 (0.04)
Loved and wanted	-	0.12*** (0.02)
x Girl	-	0.10*** (0.03)
(3) General family relationship	-0.15*** (0.02)	-0.13*** (0.02)
x Girl	-0.07** (0.03)	-0.04 (0.03)
Loved and wanted	-	0.12*** (0.02)
x Girl	-	0.08*** (0.02)

Note: All models control for changes in respondents' school attendance status, grade point average, household size, home location, and parental employment. Missing values on control variables imputed using multiple imputation with 5 replications. Robust standard errors are in parentheses and clustered at the respondent-level. All models are weighted using recommended sample weight. \*\*\*  $p < 0.01$ , \*\*  $p < 0.05$ , \*  $p < 0.10$

Appendix Table B-1: Percentage of cases missing social relationship information, by socio-demographic characteristics

	<u>Family Relationships</u>			<u>Peer Relationships</u>			<u>School Relationships</u>		
	Mother	Father	General family	Number of friends	Friends support	Friends engagement	Dating	School connectedness	Teachers
Boys	10%	32%	2%	0%	1%	0%	1%	10%	12%
Girls	8%	37%	2%	0%	1%	0%	0%	9%	10%
Difference	2%	-5%	0%	0%	0%	0%	1%	1%	2%
Age <16	5%	32%	1%	0%	1%	0%	1%	2%	3%
Age >=16	11%	35%	2%	0%	1%	0%	1%	13%	15%
Difference	-6%	-3%	-1%	0%	0%	0%	0%	-11%	-12%
White	9%	26%	2%	0%	1%	0%	1%	9%	10%
Non-White	9%	43%	2%	0%	1%	0%	1%	10%	12%
Difference	0%	-17%	0%	0%	0%	0%	0%	-1%	-2%
Mom non-college educated	5%	36%	2%	0%	1%	0%	1%	10%	12%
Mom college educated	3%	28%	1%	0%	1%	0%	0%	5%	6%
Difference	2%	8%	1%	0%	0%	0%	1%	5%	6%

Note: Figures are unweighted and may not add up due to rounding.

Appendix Table B-2: Detailed description of measures used in study

Variable	Description
<b>Main Outcome</b>	
CES-D score	<p>Index of 19 items:</p> <p>How often was the following true during the past week?</p> <ol style="list-style-type: none"> <li>1) You were bothered by things that don't usually bother you</li> <li>2) You didn't feel like eating, your appetite was poor</li> <li>3) You felt that you could not shake off the blues</li> <li>4) You felt you were just as good as other people</li> <li>5) You had trouble keeping your mind on what you were doing</li> <li>6) You felt depressed</li> <li>7) You felt that you were too tired to do things</li> <li>8) You felt hopeful about the future</li> <li>9) You thought your life had been a failure</li> <li>10) You felt fearful</li> <li>11) You were happy</li> <li>12) You talked less than usual</li> <li>13) You felt lonely</li> <li>14) People were unfriendly to you</li> <li>15) You enjoyed life</li> <li>16) You felt sad</li> <li>17) You felt that people disliked you</li> <li>18) It was hard to get started doing things</li> <li>19) You felt life was not worth living</li> </ol> <p>(0 = never or rarely; 1 = sometimes; 2 = a lot of the time; 3 = most of the time or all of the time)</p>



<b>Main predictors</b>	
<i>Family relationships</i>	
Mother-child relationship	<p>Index of 4 items:</p> <p>1) How close do you feel to your mother? (1 = not at all; 2 = very little; 3 = somewhat; 4 = quite a bit; 5 = very much)</p> <p>Do you agree or disagree with the following statement?</p> <p>2) Most of the time, your mother is warm and loving toward you.  3) You are satisfied with the way your mother and you communicate with each other.  4) Overall, you are satisfied with your relationship with your mother.</p> <p>(1 = strongly disagree; 2 = disagree; 3 = neither agree nor disagree; 4 = agree; 5 = strongly agree)</p>
Father-child relationship	<p>Index of 4 items:</p> <p>1) How close do you feel to your father? (1 = not at all; 2 = very little; 3 = somewhat; 4 = quite a bit; 5 = very much)</p> <p>Do you agree or disagree with the following statement?</p> <p>2) Most of the time, your father is warm and loving toward you.  3) You are satisfied with the way your father and you communicate with each other.  4) Overall, you are satisfied with your relationship with your father.</p> <p>(1 = strongly disagree; 2 = disagree; 3 = neither agree nor disagree; 4 = agree; 5 = strongly agree)</p>
General family relationship	<p>Index of 4 items</p> <p>1) How much do you feel that your parents care about you?  2) How much do you feel that people in your family understand you?  3) How much do you feel that you and your family have fun together?  4) How much do you feel that your family pays attention to you?</p>

	(1 = not at all; 2 = very little; 3 = somewhat; 4 = quite a bit; 5 = very much)
<i>Peer relationships</i>	
Number of friends	Sum of 2 items: 1) List the name of your five best female friends 2) List the name of your five best male friends
Support from friends	How much do you feel that your friends care about you? (1 = not at all; 2 = very little; 3 = somewhat; 4 = quite a bit; 5 = very much)
Engagement with friends	During the past week, how many times did you just hang out with friends? (0 = not at all; 1 = 1 or 2 times; 2 = 3 or 4 times; 3 = 5 or more times)
Romantic relationship	In the last 18 months, have you had a special romantic relationship with anyone? (0 = No; 1 = Yes)
<i>School relationships</i>	
School connectedness	Index of 3 items:  How much do you agree or disagree with the following statement?  1) You feel close to people at your school 2) You feel like you are a part of your school 3) You are happy to be at your school  (1 = strongly disagree; 2 = disagree; 3 = neither agree nor disagree; 4 = agree; 5 = strongly agree)
Teacher-student relationship	Index of 3 items:  1) How often have you had trouble getting along with your teachers? (0 = everyday ; 1 = almost every day; 2 = about once a week; 3 = just a few times; 4 = never) 2) How much do you feel that your teachers care about you? (1 = not at all; 2 = very little; 3 = somewhat; 4 = quite a bit; 5 = very much) 3) How much do you agree or disagree with the following statements: The teachers at your school treat students fairly.

	(1 = not at all; 2 = very little; 3 = somewhat; 4 = quite a bit; 5 = very much)
<b>Time-varying control variables</b>	
<i>School factors</i>	
Grade Point Average	Respondents were asked to report their grades across four academic subjects (English, math, science, history) on a 1-4 scale (1 = D or worse; 2 = C; 3 = B; 4 = A). Scores across these four subjects were averaged to create a grade point average measure at each wave.
School attendance	At Wave II, respondents were asked were still attending school (0 = not attending school, 1 = still attending school).
<i>Family factors</i>	
Household size	Respondents were asked to list each individual who was living in their household at each wave.
Relocated to different home	At Wave II, respondents were asked whether they lived in the same location as they did during Wave I of data collection (0 = did not move, 1 = moved to new home).
Parental employment	Respondents were asked if their mothers were being paid for work (0 = No ; 1 = Yes) and if their fathers were being paid for work (0 = No ; 1 = Yes). This item was combined into a single measure at each wave (0 = No parent employed; 1 = At least one parent employed) and converted into a change score (-1 = Parents became unemployed; 0 = No change in parental employment status, 1 = Parents became employed).
Note: All items were measured at Wave I and Wave II and converted into change scores for this analysis. This involved by subtracting the value of an item at Wave II from the value at Wave I.	

Appendix Table B-3: The association between family relationships at Wave I and depressive symptoms at Wave II, standardized coefficients

Predictors	Model 1a	Model 1b	Model 2a	Model 2b	Model 3a	Model 3b
Girl (ref = boy)	0.21*** (0.02)	0.10*** (0.02)	0.22*** (0.03)	0.11*** (0.02)	0.24*** (0.02)	0.12*** (0.02)
Relationship with mother at Wave I	-0.24*** (0.02)	-0.08*** (0.02)				
Relationship with mother at Wave I x Girl	-0.00 (0.02)	0.03 (0.02)				
Relationship with father at Wave I			-0.25*** (0.02)	-0.10*** (0.02)		
Relationship with father at Wave I x Girl			-0.03 (0.03)	0.00 (0.03)		
Relationship with general family at Wave I					-0.26*** (0.02)	-0.09*** (0.01)
Relationship with general family at Wave I x Girl					-0.07*** (0.02)	0.00 (0.02)

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Note: Each column reports the coefficients from linear regression models where the outcome is depressive symptoms, measured at Wave II. Models 1a, 2a, and 3a are unadjusted. Models 1b, 2b, and 3b control for depressive symptoms at baseline (Wave I). All models are weighted using recommended sample weight. Standard errors in parentheses. \*\*\*  $p < 0.01$ , \*\*  $p < 0.05$ , \*  $p < 0.10$

Appendix Table B-4: Association between changes in social ties and changes in alcohol consumption, physical violence, and marijuana usage

Predictors	Outcome: Alcohol usage	Outcome: Physical violence	Outcome: Marijuana usage
Family Relationships			
Mom * Gender	NS	NS	NS
Dad * Gender	NS	NS	NS
General family * Gender	NS	NS	NS
Peer Relationships			
Romantic relationship * Gender	NS	NS	NS
Friends care * Gender	NS	NS	NS
Friends hang * Gender	NS	NS	NS
Total friends * Gender	NS	NS	NS
School Relationships			
Teacher * Gender	NS	NS	NS
Total relationships * Gender	NS	NS	NS

Note: Each cell reports the results of an interaction term between gender and a change in a specific type of social tie. NS = Non-significant gender \* social relationship interaction.

