2016

# The Social Contagion of Adolescent Depression: Applying a Differential Susceptibility Model

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THE SOCIAL CONTAGION OF ADOLESCENT DEPRESSION:
APPLYING A DIFFERENTIAL SUSCEPTIBILITY MODEL

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Sociology

by
Win Guan
B.A., Louisiana State University, 2011
M.A., Louisiana State University, 2013
May 2016

For my grandpa.

## Acknowledgements

I would first like to thank Dr. Troy Blanchard for providing invaluable insight and wisdom during my time in graduate school. You gave me independence to make mistakes but directions to find my way. The milestones that I have reached and the paths that I have taken are a direct result of your advice, your guidance, and your support.

I would also like to express my appreciation for Dr. Heather Rackin, Dr. Samuel Stroope, and Dr. Yoshinori Kamo. Thank you for serving on my committee and providing assistance and advice throughout each step of this process. Additionally, I would like to acknowledge and thank my Master's advisor Dr. Susan Dumais. You have my been my mentor since I met you as my undergraduate advisor. Despite the many miles between us now, you have remained a significant source of inspiration, motivation, and encouragement.

And a special acknowledgement to Danielle for being such an incredibly giving and supportive colleague and friend. You were always one simple call away for presentation rehearsals, draft edits, a much needed happy hour, and even just a couch to crash on. This dissertation and the completion of my degree truly would not have been possible without you.

Finally, I want to express my deep gratitude for my family. You have shown unconditional love and support. Thank you for your patience and your willingness to allow me to pursue the answers to the many questions with which I used to bother you. And to my rocks: Cristina and Taylor. You ground me every single day of my life and for that, I am eternally grateful.

This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan

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**Abstract**

Recent research investigating social risk factors of depression has found evidence for a social contagion effect. The research comes from a surge in popularity of using social network analyses to examine the *spread* of various health outcomes such as obesity, smoking, substance use, and sleep. Although the finding of depressive contagion represents a significant contribution to the literature on the social etiology of depression, this is only the first step in providing meaningful research useful for the practical application of curbing the growing rates of depression especially among adolescents. Rather than simply acknowledging the existence of contagion effects, researchers must begin to answer the question of whether certain individuals or environments are more susceptible to the effects of depressive contagion. As a result, this dissertation applies a differential susceptibility model to examine three moderators of depressive contagion: social network structure, racial/ethnic identity, and genotypic variation in the serotonin transporter (SLC6A4) gene. The research in this dissertation uses data from the National Longitudinal Study of Adolescent Health (Add Health).

First, the results reveal that adolescents who are popular and/or embedded in dense peer networks are more susceptible to depressive contagion. Additionally, depressive contagion is more salient in schools characterized by dense social networks and high reciprocity in social ties. Second, racial homophily plays an important role in the effect of depressive contagion. Adolescents embedded in racially homophilous peer networks are more susceptible to depressive contagion. Further analysis shows that this effect applies primarily for Asians and Hispanics. Finally, results indicate a significant gene-environment interaction (GxE) effect between a polymorphism in the serotonin

transporter system (5-HTTLPR) and depressive contagion. Adolescents carrying one or

two short alleles in the 5-HTTLPR are more susceptible to depressive contagion.

**Chapter 1: Introduction**

Depression is a chronic disease associated with various comorbidity outcomes such as anxiety disorder, substance use disorder, and impulse control disorder (Kessler et al. 2003). Additionally, risk of depression dramatically increases during adolescence (Hankin et al. 2015) as a result of the intersection between several biological, social, and physiological processes during this stage. Kessler et al. (2003) find that this risk increase is more dramatic for recent cohorts as compared to cohorts of the past. In fact, according to a report by the World Health Organization (WHO), the number one cause of illness and disability among adolescents worldwide is depression (World Health Organization 2014).

Although the etiology of depression includes a long list of biological, psychological, and social factors, recent research shows evidence suggesting a contagion effect whereby depression can *spread* within a social network through the pipelines of social relationships (Rosenquist et al. 2011). In other words, individuals embedded in peer networks characterized by high levels of depression are more susceptible to becoming depressed (Hogue and Steinberg 1995; Prinstein 2007; Conway et al. 2011; Cheadle and Goosby 2012). Further extension of this finding regards research on moderators of this contagion effect. This literature applies a differential susceptibility model to understanding factors that may enhance vulnerability or resilience to depressive contagion. Examining this line of research presents valuable information for public health researchers in implementing network-driven intervention strategies.