All models control for changes in respondents' school attendance status, GPA, household size, home location, and parental employment. Missing values on control variables imputed using multiple imputation with 5 replications.

Alcohol usage was measured with the question "During the past 12 months, on how many days did you drink alcohol?" (0-6). A change score was computed by subtracting respondents' answer at T2 from T1.

Physical violence was measured with the question "During the past 12 months, how often did you get into a serious physical fight?" (0-3). A change score was computed by subtracting respondents' answer at T2 from T1.

Marijuana usage was measured with the question "During the past 30 days, how many times did you use marijuana?" (0-30). A change score was computed by subtracting respondents' answer at T2 from T1.

## **Chapter 4 : The cumulative impact of marriage on mental well-being**

### **Introduction**

The positive relationship between marriage and mental health is a well-established “social fact” in health and social science research (Umberson, Thomeer and Williams 2013). Hundreds of studies demonstrate that married adults experience significantly less psychological distress than unmarried adults, including having lower levels of depression and stress and higher levels of happiness, self-esteem, and overall life satisfaction (see Coombs (1991) for a review). The beneficial effects of marriage appear to hold regardless of demographic characteristics, such as sex or race/ethnicity (Umberson, Thomeer and Williams 2013), suggesting that a spouse may partially fulfill a basic human need for intimacy, companionship, and a sense of belonging (Baumeister and Leary 1995).

Life-course theory (LCT) implies that the positive impact of close social relationships such as marriage is cumulative and grow more pronounced over time (Thoits 2011; Waite 2009; Umberson and Montez 2010). A recent study drawing on this framework argues “Long durations of marriage foster economic and behavioral stability, shared obligations, and vested interests between partners, which in turn, promote healthy lifestyles and enhance socioeconomic and psychological resources” (Dupre and Nelson 2016:115). Most existing work, however, has focused on its short-term impact on mental health. For instance, one common approach involves examining the association between marital status at a single point in time—either contemporaneously or at a prior time period— and mental well-being. In contrast, we know relative little about the potential long-term effects of marriage, as few studies have considered

the consequences of an individual's marital history on well-being. Addressing this limitation is important because if the benefits of marriage accumulate over time, then existing research may be understating the full impact of marriage on mental well-being.

Estimating the cumulative effect of marriage, however, is complicated by methodological challenges associated with time-varying confounders. Income, for example, is a confounder that influences an individual's marital status and mental health, respectively. At the same time, past marital status can also influence how much income an individual has. In other words, income is both a confounder and a mediator through which marriage indirectly influences mental health. Prior studies have controlled for time-varying confounders such as income in order to eliminate a potential source of bias. But, in doing so, they have also eliminated an indirect pathway through which marriage shapes mental health, thereby underestimating the long-term impact of marriage. This problem becomes compounded in large longitudinal studies, as controlling time-varying confounders eliminates the indirect pathways through which marriage impacts mental wellbeing at each time point.

In this study, I attempt to address this methodological challenge with marginal structural models (MSMs). Unlike conventional regression approaches, these models can adjust for observed time-varying confounders without blocking indirect pathways through which marriage influences mental health (Robins, Hernan and Brumback 2000). As a result, I am able to more accurately estimate the cumulative association between marriage and mental health. Data for this analysis come from the U.S. the 16 waves of the National Longitudinal Study of Youth of 1979 (NLSY79). I focus here specifically on depressive symptoms, as it is the most common measure of mental health in the existing research and has important implications for an individual's overall health (Moussavi et al. 2007).



## **Background**

### **Links between marriage with mental health**

Theorists speculate that marriage improves mental health through a number of overlapping channels. Three of the most common mechanisms in the literature include (1) socio-economic resources; (2) social support; and (3) sense of purpose and belonging (Umberson, Thomeer and Williams 2013).

Economic resources are positively associated with mental health (Lorant et al. 2003; Muntaner et al. 2004), and married individuals tend to have more socio-economic resources than unmarried people (Waite 2009; Lupton 2003; Holden and Kuo 1996). This economic advantage is typically attributed to being part of a dual earning household (Umberson, Thomeer and Williams 2013), as well as the “marriage premium,” whereby married individuals earn higher wages than comparable unmarried people (Cheng 2016; Killewald 2013). These resources are important because they protect married people from economic hardship, which can be a major source of psychological distress. Increased resources also enable married people to purchase items or services that can improve or sustain their personal well-being (Waite 2009).

Social support is the level of help and care (either perceived or actual) individuals have available from people in their social network (House, Umberson and Landis 1988). This support can come in a variety of forms, including emotional, financial, or informational. Regardless of the specific type of help, social support is critical for mitigating the negative effect of stressors and for helping individuals feel cared for (Kawachi and Berkman 2001; Cohen and Wills 1985). Consequently, studies consistently find a positive relationship between social support and mental-wellbeing (Umberson and Montez 2010). Compared to the unmarried, married individuals consistently report having greater social support (Umberson et al. 1996), with most

identifying their spouses as their most important source of emotional connection and support (McPherson, Smith-Lovin and Brashears 2006).

Marriage is also thought to improve mental well-being by providing individuals with a sense of purpose and belonging. As part of being married, individuals become embedded within a shared system where they collaborate with their spouses to meet their needs and desires (Umberson, Thomeer and Williams 2013; Waite 2009). Participation within this system helps create a sense of purpose in one's life, as individuals work towards goals that are greater than themselves. It also helps foster a sense of belongingness, as individuals become part of a larger collective (Kawachi and Berkman 2001). Prior studies have demonstrated that a sense of purpose and belonging are important factors that help individuals maintain their mental well-being (Durkheim 1951; Berkman et al. 2000).

### **Life course theory and marriage**

LCT theory posits that initial forms of inequality between groups often become more pronounced throughout the life course (DiPrete and Eirich 2006; Elder Jr, Johnson and Crosnoe 2003). Applied to the study of marriage and mental health, LCT theory suggests that the differences in psychological functioning between married and unmarried individuals may become larger over time (Thoits 2011; Waite 2009; Umberson and Montez 2010). One central mechanism thought to drive this divergence is the duration of exposure (Kuh et al. 2003) : the longer an individual is married, the more he/she may be exposed to the potentially beneficial economic, social, and psychological effects of marriage. For instance, remaining married long-term may enable individuals to pool resources together for an extended period of time, allowing them to secure expensive items (e.g., a home) that promote financial stability and, ultimately, long-term mental wellbeing (Lupton 2003; Wilmoth and Koso 2002 ). Conversely, remaining

unmarried long-term may allow the negative consequences of being single to accumulate and compound. For instance, individuals who never get married experience higher levels of loneliness and social isolation than their counterparts (Stack 1998; Cornwell and Waite 2009), and it is possible that these challenges may become progressively worse over time. Thus, LCT theory implies that the impact of marriage on mental well-being may be duration dependent.

### **Prior research on marriage and health**

The association between marriage and mental health has been studied extensively using cross-sectional and longitudinal designs. The general consensus among this work is that marriage is strongly related to mental health, net of standard socio-demographic covariates (Coombs 1991; Umberson, Thomeer and Williams 2013). These studies, however, typically use marital status at a single point in time, implicitly treating marriage as a static concept. While this assumption simplifies the analysis, I show below that it ignores a great deal of heterogeneity, as it conflates people who have been married for varying amounts of time into a single category. And, by failing to properly disentangle short versus long-term marriages, previous studies provide an oversimplified account of the relationship between marriage and mental well-being.

A handful of studies have considered how individuals' entire marital history influences their health, though these focus largely on physical health. In one of the first studies on the topic, Lillard and Waite (1995) use the Panel Study of Income Dynamics dataset to examine the association between marital history and mortality. Their analysis found that for both men and women, the risk of mortality was negatively associated with the duration of marriage, even after adjusting for socio-economic covariates. Using the Human Retirement Study (HRS), a longitudinal study of older adults, Dupre, Beck and Meadows (2009) also find a significant, negative relationship between marital history and mortality for both men and women, net of

standard socio-demographic covariates. Both Zhang and Hayward (2006) and Dupre and Meadows (2007) show in separate analyses of the HRS that older adults who remained continuously married were less likely to develop chronic conditions compared to those who had suffered marital loss. Using biomarker data, McFarland, Hayward and Brown (2013) show that long-term marriage may protect individuals' health at a biological level. These studies together suggest that marital duration is positively associated with various measures of physical health.

To my knowledge, only two studies have conducted similar analyses with mental health outcomes as the primary dependent variable. The first is Barrett (2000), who shows in her cross-sectional analysis of a community sample that among married respondents, those who were continuously married were less likely to have anxiety problems or substance abuse issues compared to who experienced marital disruptions (e.g., previously divorced). No significance difference, however, was found for depressive symptoms—the main outcome of interest for this study. The second is Hughes and Waite (2009), who similarly find that marital duration (defined in their study as the percentage of a respondent's life he/she was married) was not associated with depression among people who were continuously married. Thus, unlike the research on physical health, these two studies suggest that marriage may not have a cumulative effect on depression.