As a result, the purpose of this dissertation is to further existing literature on depressive contagion by incorporating three factors that significantly moderate the social contagion of adolescent depression. This research rests upon the differential susceptibility

perspective suggesting that the salience of depressive contagion is not uniform in individuals nor environments. Three separate studies are included in this dissertation:

Study 1 (Chapter 2): The first study in this dissertation aims to demonstrate the significance of social network context in moderating depressive contagion. Prior research suggests that studies aimed at finding evidence for contagion must adjust for potential homophily (selection) and shared environment (spurious) effects. However, shared environments can play an important role in determining the salience of contagion given the nested nature of social networks within which contagion occurs. In this study, I hypothesize that the effect of depressive contagion varies depending on structural characteristics at the peer network and school social network levels.

This study presents two important contributions. First, much of the research on moderators of contagion focus on individual characteristics such as psychosocial or demographic measures. Empirical studies of contextual moderators (Prinstein 2007) are largely non-existent due to limitation in data availability. In order to study contextual moderators, data samples must include a large enough group-level sample size in order to study variation in context in addition to variation in individual attributes. The Add Health survey utilized in this study presents a unique opportunity for this research endeavor due to its large sample size of adolescents nested within schools. Second, this study demonstrates that the spread of social phenomenon through social network ties depends on the structural composition of these network ties at both the ego-social and broader social network levels.

Study 2 (Chapter 3): A basic application of the differential susceptibility model to depressive contagion includes the investigation of demographic differences in the salience of depressive contagion. For instance, despite conflicting results, most research

(Hogue and Steinberg 1995; Conway et al. 2011; Cheadle and Goosby 2012) acknowledges the potential for variation in depressive contagion among males and females. Missing in this literature however, is the examination of racial/ethnic group differences. For instance, there is no mention of racial/ethnic group variation in Joiner and Katz's (1999) meta-analytical review of more than 40 studies on depressive contagion. Similarly, Brechwald and Prinstein (2011) fail to recommend race as a significant moderator of depressive contagion despite mentioning other demographic variables such as gender and age.

As a result, this study presents an empirical investigation into two potential aspects of race that can significantly moderate the salience of depressive contagion. First, I examine whether depressive contagion is different among racial/ethnic groups, specifically between black adolescents and Hispanic and Asian adolescents. This research question is guided by prior literature suggesting variation in minority experiences between the black population and the Asian and Hispanic population (Quillian and Campbell 2003). Second, I investigate the role of racial homophily in moderating the effect of depressive contagion. This effectively shifts the significance of race as not simply a feature of the individual being influenced but also a feature of the peer network regarding racial composition.

Study 3 (Chapter 4): The final study in this dissertation integrates research on the moderators of social contagion with the gene-environment interaction (GxE) paradigm. In this study, I analyze whether a functional polymorphism (5-HTTLPR) in the serotonin transporter system moderates the effect of depressive contagion. In a previous empirical study that has since been cited over 6,000 times, the 5-HTTLPR polymorphism is shown to moderate the effect of stress on depression (Caspi et al. 2003). Individuals carrying one

or two S alleles in 5-HTTLPR are shown to become more depressed when faced with "stressful life events." Subsequent replications (e.g. Eley et al. 2004; Karg et al. 2011) and meta-analytic reviews (Munafo et al. 2009; Risch et al. 2009) suggest mixed results. Although I acknowledge the importance of replication, I argue that the GxE literature is limited in its lack of alternative measurements for environmental exposure, the "E" component of the GxE effect, given the vast literature on social risk factors of depression.

**Chapter 2: Contextualizing Depressive Contagion:**
**Applying a Multilevel Network Approach**

## 2.1 Introduction

According to the *Health for the World's Adolescents* report (World Health

Organization 2014) conducted by the World Health Organization (WHO), depression is

the number one cause of illness and disability among adolescents globally. Additionally,

a National Institute of Mental Health survey (National Institute of Mental Health 2007)

estimates that approximately 11% of adolescents in the United States have experienced

depression by the age of 18 while the National Survey on Drug Use and Health in 2012

estimates that approximately 9.1% of adolescents 12-17 have experienced at least one

major depressive disorder in the past year (Substance Abuse and Mental Health Services

Administration 2013). Moreover, depression has significant effects on the adolescent's

self-esteem, academic performance, and interpersonal experiences which in turn can

increase depression within the adolescent (Joyner and Udry 2000).