While these studies make an important contribution to the literature by exploring the link between marital duration and health, they are nevertheless still limited because they rely on conventional regression methods, which do not properly account for the influence of time-varying confounders. Consider income, for example, a covariate that is associated with both marital status and depression. If an individual gets a divorce, this may influence how much income he/she has, which may in turn influence his/her ability to remarry in the future. Thus,

marital status influences, and is influenced by, time-vary factors such as income. Omitting these covariates results in upwardly biased estimates, as a confounder is excluded from the analyses. On the other hand, controlling these measures, as most prior studies have done, likely results in downwardly biased estimates, as it eliminates an indirect pathway through which marriage shapes mental health. The central contribution of this study is to use an analytic method, MSMs, to properly handle this challenge and ultimately provide more credible estimates of the association between marital duration on depressive symptoms.

### **Determinants of marital status**

For MSMs to properly account for time-varying confounders and provide accurate estimates, it is necessary to specify a model that includes variables that predicts movement into and out of marital states at different points in time. Broadly speaking, prior studies have shown that a confluence of individual, socio-demographic, and contextual factors together shape an individual's propensity for marriage throughout the life course.

In terms of individual-level characteristics, Sampson, Laub and Wimer (2006) show that commonly neglected factors, including aptitude and personality traits are significant predictors of marriage. Further, while research commonly presumes that marriage improves health, there is some evidence of reverse causality: that is, healthy people may be more likely than unhealthy people to opt into marriage (Stutzer and Frey 2006).

In terms of socio-demographic characteristics, prior work shows that sex, race, and parental SES are related to marriage: women, whites, and those from more advantaged family backgrounds are, on average, more likely than their counterparts to marry (Avery, Goldscheider and Speare 1992; Lichter et al. 1992; Manning and Smock 1995; Axinn and Thornton 1992). The presence of children also appears to lower the risk for divorce among married couples and

promote the transition into marriage (Manning and Smock 1995). Finally, research shows that economic factors, such as income and employment status, and cohabitation with a partner, are two of the most robust predictors of an individual's marital status (Edin and Kefalas 2011, Kennedy and Bumpass 2008; Avery, Goldscheider and Speare 1992; Clarkberg 1999; Burgess, Propper and Aassve 2003) .

In terms of contextual factors, ecological studies find significant differences in marriage rates across different areas of the United States. Because of cultural, economic, and social differences, individuals living in rural areas are more likely to become married and less likely to get divorced than their counterparts in urban areas (Census 2017). Related to this, those living in the northeastern portions of the United States are significantly less likely to get married or divorced, respectively, compared to people living in other regions of the United States (Elliott and Simmons 2011).

## **Data and Methods**

### **Data**

This paper used the NLSY79, a nationally representative study of American youth. Beginning in 1979, 12,686 young people aged 14-22 were interviewed annually until the year 1994 and bi-annually thereafter. The NLSY contains rich, longitudinal information on respondents' family background, marital histories, work experience, and living situations, which are important for properly adjusting for time-varying confounders with MSMs. Respondents were included in the study when they were 18 years or older<sup>6</sup> and followed for 16 rounds of

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<sup>6</sup> While many respondents were in the study prior to being age 18, I do not include them until age 18. This is because the main treatment variable (marital duration) is measured as the proportion of time a respondent is married during the study. Since child marriages are relatively rare occurrence (less than 1% in the analytic sample), younger respondents will likely have a lower "dosage" by virtue of entering the study earlier in their lives. Setting the starting point to 18 years thus helps make marital duration more comparability with others who began the study as young adults. As a consequence of this decision, respondents younger than age 18 at the beginning of the study are

interviews, when they were 32-38 years old. Time point 16 was selected as the endpoint because it was the period directly preceding when the oldest members of the study had their depressive symptoms measured (at age 40). Because respondents' first year was used as the baseline period, this study contained 15 waves of follow-up information. Figure 1 depicts this information graphically.

To construct the final analytic sample, I employed the following restrictions. First, I dropped 2,923 respondents because they were part of special subsamples that were not followed until they were 40 and thus did not answer questions about their mental health. Second, I dropped 2,996 respondents because they missed at least one follow-up before they turn 40 years old. Third, I dropped 355 respondents who did not have their complete marital history available or who had missing mental health information. The final analytic sample contained a total of 6,412 respondents.

## **Measures**

### *Main predictor variable: Marital duration*

The main predictor of interest was marital duration, measured as the proportion of time respondents were married during the study. During each interview, respondents were asked to state whether they were married, divorced, widowed, or never married. This item was dichotomized, with 1 representing married and 0 representing non-married. I then computed the total number of years respondents reported being married during the study and divided this figure by the total number of years they participated in the study. For example, if a respondent reported being married in 10 years and participated in the study for 20 years, then he/she was given a marital duration value of 0.5 (10/20).

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left-censored until they reach adulthood. Moreover, the first year that they are 18 or older is treated as their baseline year, rather than the first year of the study.

### *Dependent Variable: CES-D Score*

Mental health was operationalized in this study as depressive symptoms, measured during the first interview a respondents was age 40 years of age or older.<sup>7</sup> Respondents were asked 7 questions related to how often they experienced different depressive symptoms over the past week.<sup>8</sup> These items make up an abbreviated version of the Center for Epidemiologic Studies' Depression (CES-D) scale, a reliable, well-validated 20-item instrument used to identify individuals at risk of major depression (Radloff 1977). Responses to these 7 questions ranged from 0 to 3, (0 = never or rarely; 1 = sometimes; 2 = a lot of the time, 3 = most or all of the time) with higher values indicating higher levels of depressive symptoms. These items were summed to create a single index ( $\alpha=0.82$ ) that ranged from 0 to 21. To facilitate the interpretation of this scale, I standardized this measure, with a mean of 0 and a SD of 1.

### *Covariates*

This study included a variety of time-invariant and time-varying individual, socio-demographic, and contextual covariates that predict the dynamic selection into marriage, as described above.

For individual-level characteristics, I included aptitude, personality traits, and health status. Aptitude was measured using respondents' performance on the Armed Forces Qualification Test (AFQT), a standardized test measuring their math and verbal abilities. Raw scores for this test were converted into age-referenced percentiles. Personality was measured

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<sup>7</sup> Because the NLSY79 interviewed respondents on a bi-annual basis after the year 2000, some respondents were slightly older than 40 when information about their depressive symptoms was collected. For instance, a respondent may be interviewed at age 39 in 2000 and then age 41 in 2002. In these cases, the NLSY79 administered health questions the first interview respondents were age 40 or above.

<sup>8</sup> The seven items that form the 7 item CES-D scale are as follows: (1) I didn't feel like eating; my appetite was poor; (2) I had trouble keeping my mind on what I was doing; (3) I felt depressed; (4) I feel like everything I do takes extra effort; (5) My sleep was restless; (6) I felt sad; (7) I could not get "going".



using an abbreviated 4-item version of Rotter's locus of control scale. Respondents were asked 4 questions related to the degree to which they believed they personally controlled the events in their lives, rather than outside forces. These items were summed to create a single index ranging from 0 to 16 ( $\alpha = 0.36$ ), with higher values representing a stronger belief that events are out of people's control. Health status was a binary indicator created by combining 2 items. Respondents were asked whether their health limited the (1) type or (2) amount of work they could perform. Those who responded "yes" to either of these questions were coded as having health problems, while those who responded "no" to both questions were coded as being healthy.

For socio-demographic characteristics, I included: sex (categorical; 1= male, 2=female), race (categorical; 1= White, 2= Black, 3= Hispanic), highest level of education completed by either parents (categorical; 1 = less than high school; 2 = high school; 3 = some college; 4 = 4 years of college; 5 = 4+ years of college), family income (logged, continuous), poverty status (binary; 0 = income not below poverty line; 1 = income below poverty line), number of weeks spent unemployed in the past year (continuous), whether respondents had children of their own in the household (binary, 0 = no children in household; 1= At least one child in the household), age and age-squared (continuous), and cohabitation with an opposite sex partner (binary; 0 = no; 1 = yes). For contextual variables, I included two variables: a binary indicator for whether the respondent lived in an urban or rural area (categorical; 1= rural, 2 = urban); and a measure of the region of the country where the respondent resides (categorical; 1 = Northeastern, 2= Midwest, 3 = South, 4 = West).

I used multiple imputation to handle missing information for covariates. This is a technique that replaces missing data with values generated from an imputation model. This process is repeated multiple times and results in multiple complete datasets. Each of these

complete datasets is then analyzed separately, and results are averaged to produce estimates and standard errors that incorporate uncertainty associated with the missing information. For this study, I generated 5 complete datasets using chained equations to simulate missing values (White, Royston and Wood 2011).

### *Marginal Structural Models*

In the presence of time-varying confounders (i.e., covariates that predict and are predicted by an exposure), prior studies have shown that conventional regression models typically understate the relationship between an exposure and an outcome (Robins, Hernan and Brumback 2000). This is because these models directly adjust for these confounders, which can inadvertently eliminate intermediate pathways through which an exposure operates. For instance, as noted above, while controlling for income is necessary for removing an important source of bias, it also eliminates a key indirect pathway through which marriage shapes mental health.