An emerging literature on adolescent depression focuses on the explanations for

the similarities in the level of depression between adolescents and their immediate

friends. One explanation for this similarity is through the depressive contagion

mechanism suggesting that adolescent depression is significantly influenced by friends'

level of depression. However, this contagion effect is often conflated with two other

explanations: homophily and shared environments (Shalizi and Thomas 2011).

Homophily assumes that depressive similarities in friendship networks are caused by

individuals selecting friendships based on similar levels of depression. Shared

environment effects point to spurious effects caused by shared contexts in which the

friendship network is embedded such as a classroom, neighborhood, or school. Studies

aimed at disentangling contagion effects from homophily and shared environments use various statistical methods including longitudinal regression models (e.g. Conway et al. 2011) and stochastic actor-based models (e.g. Zalk et al. 2010b; Cheadle and Goosby 2012). However, few studies (e.g. Prinstein 2007) have investigated the role of environments in moderating the effect of depressive contagion.

This study aims to demonstrate the utility of a longitudinal multilevel mixed effects model in examining contextual moderators of depressive contagion. According to Prinstein (2007), contextual moderators "refer to aspects of the environment in which peer contagion potentially may occur." Only one study to our knowledge examines contextual moderators of depressive contagion[1] (Conway et al. 2011). Using a measure of received friendship nominations, the authors find that depressive contagion is more salient in less popular students. Although this finding is a significant contribution to the literature, there is still much to be explained for other contextual moderators such as network structural characteristics.

Furthermore, much of prior research on contextual moderators of contagion (e.g. Haynie 2001; Prinstein 2007; Conway et al. 2011) examines elements at purely the "microsocial," or local peer group level (Dishion 2013). This tendency ignores higher level moderating effects where the adolescent is not only nested within a local peer group, but also a classroom, a school, a community, and so forth. Dishion (2013) refers to these higher level social contexts as the "macrosocial." As a result, we investigate several

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[1] Contextual moderators of the peer contagion of other outcomes do exist. For example, Haynie (2001) finds that popular students are more susceptible to peer contagion of delinquency. Additionally, Rambaran, Dijkstra, and Stark (2013) find that peer contagion of risk attitudes is more salient when embedded in classrooms with stronger associations between peer status and risk attitudes.

measures of social context at both the microsocial and macrosocial levels. The multilevel

model used in this analysis presents a unique opportunity for this investigation. First, this

model enables us to replicate prior findings of depressive contagion by statistically

adjusting for 1) school-level effects that may bias parameter estimates of depressive

contagion (Raudenbush and Bryk 2002) and 2) prior levels of depressive symptoms

which in effect produces parameter estimates of change in ego depressive symptoms from

Wave 1 to Wave 2 as a result of friends' level of depressive symptoms during Wave 1

(Christakis and Fowler 2013).

Second, this model allows for the use of a large-scale, nationally representative

sample of adolescents nested within schools from the first and second waves of the

National Longitudinal Study of Adolescent Health (Add Health). This allows for a

simultaneous test of both individual-level interactions between friends' depressive

symptoms and microsocial context and cross-level interactions between friends'

depressive symptoms and macrosocial context. Rather than viewing these social contexts

as shared environments to be controlled, we examine their potential as social buffers or

vulnerabilities to depressive contagion. In the following sections, we review the literature

on the association between health and social relationships and depressive contagion.

**2.2 Social Relationships and Health**

The association between social relationships and health has long been established

both empirically and theoretically (House et al. 1988; Umberson and Montez 2010).

Social relationships consist of potential features such as the level of social integration, the

quality of the social relationship, and various social network characteristics (Umberson

and Montez 2010). These features detail three significant components of the association:

existence of social relationships, the nature of those relationships, and the context within

which those relationships are embedded. First, those individuals who are less socially isolated and more socially integrated have better health outcomes (House et al. 1988). While social isolation simply means the lack of social relationships, social integration is defined as the "level of involvement" with informal and formal relationships (Umberson and Montez 2010). Informal relationships can include marriage or family structure while formal ones can include participation or membership in various religious or volunteer organizations.