MSMs are a class of models that were designed in part to more properly account for the influence of time-varying confounders. More specifically, rather than direct adjustment, these models rely on inverse-probability of treatment weights (IPTWs) to account for selection bias. Informally, these down weight particular types of respondents who are over-represented in their treatment group while up weighting individuals that are under-represented in their treatment group (Robins, Hernan and Brumback 2000; Wodtke, Harding and Elwert 2011). For this study, individuals who have a high probability of being married—and are married—are given less weight, while individuals who have a low probability of being married—but are married—are given more weight. Weighing an unadjusted sample by the IPTWs creates a pseudo-population in which treatment (in this case marital duration) is independent of the observed confounders, making it unnecessary to condition on these variables. At the same time, these weights do not

block intermediate pathways, thus offering more accurate estimates of the impact of marriage on mental well-being.

Creating the IPTWs proceeded in several steps (described in greater detail in Cole and Hernán 2008). First, I arranged the data longitudinally in person-year format. Next, I estimated a series of pooled logistic regression models predicting respondents' probability of having their observed marital status ( $a_{ki}$ ) at each time point ( $k$ ), conditional on all past marriage statuses ( $\bar{a}_{k-1}$ ), and time-invariant characteristics and past values for time-varying confounders ( $\bar{l}_{ki}$ ). Standard errors were clustered at the person level in these models to account for potential dependence of observations over time within respondents. I then multiplied these probabilities across all waves, resulting in a cumulative probability that reflected the odds that an individual experiences a particular marital history. Taking the inverse of this probability yields the IPTW ( $\omega$ ).

$$\omega_i = \prod_{k=1}^K \frac{1}{P(A_k = a_{ki} | \bar{A}_{k-1} = \bar{a}_{k-1}, \bar{L} = \bar{l}_{ki})}$$

Unfortunately, IPTWs can be highly variable and produce imprecise estimates for non-normal sampling distributions (Cole and Hernán 2008). Thus, following prior studies (Wodtke, Harding and Elwert 2011; Sampson, Laub and Wimer 2006; Sharkey and Elwert 2011), I created stabilized versions of the IPTWs ( $s\omega$ ):

$$s\omega_i = \prod_{k=1}^K \frac{P(A_k = a_{ki} | \bar{A}_{k-1} = \bar{a}_{k-1}, \bar{L}_0 = \bar{l}_0)}{P(A_k = a_{ki} | \bar{A}_{k-1} = \bar{a}_{k-1}, \bar{L} = \bar{l}_{ki})}$$

The denominator in the stabilized IPTW is the original IPTW. The numerator is computed in a similar manner: we estimate a pooled logistic regression model predicting marriage at each time point, except this time we condition on time-invariant/baseline characteristics ( $\bar{l}_0$ ) and prior marriage history, but not time-varying factors. These probabilities

are then multiplied across all years to create a cumulative probability of a particular marriage history. Because baseline characteristics were included in both the numerator and denominator, it is necessary to condition on these factors in the final model to obtain unbiased estimates. The coefficients from the model used to create the IPTWs are available in Appendix C-1.

To highlight the importance of using methods that properly account for time-varying confounders, I compared results from MSMs with those from unadjusted and conventional models in the main analysis. The unadjusted model was unweighted and regressed respondent's CES-D score (Y) on the treatment measure, marital duration ( $B_1$ ):

$$Y_i = B_0 + B_1 (\text{Marital Duration})_i + \varepsilon_i$$

The conventional model was also unweighted and regressed respondents' CES-D score (Y) on marital duration, a vector of time-varying covariates averaged from time 1 to time 15 ( $B_3$ ), and a vector of time invariant characteristics ( $B_4$ ):

$$Y_i = B_0 + B_1 (\text{Marital Duration})_i + B_2 (\text{Time - varying covs})_i + B_3 (\text{Time - invar covs})_i + \varepsilon_i$$

Finally, the MSM was weighted using the stabilized IPTWs and regressed respondents' CES-D score (Y) on the treatment ( $B_1$ ) and a vector of time-invariant characteristics ( $B_2$ ):

$$Y_i = B_0 + B_1 (\text{Marital Duration})_i + B_2 (\text{Time - invar covs})_i + \varepsilon_i$$

In order for MSMs to provide unbiased and consistent estimates, several assumptions must be made (Robins, Hernan and Brumback 2000). First, we must assume that there are no unmeasured confounders (i.e., we have included all variables that are linked with depressive symptoms and the probability of being married). Second, we must assume that the MSMs and the treatment models used to derive the IPTWs are correctly specified. Third, we must assume that all respondents have a non-zero probability of receiving each level of treatment across all

levels and combinations of covariates. While these are strong assumptions, they are the same ones that for conventional regression. However, conventional approaches require researchers to assume that time-varying confounders are not associated with past treatment status. For MSMs, it is not necessary for to make this latter assumption, as IPTW accounts for this type of time-related confounding. Thus, MSMs actually require fewer assumptions while providing more credible estimates than the conventional approach.

### *Sample Attrition*

In this study, I define attrition as (1) missing a follow-up interview; (2) having missing depressive symptoms information; or (3) having missing marital status information. Respondents were considered lost the first year any of these three conditions were met and are excluded from the final IPTW analyses. However, their information prior to attrition was retained for the construction of the IPT weights. Among 9,763 individuals who participated in the 1979 baseline interviews (excluding special subsample respondents), 3,351 (32%) were dropped from the final analysis. This level of attrition is not uncommon for large longitudinal studies that cover such an extended period of time. In order to account for the potential of non-random attrition, I created stabilized censor-weights ( $sc\omega_i$ ) in a similar manner as the stabilized IPTW, only this time the outcome is not being censored at each wave of the study.

$$sc\omega_i = \prod_{k=1}^K \frac{P(C_k = 0 | \bar{C}_{k-1} = 0, \bar{A}_{k-1} = \bar{a}_{k-1}, \bar{L}_0 = \bar{l}_0)}{P(C_k = 0 | \bar{C}_{k-1} = 0, \bar{A}_{k-1} = \bar{a}_{k-1}, \bar{L} = \bar{l}_{ki})}$$

More specifically, I estimated the probability that a respondent is not censored at time  $k$  ( $C_k = 0$ ), conditional on past attrition history ( $\bar{C}_{k-1}$ ) and time invariant and past time-varying confounders ( $\bar{l}_{ki}$ ). These probabilities were then multiplied across all time periods to create a cumulative probability of attrition. This value is the numerator of the stabilized censor weight. For the denominator, I estimated a similar model but excluded time-varying factors other than

prior censorship history. The product of the stabilized IPTW and the stabilized censor weight ( $sw * sc\omega$ ) was used as the final weight in the analysis.

## **Weights**

One of the main concerns related to the use of IPW is highly variable weights, which may have an extreme effect on the results (Robins, Hernan and Brumback 2000; Cole and Hernán 2008). To minimize this problem, I first created stabilized versions of the IPTWs, as described above. To further ensure that the influence of extreme weights do not drive the results, I top-code the stabilized IPT and censor weights at the 99th and 1th percentile. Overall, these truncated weights are relatively “well-behaved” (mean close to 1, small range) as shown in Table 4. The stabilized IPT weight has a mean of 0.99, a SD 0.39, a range of 0.33 to 2.61. The stabilized censored weight has mean of 0.99, a SD of 0.05, and a range of 0.83 to 1.12. Together, the product of these two weights (the final weight used in the analysis) has a mean of 0.99, a SD of 0.40, and a range of 0.27 to 2.92.

## **Results**

### **Descriptive Results**

Table 1 provides descriptive statistics for time-invariant and baseline characteristics of the study sample. Overall, 55% of the analytic sample was female, 53% was White, and 34% had parents with less than a high school diploma. At baseline, roughly 11% of respondents were married, 12% had a child living in their household, and around 2% were cohabitating with an opposite sex partner. Table 2 reports descriptive statistics for time-varying characteristics. Rates of marriage steadily increased over time: at time point 1, 17% of respondents were married, compared to 38% at time point 5, 53% at time point 10, and 58% at time point 15. Likewise,

cohabitation, having at least one child in the household and family income generally increased over the course of the study.

Table 3 presents a more detailed descriptive look at marital duration, the primary treatment of interest in this study. The average respondent in the analytic sample spent around 45% of his/her time in the study married. This average, however, conceals much heterogeneity. For example, 31% of respondents were married 0-19% of the time, while 22% of respondents were married 81-100% of the time. A simple average also overlooks the different marital trajectories respondents experienced. For instance, 15% of respondents experienced 2 changes in marital status (e.g., (1) single to married and (2) married to divorced) and another 12% experienced over three changes. Thus, though respondents may spend the same proportion of their lives married/unmarried, the way they arrive at that figure may be fundamentally different.

### **Main Results**

Table 5 shows the estimated association between marital duration on depressive symptoms using unadjusted, conventional, and marginal structural models. In the unadjusted model (Model 1), we find that marital duration is negatively associated with depressive symptoms ( $\beta = -0.25$ ;  $p < 0.01$ ). This model implies that an individual who was continuously married had a CES-D score that was 0.25 SDs less than someone who was continuously unmarried. This suggests that the benefits of marriage may be cumulative and becomes more pronounced over time. These results, however, cannot be interpreted as a causal relationship, as they do not account for known confounders.

In the conventional regression model, I condition on time-invariant and time-varying covariates averaged from time 1 to time 15. Overall, we find that controlling for these variables reduces the coefficient by around 48% ( $\beta = -0.25$  to  $\beta = -0.13$ ). This suggests that conditioning

on observed confounders “explains away” much of the relationship between marital duration and depressive symptoms. However, as mentioned above, these results are likely over controlled because they condition on time-varying that influence, and are influenced by, marital history.