Second, the nature of social relationships can produce both favorable and unfavorable health outcomes (House et al. 1988; Haynie 2001; Baller and Richardson 2009). They can be positive in that social and emotional support is offered through these social relationships that can help buffer stressful life-events (Pearlin et al. 1981; Wethington and Kessler 1986; Pearlin 1989). Additionally, they can present opportunities for the social control, or regulation of unhealthy behavior and the social learning of healthy ones (Umberson 1987). However, social relationships can also act as significant sources of role strain and role conflict. For instance, while marriage in general can provide social integration, a bad marriage reduces physical health by compromising immune and endocrine systems (Umberson et al. 2006). Additionally, in the same way that social relationships serve as pipelines for positive health resources, they can present opportunities for the social learning of unhealthy behavior. This aspect of negative influence has been researched extensively using explanations of differential association theory (Sutherland 1947) and social learning theory (Akers et al. 1979).

Finally, social relationships are embedded within social contexts or environments. These can refer to structural units such as the classroom or school within which students are nested (Entwisle et al. 2007) or it can be the web of social ties that ultimately form

into a social network (Umberson and Montez 2010). The examination of contextual effects on individual phenomena has dramatically increased in the past several decades (Entwisle et al. 2007). For instance, researchers adopt census demographic measures at the neighborhood level to examine contextual effects on individual-level self-reported health while controlling for other individual-level covariates. Using multilevel models (Raudenbush and Bryk 2002), researchers are then interested in the effects of neighborhood above and beyond individual covariates (Blau 1960). Additionally, cross-level interactions may be examined to test whether variation in individual effects exists from one neighborhood to another. While this is an empirically rich area of sociological research, this same methodological investigation rarely exists when examining the social interactional aspect of social context (Entwisle et al. 2007) due in large part to the relative lack of large-scale social network data.

## 2.3 Social Contagion of Depression

More recently as a result of the increase in the availability of social network data, an emerging literature focuses on the network autocorrelation of depression (Hogue and Steinberg 1995; Prinstein 2007; Conway et al. 2011; Cheadle and Goosby 2012). This phenomenon can be traced to three social causes: homophily, contagion, and shared environment effects. (Christakis and Fowler 2013). Homophily refers to the idiom of "birds of a feather flock together." Individuals form ties based on similarities in their level of depression. The second cause of network autocorrelation involves the spread of phenomena among individuals within a social network, or what is often referred to as social contagion (Rosenquist et al. 2011). The final cause of network autocorrelation is shared environments, essentially spurious effects from the individuals attending the same school or residing in the same community.

Since these effects are often conflated with each other (Shalizi and Thomas 2011), many studies have utilized stochastic actor-based models, using the RSIENA package developed by Snijders et al. (2010), to simultaneously examine the effects of homophily and contagion. A basic assumption of regression analysis is that actors in the analysis are independent from one another resulting in uncorrelated errors. Actor-based models depart from this potentially "reductionist" assumption and directly analyze interactions between actors and behaviors. Moreover, whereas longitudinal regression models estimate the effects of network predictors at time *t* on the behavioral outcome at time *t+1* adjusting for the lagged effect of the behavioral outcome at time *t,* actor-based models simultaneously estimate network and behavioral change. Therefore, actor-based models are able to directly analyze the effects of network on behavior and potential feedback loops (El-Sayed et al. 2012). Despite the advantages, SIENA models require complete and longitudinal network data that is seldom available. For instance, although the Add Health survey collected complete network data for all 140 schools in the core probability survey in the Wave 1 in-school questionnaire, only 16 of these schools, referred to as the "saturated" oversample, have complete network data in subsequent waves. Additionally, only 9 schools within the saturated oversample prove suitable for actor-based models (for further discussion, refer to Cheadle and Goosby 2012). As a result, it is apparent that longitudinal regression models remain useful in analyzing currently available, large-scale sample surveys.

Regardless of whether research utilizes actor-based models or longitudinal regression analysis, studies consistently show support for the effect of contagion in explaining similarities of depression in connected individuals (Hogue and Steinberg 1995; Stevens and Prinstein 2005; Prinstein 2007; Zalk et al. 2010a; Zalk et al. 2010b;

Conway et al. 2011; Cheadle and Goosby 2012). For instance, Cheadle and Goosby (2012) utilize stochastic actor-based models to analyze contagion and homophily in seven small schools. The authors find evidence supporting peer contagion effects of depression above and beyond social selection and social exclusion based on individual levels of depression. Conway et al. (2011) also find support for peer contagion effects using a multilevel model of more than 600 adolescents nested within peer groups.