In the MSM, we find that marital duration has a negative relationship with depressive symptoms, net of time-varying and time-invariant confounders ( $\beta = -0.20$ ;  $p < 0.001$ ). More specifically, we find that an individual who was continuously married had a CES-D score that was 0.20 SDs less than someone who was continuously unmarried. Compared to the estimates from the conventional model, the MSM estimate is roughly 0.07 SDs larger ( $\beta = -0.13$  versus  $\beta = -0.20$ ). These differences highlight the importance of appropriate methods, as the only difference between the conventional and MSMs is their handling of time-varying confounders.

### **Secondary analysis**

Though the analysis above suggests that the benefits of marriage may accrue over time, it is possible that this association may vary across sub-groups. Table 6 reports the results from analyses exploring effect heterogeneity across socio-demographic characteristics. MSMs show that marital duration had a similarly positive association on depressive symptoms across gender and parental education, respectively, as indicated by the similar magnitude of the coefficients and the non-significant interaction terms. However, the association between marital duration and mental health was approximately three times larger for Blacks compared to Whites ( $\beta = 0.33$  versus  $\beta = 0.11$ ,  $P = 0.02$  for interaction), suggesting that the former may be much more sensitive to the benefits of sustained exposure to marriage.

The main analysis also treated unmarried adults as a uniform group, potentially overlooking important heterogeneity. In particular, there is much debate in sociological research around whether marriage and cohabitation confer similar protective effects against psychological



distress. Table 7 explores differences in mental health between married and cohabiting individuals. Estimates for long-term cohabitation were fairly imprecise, in large part because relatively few respondents cohabitated for long periods of time. Nonetheless, results indicated that individuals who continuously cohabitated with a partner were predicted to have significantly more depressive symptoms ( $\beta = 0.31$ ) relative to continuously married individuals. These results suggest that long-term cohabitation may not offer the same protective benefits as long-term marriage.

Table 8 further explores heterogeneity by examining how the association between marital duration and depressive symptoms might vary across age. For this, I computed measures for the proportion of time a respondent was married during the first (wave 1-8) and second (wave 9-15) half of the study, respectively. Estimates for the first half of the study found that individuals who were continuously married were predicted to have essentially the level of depressive symptoms at midlife as those were who continuously unmarried. On the other hand, continuous marriage during the second half of the study conferred large mental health benefits ( $\beta = 0.18$ ). These results indicate that long-term marriage may be more consequential when respondents are older.

### **Sensitivity Analysis**

To assess the robustness of the results presents above, I employ several sensitivity checks. First, there may be concerns that the results may be sensitive to the truncation point used for the final weights. To explore this further, I re-estimate the main models, truncating the IPT and censor weights at different common percentiles (Robins, Hernan and Brumback 2000). As shown in Appendix C-2, the results are virtually identical, regardless of whether I use a 1%, 2.5%, or 5% cut-point, indicating that the choice of weights used do not determine the main findings.

Third, I assess the positivity assumption, which requires that respondents have a non-zero probability of receiving treatment at every level and combination of covariates. Because the US does not legally restrict people from becoming married based on socio-demographic factors, there is no to expect that any specific sub-groups have a zero probability of getting married (other than issues related to sampling). Appendix C-3 examines the distribution of marriage across several key confounders. Overall, we find that there are respondents for every treatment level (married and unmarried) across all combinations of these variables, suggesting that the positivity requirement is plausibly met in this analysis.

Fourth, there may be some concern about the relatively large number of cases that were lost because of missed follow-ups or missing marital status information. To examine this issue, I impute values for these censored respondents and re-estimate the main models (Appendix C-4). Results were virtually identical to the ones in the main analysis, indicating that the main finding was not solely a function of the analytic sample used.

Fifth, I test for nonlinearity by modeling marital duration as a higher order polynomial. Quadratic, cubic, and fourth order terms were all non-significant (data not shown), suggesting that a linear term is a good approximation of the relationship between marital duration and depressive symptoms. Sixth, I examine whether measuring depressive symptoms as a binary variable change the interpretation of the findings (Appendix C-5). Results were substantively similar to the main findings.

## **Discussion and Conclusion**

In this paper, we utilized 16 waves of data from the NLSY79 to assess the impact of an individual's marital history during early adulthood (from age 19-23 to 32-39) on their mental well-being at midlife (age 40-41). Results from marginal structural models showed that greater

exposure to marriage during this period was associated with fewer depressive symptoms, even after adjusting for a host of observable time-invariant and time-varying characteristics. Overall, these results are consistent with LCT theory, which posits that marriage may be a cumulative process that offers increasing health benefits over time.

These findings differ from the two existing studies that have investigated the relationship between marital history and depressive symptoms. Among those that are married, Barrett (2000) finds no difference in depressive symptoms between those who have been continuously married and those who have remarried. Similarly, among those who have been continuously married, Hughes and Waite (2009) find no difference in depression among those who have spent a larger proportion of their lives married. Some of this discrepancy may be due to differences in samples, as both studies condition among who are currently married while I include those who married and unmarried. More importantly, however, both rely on conventional regression for their analysis, which fail to properly adjust the dynamic nature of marriage. Without using methods that properly accounting for the non-random selection into and out of marriage over time, estimates of marital history are likely biased. The analysis here provides evidence for this claim, as I use conventional regression methods and observe a significantly diminished marital duration coefficient compared to the MSMs. The present work thus advances existing research by partially correcting for this methodological problem and providing more credible estimates of the long-term relationship between marriage and well-being.

It is unclear what mechanisms may be driving the accumulating benefits of marriage. Existing theories suggest that marriage may promote mental well-being through increased economic resources, social support, and sense of belonging and purpose. Of the three mechanisms, economic resources may be especially relevant within the life-course framework,

given that the income differential between married and unmarried generally grows over time (Zagorsky 2005). These additional resources may permit persistently married individuals to obtain goods and services that promote financial stability, self-fulfillment, and, ultimately, long-term mental wellbeing (Lupton 2003). Remaining unmarried long-term may, in contrast, may expose individuals to persistent financial hardship, which is significantly more detrimental to one's mental health than short-term financial distress (Lynch, Kaplan and Shema 1997). Further research is necessary to test this and other potential mechanisms.

These findings have important implications for existing research on marriage and mental health. In particular, they suggest that researchers should not only investigate whether individuals are married, but for how long. While the relationship between marital status and mental health is well established, the relationship between marital duration and mental health has only received scant attention in the existing empirical research. Yet, as this study demonstrates, ignoring marital history likely provides an oversimplified picture of how marriage shapes one's well-being. A focus on the cumulative benefits of marriage brings the literature into better alignment with LCT theory, a framework that guides much of work on mental well-being and health more generally. This finding also builds on several recent studies that find a positive relationship between marital duration and physical well-being (e.g., Dupre, Beck and Meadows 2009; Zhang and Hayward 2006). Thus, for empirical and theoretical reasons, I argue it is important for future research to account for marriage history in order to establish a more accurate and comprehensive understanding of the linkage between marriage and mental health.

Secondary analyses found suggestive evidence of effect heterogeneity. More specifically, while the positive association between marital duration and mental well-being was consistent across gender and SES, Blacks appeared to benefit significantly more than Whites from

sustained exposure to marriage. These findings complement two prior studies that find larger mental health returns from marriage for Blacks in the short term (Kiecolt, Hughes and Keith 2008; Liu and Umberson 2008). While the mechanisms driving this differential effect are unclear, researchers have speculated that Blacks may be more reliant on familial ties to cope with psychological distress because other types of resources (e.g. money) are not as readily available (Mouzon 2013).

Though estimates were fairly imprecise, analyses found that long-term marriage offered greater mental health benefits relative to long-term cohabitation. This result aligns with prior research showing that cohabitating couples, on average, have worse mental health than their married counterparts (see Umberson, Thomeer and Williams 2013 for a review). It is possible that resource sharing and the provision of social support increase faster for married couples compared to their cohabitating couples, leading to the discrepancy in mental well-being. Prolonged cohabitation may also lead to be a source of frustration and unhappiness, as couples typically cohabit temporarily before transitioning into marriage or ending the relationship (Smock 2000).

Analyses stratified by age also revealed that marital duration may be most consequential during the latter half of early adulthood. This finding is substantively consistent with a recent study, which found that the mental health benefits of marriage were larger for adults married after the age of 21 (Uecker 2012). They speculate that part of the difference across age was related to social approval; marriages after the age of 21 are generally aligned with dominant social norms. It may also be possible that persistent marriage during the latter half of the early adulthood is characterized by greater levels of commitment and pooling of important resources.

It is important to view the findings and implications here in light of several limitations. First, while this study adjusted for an extensive set of covariates, there still may be unmeasured variables confounding the relationship between marriage and mental well-being. For instance, the analysis could not control for mental health prior to midlife because the NLSY79 did not measure depressive symptoms at baseline or repeatedly during the study period. Neither marginal structural models nor conventional regression models can directly solve the problems related to unmeasured confounders. Thus, it is important to view the results here as suggestive rather than causal.

Second, this study only focused on a single outcome measure: depressive symptoms. Mental health is a complex construct and can be operationalized in a variety of ways. Moreover, depression is often the results of a complex inter-play between biological, psychological and social forces that take place over the life course. Future work can help address this gap by examining a broader range of health measures, including more intermediate outcomes that may shape the development of depression later in life. Third, I only focus on one type of social relationship (marriage) for this study. It is conceivable that the relationship between well-being and other social ties, such as friendships, also are cumulative, as they are thought to influence mental health through similar channels. However, in order to limit the scope of this study, I only examined the influence of marital duration.

These limitations notwithstanding, this study is among the first to show that the relationship between marital duration and mental health increase over time. This finding highlights the need consider the link between marriage and health beyond cross-sectional or short-term settings. This study also illustrates the value of new analytic tools such as marginal structural models, which enable researcher to investigate questions related to time in more

empirically credible ways. Moving forward, merging rigorous methods with a focus on temporality has the potential to help advance our understanding of how social relationships improve our health in new and important ways.

Table C-1: Time-invariant and baseline characteristics, NLSY79, 1979-2006

Variable	Mean
CES-D Score (standardized)	-0.16
	(0.98)
Sex	
• Male	0.45
• Female	0.55
Race	
• White	0.53
• Black	0.30
• Hispanic	0.18
Parental Education	
• Less than HS	0.34
• HS only	0.39
• Some college	0.12
• 4-year of college	0.09
• 4+ years of college	0.07
Locus of control	8.71
	(2.39)
Aptitude	41.09
	(29.10)
Baseline Age	18.77
	(1.16)
Baseline Age Sq	353.95
	(45.29)
Baseline marital status	
• Married	0.11
• Not married	0.89
Baseline cohabitation	
• Not cohabiting with a partner	0.98
• Cohabiting with a partner	0.02
Baseline children in HH	
• At least 1 child in HH	0.12
• No children in HH	0.88
Baseline family income (logged)	9.40
	(1.18)
Baseline weeks unemployed	21.83
	(21.51)



Baseline health	
• Health problems	0.05
• No health problems	0.95
Baseline Poverty	
• Below poverty line	0.25
• Above poverty line	0.75
Baseline Region	
• NE	0.17
• MW	0.26
• S	0.38
• W	0.18
Baseline Urbanicity	
• Urban	0.80
• Rural	0.20
Note: Figures come from the first multiply imputed dataset. Standard deviation in parentheses.	

Table C-2: Time-varying measured, NLSY79, 1979-2006

	Time 1	Time 5	Time 10	Time 15
Age	19.73	23.82	29.13	35.25
	(1.16)	(1.13)	(1.21)	(1.34)
Marital Status				
• Married	0.17	0.38	0.53	0.58
• Not married	0.83	0.62	0.47	0.42
Cohabitation				
• Not cohabiting with a partner	0.97	0.93	0.92	0.92
• Cohabiting with a partner	0.03	0.07	0.08	0.08
Children in HH				
• At least 1 child in HH	0.18	0.36	0.57	0.69
• No children in HH	0.82	0.63	0.43	0.31
Family Income (logged)	9.40	9.57	9.97	10.13
	(1.27)	(1.27)	(1.45)	(1.68)
Weeks unemployed	17.26	13.17	9.67	13.89
	(19.33)	(20.20)	(17.59)	(28.22)
Health				
• Health Problems	0.04	0.04	0.05	0.07
• No health problems	0.96	0.96	0.95	0.93
Poverty				
• Below poverty line	0.24	0.22	0.18	0.20
• Above poverty line	0.76	0.78	0.82	0.80
Region				
• NE	0.17	0.17	0.16	0.15
• MW	0.25	0.24	0.25	0.25
• S	0.38	0.38	0.39	0.40
• W	0.19	0.20	0.20	0.20
Urban/Rural				
• Urban	0.79	0.81	0.80	0.77
• Rural	0.21	0.19	0.20	0.23
Cumulative number of changes in marital status	0.00	0.36	0.80	1.13
	(0.00)	(0.59)	(0.90)	(1.14)

Note: Figures here come from the first multiply imputed dataset. Standard

deviation is in parentheses.

Table C-3: Proportion of time spent married, NLSY79, 1979-2006

	Mean
Average proportion of time in study spent married	0.45
0%-19%	0.31
20%-39%	0.12
40%-60%	0.16
61%-80%	0.19
80%-100%	0.22
Average # of changes in marital status	
0 changes in marital status	0.31
1 changes in marital status	0.41
2 changes in marital status	0.15
3+ changes in marital status	0.12

Table C-4: Weights

	Mean	SD	Min.	Max
Stabilized IPT weight (sw)	0.99	0.39	0.33	2.61
Stabilized censor weight (cw)	0.99	0.05	0.83	1.12
Final weight (sw * cw)	0.99	0.40	0.27	2.92
Note: All figures here come from the first imputed dataset. All weights are truncated at the 1 <sup>th</sup> % and 99 <sup>th</sup> percentile to reduce the impact of outliers.				

Table C-5: Association between marital duration and depressive symptoms

	(1) Unadjusted model	(2) Conventional model	(3) Marginal structural model
Marital duration	-0.25*** (0.04)	-0.13** (0.05)	-0.20*** (0.04)

Note: The unadjusted model regresses CES-D score on marital duration. The conventional model regresses CES-D on marital duration, time-invariant characteristics, and time-varying characteristics. The marginal structural model regresses CES-D on marital duration and is weighed by the final weight (the product of the IPT and censor weight). Coefficients and standard errors are combined from five multiply imputed datasets. Robust standard errors in parentheses. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table C-6: Association between cohabitation and marital duration on depressive symptoms

	(1) Unadjusted model	(2) Marginal structural model
Cum. Exposure		
Married	Ref.	Ref.
Cohabiting	0.47*** (0.10)	0.31*** (0.11)

Note: The unadjusted model regresses CES-D score on relationship status. The marginal structural model regresses CES-D on relationship status and is weighed by the final weight (the product of the IPT and censor weight). Coefficients and standard errors are combined from five multiply imputed datasets. Robust standard errors in parentheses. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Table C-7: Association between marital duration and depressive symptoms, stratified by gender, parental education, and race/ethnicity

	$\beta$ for marital duration	P-value for interaction
<b>Gender</b>		
Men (ref)	-0.20 (0.05)	-
Women	-0.22 (0.05)	0.62
<b>Parental Education</b>		
Non-college educated (ref)	-0.21 (0.04)	-
College-educated	-0.20 (0.09)	0.53
<b>Race/Ethnicity</b>		
White (ref)	-0.11 (0.06)	-
Black	-0.33 (0.07)	0.02
Hispanic	-0.26 (0.10)	0.13

All estimates from the second column come from separate marginal structural models regressing CES-D on marital duration and is weighed by the final weight (the product of the IPT and censor weight). Coefficients and standard errors are combined from five multiply imputed datasets. Robust standard errors in parentheses. P-values for the third column from marginal structural models regressing CES-D on marital duration interacted with different socio-demographic characteristics (e.g., marital duration X gender).

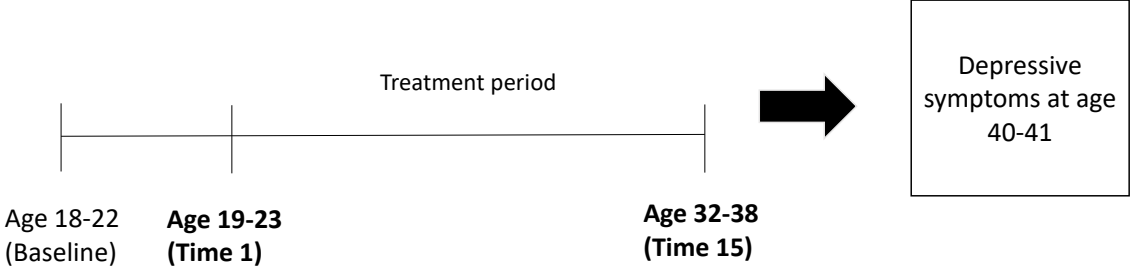


Table C-8: Association between marital duration and depressive symptoms, stratified by age

(1)	
Marginal structural model	
First half of study (Wave 1-7)	
Marital Duration	0.01 (0.05)
Second half of study (Wave 8-15)	
Marital Duration	-0.18*** (0.04)

Note: Estimates come from a marginal structural model regresses CES-D on relationship status and is weighed by the final weight (the product of the IPT and censor weight). Coefficients and standard errors are combined from five multiply imputed datasets. Robust standard errors in parentheses. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Figure C-1: Diagram of the research study period



Appendix Table C-1: Treatment model regressing marital status on time varying and time invariant measures

Variables	Odds Ratio (SE)
<b><u>Time varying measures (measured at t – 1)</u></b>	
Prior marital status	110.79*** (3.51)
Cohabitation (ref = not cohabitating w/ a partner)	
• Cohabiting with a partner	2.61 (0.10)
Children in HH ( ref = no children)	
• At least one child	1.15*** (0.04)
Family Income	1.00 (0.01)
Weeks spent unemployed	1.00*** (0.00)
Region (ref = NE)	
• NW	1.04 (0.08)
• S	1.11 (0.07)
• W	1.03 (0.08)
Urbancitiy ( ref = rural )	
• Urban	0.88*** (0.03)
Age	1.26*** (0.04)
Age squared	1.00*** (0.00)
Poverty Status (ref = not under poverty line)	
• Under poverty line	0.78*** (0.03)
Health (ref = no health problems)	
• Health problems	0.94 (0.05)
Cumulative number of changes in marital status	1.06***

	(0.02)
<b><u>Time invariant measures</u></b>	
Sex (ref = Male)	
• Female	1.13*** (0.03)
Race (ref = White)	
• Black	0.57*** (0.02)
• Hispanic	1.02 (0.04)
Parental Educ (ref = less than HS)	
• HS only	1.02 (0.03)
• Some College	0.98 (0.04)
• 4 years of college	0.94 (0.04)
• 4+ years of college	0.96 (0.05)
Locus of control	1.00 (0.01)
AFQT score	1.00*** (0.00)
Baseline age	0.52 (0.22)
Baseline age sq	1.02 (0.01)
Baseline children in HH (ref = no children)	
• At least one child	0.84*** (0.04)
Baseline family income	1.02 (0.01)
Baseline not working	1.00** (0.00)
Baseline marital status	1.13*** (0.05)
Baseline urbancity (ref = rural)	
• Urban	0.95 (0.04)
Baseline region (ref = NE)	
• NW	1.05 (0.08)
• S	1.11 (0.07)
• W	1.04

Baseline poverty (ref = impoverished)	(0.08)
• Not impoverished	0.99 (0.04)
Baseline health (ref = no health problems)	
• Health problems	0.91* (0.05)

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Note: The coefficients and standard errors reported here come a pooled logistic from the first multiply imputed dataset. Data is in person-year format. Robust standard errors in parentheses. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

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Appendix Table C-2: Marginal structural models examining the association between marital duration and depressive symptoms using different truncation points for the final weight

	(1)	(2)	(3)
	Final weight truncated at 5%	Final weight truncated at 2.5%	Final weight at truncated 1%
Marital duration	-0.21*** (0.04)	-0.21*** (0.04)	-0.20*** (0.04)

Note: Coefficients and standard errors are combined estimates from 5 multiply imputed datasets; All models include time-invariant measures; Robust standard errors in parentheses. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Appendix Table C-3: Distribution of treatment across different key confounders

Males				
<b>Parental Education</b>	<b>Race</b>	<b>Poverty status</b>	<b>Married</b>	<b>Not Married</b>
Less than HS	White	Impoverished	0.41	0.59
		Not Impoverished	0.56	0.44
	Black	Impoverished	0.10	0.90
		Not Impoverished	0.33	0.67
	Hispanic	Impoverished	0.29	0.71
		Not Impoverished	0.51	0.49
HS only	White	Impoverished	0.27	0.73
		Not Impoverished	0.54	0.46
	Black	Impoverished	0.12	0.88
		Not Impoverished	0.39	0.61
	Hispanic	Impoverished	0.36	0.64
		Not Impoverished	0.52	0.48
Some college	White	Impoverished	0.19	0.81
		Not Impoverished	0.50	0.50
	Black	Impoverished	0.16	0.84
		Not Impoverished	0.39	0.61
	Hispanic	Impoverished	0.22	0.78
		Not Impoverished	0.44	0.56
4 years of college	White	Impoverished	0.09	0.91
		Not Impoverished	0.49	0.51
	Black	Impoverished	0.11	0.89
		Not Impoverished	0.32	0.68
	Hispanic	Impoverished	0.12	0.88
		Not Impoverished	0.53	0.47
4+ years of college	White	Impoverished	0.08	0.92
		Not Impoverished	0.52	0.48
	Black	Impoverished	0.16	0.84
		Not Impoverished	0.36	0.64
	Hispanic	Impoverished	0.15	0.85
		Not Impoverished	0.54	0.46

Note: Figures are based on data from the first imputed dataset for all respondents in the analytic sample. Data is arranged in person-year format. Each figure denotes the row proportion of person-years that were married or unmarried.

		Females		
<b>Parental Education</b>	<b>Race</b>	<b>Poverty status</b>	<b>Married</b>	<b>Not Married</b>
Less than HS	White	Impoverished	0.27	0.73
		Not Impoverished	0.67	0.33
	Black	Impoverished	0.11	0.89
		Not Impoverished	0.41	0.59
	Hispanic	Impoverished	0.36	0.64
		Not Impoverished	0.63	0.37
HS only	White	Impoverished	0.26	0.74
		Not Impoverished	0.65	0.35
	Black	Impoverished	0.09	0.91
		Not Impoverished	0.37	0.63
	Hispanic	Impoverished	0.25	0.75
		Not Impoverished	0.56	0.44
Some college	White	Impoverished	0.27	0.73
		Not Impoverished	0.61	0.39
	Black	Impoverished	0.08	0.92
		Not Impoverished	0.35	0.65
	Hispanic	Impoverished	0.10	0.90
		Not Impoverished	0.45	0.55
4 years of college	White	Impoverished	0.18	0.82
		Not Impoverished	0.59	0.41
	Black	Impoverished	0.13	0.87
		Not Impoverished	0.38	0.62
	Hispanic	Impoverished	0.53	0.47
		Not Impoverished	0.54	0.46
4+ years of college	White	Impoverished	0.17	0.83
		Not Impoverished	0.51	0.49
	Black	Impoverished	0.24	0.76
		Not Impoverished	0.40	0.60
	Hispanic	Impoverished	0.60	0.40
		Not Impoverished	0.62	0.38

Note: Figures are based on data from the first imputed dataset for all respondents in the analytic sample. Data is arranged in person-year format. Each figure denotes the row proportion of person-years that were married or unmarried.



Appendix Table C-4: Association between marital duration and depressive symptoms, missing cases included

	(1) Unadjusted model	(2) Conventional model	(3) Marginal structural model
Marital duration	-0.26*** (0.03)	-0.11** (0.05)	-0.22*** (0.04)

Note: These models impute missing information measures except the outcome variable, resulting in 1,769 additional respondents being added back into the analytic sample. The unadjusted model regresses CES-D score on marital duration. The conventional model regresses CES-D on marital duration, time-invariant characteristics, and time-varying characteristics. The marginal structural model regresses CES-D on marital duration and is weighed by the final weight (the product of the IPT and censor weight). Coefficients and standard errors are combined from five multiply imputed datasets. Robust standard errors in parentheses. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

Appendix Table C-5: Marginal structural models regressing different measurements of depressive symptoms on marital history

	(1)	(2)	(3)
	Binary	Binary	Continuous
	(CES-D $\geq$ 8 cutoff)	(CES-D $\geq$ 7 cutoff)	(CES-D score)
Marital duration	0.54*** (0.06)	0.54*** (0.07)	-0.20*** (0.04)

Note: Coefficients and standard errors are combined estimates from 5 multiply imputed datasets. All models include time-invariant measures and are weighted using the final weight, truncated at the 1th and 99th percentile. Models 1 and 2 estimate logistic regression models and coefficients are presented as odds ratios, while model 3 estimates an OLS model. Robust standard errors in parentheses. \*\*\* p<0.01, \*\* p<0.05, \* p<0.1

## **Chapter 5 : Conclusion**

### **Summary**

The link between social ties and mental well-being has been a classic sociological research topic dating back to Durkheim's classic study on suicide and social integration. Hundreds of studies have sought to examine the association between our families, friends, peers, and our mental health. Much of the literature, however, has been dominated by an implicit binary perspective focused on ultimately providing a definitive, yes/no answer to the question: do social relationships matter? As a result, major substantive areas, including effect heterogeneity, causal mechanisms, and temporality remain under examined. These gaps not only limit our theoretical understanding of a critical social determinant of mental health, but also reduce the effectiveness of possible policy interventions. The overarching goal of this dissertation was to explore these issues, utilizing three prominent sociological hypotheses as case studies.

In Chapter 2, I examined the extent to which depressive symptoms are “contagious,” as suggested by social contagion theory. A number of studies find suggestive evidence for this hypothesis, showing that depression, happiness, and loneliness among family and friends are related to one's own odds of being depressed, happy, or lonely (Cacioppo, Fowler and Christakis 2009, Coviello et al. 2014, Fowler and Christakis 2008, Rosenquist, Fowler and Christakis 2011). However, this line of research has often been criticized for its inability to account for important confounding factors (Cohen-Cole and Fletcher 2008). For instance, individuals with depression may actively choose friends who are also depressed because they share a similarly negative outlook on life. Thus, while these symptoms are related between individuals, this

pattern is the result of self-selection rather than peer influence. Because few studies have been able to deal with these issues, it remains unclear whether depressive symptoms are truly “contagious.”

In this study, I used a within-school between-cohort design to more rigorously test the contagion hypothesis (Hoxby 2000). Data for the analyses came from the National Study of Adolescent Health (Add Health), a longitudinal study that tracks the health outcomes of roughly 20,000 US adolescents over time. My results indicated that exposure to peers with depressive symptoms had a small, positive effect on a student’s own odds of developing depressive symptoms a year later. This effect was particularly pronounced for low-SES and Black students and driven partly by the lower level of social support students perceived they had from classmates with depressive symptoms.

In Chapter 3, I reassessed the reactivity hypothesis, which posits that girls are more relationship-oriented than boys, making them more vulnerable to the depressogenic effects of interpersonal stress (Cyranowski et al. 2000). Numerous studies have tested this hypothesis, but results remain mixed (Aneshensel 1992). Much of the existing research, however, is limited at least two ways. The first relates to methodological limitations. Most prior studies rely on small, non-representative, cross-sectional data and conventional regression approaches, which are highly sensitive to the variables included in the models. Second, few studies using representative data have actually tested the underlying mechanism implied in the reactivity hypothesis—that is, girls’ greater response to interpersonal stress compared to boys.

In this study, I used data from the Add Health to estimate first-difference, person fixed-effects models (Allison 2009) In essence, these models examine how changes in the number and quality of social relationships relate to changes in depressive symptoms within a person. Results

indicated that peer and school-based interpersonal stress had a similarly negative impact on well-being for boys and girls. Familial interpersonal stress, however, was more strongly associated with depressive symptoms for girls. An exploratory mediational analysis indicated that this gender difference was attenuated by girls' stronger emotional response to familial ties. Taken together, these results suggest that girls may be more reactive than boys to certain types of interpersonal stress, especially those related to the family.

In Chapter 4, I tested the hypothesis that the benefits of marriage on mental health become stronger over time. While the protective effect of marriage against mental health problems is well-documented (Umberson, Thomeer and Williams 2013), life course theory speculates that the beneficial health effects of social ties such as marriage become stronger over time (Umberson, Crosnoe and Reczek 2010). Few studies, however, have empirically tested this claim. Instead, most work conflates all married people, from newlyweds to long-term couples, into a single category, ignoring the potentially important role of marital duration.

In this study, I used 16 waves of the NLSY79 to examine the association between individuals' marital history during early adulthood (age 19-23 to age 32-38) and their mental well-being at midlife (age 40-41). To address issues related to time-varying confounders, I estimated marginal structural models, which can adjust for these variables without blocking indirect pathways through which marriage influences mental health (Robins, Hernan and Brumback 2000). Results indicated that increased exposure to marriage was positively associated with mental well-being later in life, even after adjusting for a host of time-varying and time-invariant characteristics. Findings from conventional models were substantively similar, though the magnitude of the coefficient was significantly smaller, highlighting the value of improved estimation tools. Secondary analyses also indicated that (1) the cumulative benefits of marriage

may be stronger for Blacks than Whites and (2) long-term cohabitation may not offer the same cumulative benefits as marriage.

Considered collectively, these papers directly highlight the limitation of the binary perspectives. Social groups differentially respond to social ties and their potential impact may depend to some degree on how long they have been maintained. Social ties also operate through a multitude of overlapping channels that may vary depending on the type of relationship examined. All this suggests that simply examining whether social ties matter in a black and white manner misses an important opportunity to extend our understanding around how and under what conditions relationships are most salient.

Findings from all three empirical chapters must be viewed in light of several important limitations. First, many of the key measures relied on self-reported information. For instance, across all three studies, mental-well-being was measured using a self-reported scale (CES-D). Though common in social science and health research, self-reported items are subject to measurement issues such as social desirability bias. Second, all of the data used in this dissertation relied on older nationally representative data. For instance, the Add Health began in 1994-1995 while the NLSY79 began in 1979. Social norms and behaviors may have fundamentally changed since these data were first collected, potentially limiting their applicability to the world today. This may be especially true for the findings in Chapter 4, as the nature of marriage has changed substantially since the late 1970s. Third, while the research designs employed throughout the dissertation attempt to address important sources of bias, it is important to note they are still non-experimental and thus do not yield true causal estimates. For instance, while first difference models in Chapter 3 address time-invariant covariates, time-varying factors may still bias results. Thus, while they may be more accurate, it is still important

to view results as associations. Fifth, several of the studies relied on crude measures to approximate complex constructs. For instance, Chapter 2 sought to measure cohort-average mental health using a single item, while Chapter 3 did the same for emotional reactivity. This shortcoming is mainly a function of data limitations.

### **Research implications**

Notwithstanding these limitations, these studies collectively offer several broad substantive implications for researchers interested in social ties and health. First, they suggest that relationships may not have a uniform impact on all individuals. Thus, researchers should to move beyond an “average effect” approach and to explore who is most impacted by social ties. Nonetheless, sub-group analyses should not simply undertaken without theoretical justification, since the risk of Type-I error increases substantially with additional tests. For instance, Chapter 2’s focus on gender differences was motivated by the long and contentious debate around reactivity and depression. This also implies that there should be also be greater efforts focused on understanding how social relationships are differentially experienced by different groups. This theoretical development could draw from insights across a range of fields, including history, psychological, and economics. Taking such an approach will help researchers identify meaningful differences between groups and understand the sources the heterogeneity.

Second, as indicated in Chapter 4, social ties may have a cumulative impact on our physical and mental well-being. It would thus be useful for researchers to further explore and build on this idea, by, for instance, considering the long-term association between other types of social ties, such as close friendships or relations with family members, and well-being. Likewise, it would be useful to discern what characteristics help give rise to growing benefits. For instance, it is unlikely that staying in a tumultuous marriage long-term offers the same benefits as a long-

term marriage characterized by love and commitment. Conducting such analyses, however, requires detailed, long-term, longitudinal data on individuals' relationships. Therefore, important key challenge for researchers is to find ways to collect such information in a comprehensive yet cost-efficient manner. Theoretically, a focus on cumulative impact also implies a greater need for research to adopt a life-course orientation. That is, it is important for researchers to recognize that relationships are not static features of our social lives, but rather phenomena that have lasting effects over long periods of time.

Third, these papers highlight the clear need to further investigate mechanisms. An influential review article notes that while there are an overwhelming number of theories on social relationships, relatively few studies actually tested their existing. Again, part of the key challenge is related to data; as noted above, several of the studies in this dissertation, which relied on well-known national datasets, only had measures approximating theoretical mechanisms in the social relationship literature. Thus, an important next step for researchers is to more actively include well-validated scales and instruments in data collection efforts. It is also important to consider these mechanisms within the context of new technologies (e.g., social media), as these have become a pervasive new mode of communication, especially for younger adults and adolescents.

These papers also offer useful methodological tools that researchers may consider adopting moving forward. The within-school between-cohort design utilized in Chapter 2 is a common approach utilized in economics (and increasingly sociology) to estimate peer effects. While I focused on mental well-being, researchers with access to data on successive cohorts can extend this method to examine the impact of group-level characteristics on any number of outcomes. Chapter 3 utilized first-difference fixed effects models. These can be easily implemented by researchers interested in addressing bias stemming from time-invariant



confounders, both measured and unmeasured. Chapter 4 employs marginal structural models, which attempt to properly account for time-varying characteristics. These have been increasingly utilized in sociological and health research and offer researchers with longitudinal data the ability to more accurately estimate the long-term association of an exposure, including but not limited to social relationships.

### **Policy implications**

Aside from guiding future research, this dissertation may also have important policy implications, especially as it relates to adolescent mental health. Mental health issues, including depression, psychological distress, and suicidal behavior, have risen dramatically among American adolescents over the past decade (Twenge et al. 2019). In response, health practitioners and policymakers have increasingly sought to develop different policy strategies and interventions to slow this growing public health issue. One popular (and controversial) approach involves the adoption of mental health screenings within schools (McCormick et al. 2009; Dowdy, Ritchey and Kamphaus 2010). These may enable early identification and intervention for “at-risk” youth, which may ultimately slow the progression of mental health conditions and reduce their long-term impact on youth’s well-being. Despite their potential public health value, critics contend that mental health screenings and interventions carry important risks, including: being overly invasive, being outside the scope of the schools’ responsibilities, and potentially stigmatizing and overmedicating students.

The findings from this dissertation suggest that one practical way to determine students’ risk for mental health problems may be to assess their relations with peers, family, and social environment. Teachers and school health professionals, for instance, might administer questionnaires that track students’ sense of connectedness with those around them. It may also be

useful to include questions related to social media usage, as this is an important medium through which adolescents communicate with one another. Upon identifying students “at-risk” of becoming socially disconnected, schools might develop practical interventions aimed at encouraging greater social engagement. For instance, schools might recommend these youth join social clubs or extracurricular activities, such as sports teams. Schools might also consider encouraging close social ties (e.g., family members) to participate in social activities with these youth, as this has the potential to create a “contagion” effect within these social networks. Such a strategy is consistent with recent recommendations set out by the Centers for Disease Control, which describe improving “school connectedness” as a viable way of improving adolescent health outcomes (Center for Disease Control 2009).

Developing screenings around social connectedness is not meant to replace more traditional mental health tools. Indeed, integrating items around relationships may complement standard approaches and offer a more holistic view of youth’s risk of mental health issues. Nonetheless, in situations where there is a lack of support for traditional mental health screenings, social relationships-based screeners and interventions may be viable as stand-alone alternatives, as they provide useful information while avoiding many of the limitations of mental health screenings. Social relationship based questions, for instance, tend to be less invasive than the ones posed in many traditional mental health screeners: asking adolescents about how frequently they hang out with friends is arguably less controversial than asking them about illicit drug use. Interventions also focus on social integration rather than medication—a major concern for parents. Finally, while some question whether schools should be providing mental health services, it is certainly within their purview to help students develop a sense of attachment to their teachers, peers, and school community.

Still, for social connectedness based screening and interventions to materialize, several important research questions must be addressed. First, how should social connectedness be conceptualized and subsequently measured within the school setting for adolescents today? While theories and scales exist, it is unclear the extent to which these adequately capture the nature of social relationships for youth today, especially given the rise of new modes of communication such as social media. This question requires further theoretical development, along with rigorous validation of instruments within classrooms. Second, what are the optimal ways to build greater ties between disconnected youth and those around them? While no “one-size-fits-all” approach will likely emerge, it is important to develop a realistic set of best practices that policy makers and school officials might be able to utilize during interventions. This question similarly merits further research.

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