Furthermore, researchers find that the effect of depressive contagion is moderated by several characteristics. Prinstein (2007) presents a theoretical typology of these potential moderators: target-oriented, prototype, relationship-oriented, and contextual. Target-oriented moderators refer to characteristics of the individual being socially influenced. For instance, Hogue and Steinberg (1995) find that peer contagion effects are stronger for male adolescents. Cheadle and Goosby (2012) echo this finding using actor-based models. On the other hand, prototype moderators refer to characteristics of the individual exerting influence on the target. Prinstein (2007) finds that male adolescents are more susceptible to peer influence in depression by friends who are perceived to be popular. Relationship-oriented moderators refer to characteristics of the relationship between the target and prototype such as strength of ties or directionality. In a study of almost 400 adolescents, Stevens and Prinstein (2005) find support for peer contagion of depression only between adolescents who were characterized as best friends.

The final type of moderator concerns the environment within which the contagion effect is embedded. Unfortunately, research on contextual moderators of depressive contagion is largely non-existent. To our knowledge, only one study examines a contextual moderator of depressive contagion. In a study of more than 600 adolescents, Conway et al. (2011) find that popularity, measured as received nominations, is a

significant moderator of depressive contagion. The authors find that popular adolescents were less susceptible to depressive contagion. According to Conway et al. (2011), this finding suggests that popular students are less likely to conform to group norms given their already high level of status. On the other hand, less popular students are more motivated to conform in order to achieve greater status. Although this explanation is conceivable, research on contagion of other phenomena suggests the opposite relationship (Aloise-Young et al. 1994; Urberg et al. 2003; Haynie 2001). Popular students may instead be more susceptible to peer influence due to either a greater exposure to contagion or an increased motivation to maintain their status.

Consequently, it appears that more research on contextual moderators of depressive contagion is necessary. We suggest a potentially fruitful area of investigation involving structural characteristics of the microsocial context and the school-level macrosocial context. This suggestion is motivated by prior theoretical research asserting that social relationships are embedded within a web of other relationships that ultimately form a social interactional structure (Umberson and Montez 2010). As a result, we suggest that peer contagion of adolescent depression does not exist in a vacuum but rather within an environment with differential characteristics that can have significant influences on the saliency of peer contagion.

**2.4 The Present Study**

The following analysis examines contextual moderators of adolescent depressive contagion. First and foremost, we intend to replicate prior research on depressive contagion using a multilevel random effects model. Therefore, we expect that the average level of depressive symptoms among an adolescent's peer network is significantly associated with the adolescent's own level of depressive symptoms even after controlling

for the potential effects of formal and informal social relationships and basic sociodemographic characteristics.

The next series of hypotheses concern the primary focus of this study: contextual moderators of depressive contagion. We operationalize context as two separate levels consisting of the microsocial, local ego-centric peer networks, and the macrosocial, school-level structural characteristics (Dishion 2013). First, we test several hypotheses regarding interactions between the average level of depressive symptoms among friends and microsocial density, in-degree centrality (number of received friendship nominations), and out-degree centrality (number of sent friendship nominations). We expect that depressive contagion is more salient in dense peer networks as it allows for more indirect social ties which in turn provides reinforcement of peer group norms (Conway et al. 2011). Moreover, we test the moderating effects of in-degree and out-degree centrality on depressive contagion. We do not hypothesize a specific direction for the association as prior research has shown varying results for in-degree centrality (Aloise-Young et al. 1994; Haynie 2001; Urberg et al. 2003; Conway et al. 2011) and no study to our knowledge has examined the effect of out-degree centrality.

Second, we test hypotheses regarding cross-level interactions between friends' average level of depressive symptoms and school mean level of depressive symptoms, network size, density, and mutuality. Due to the lack of prior literature on school-level moderators of peer contagion, we do not suggest any one directional moderation over another. However, we suspect that depressive contagion would be more salient in schools characterized by small, dense networks with high levels of mutuality. Moreover, we suspect that the effect of depressive contagion is more notable in schools with higher

average levels of depressive symptoms. These schools essentially provide reinforcement of peer group norms but at a higher level within which peer groups are nested.

**2.5 Data**

The analysis in this study uses data from the National Longitudinal Study of Adolescent Health (Add Health). This study follows a nationally representative cohort of adolescents beginning in grades 7-12. Adolescents were chosen based on a step-by-step stratified sampling process. First, high schools around the country were sampled based on region, urbanicity, size, public/private designation, and ethnicity. Qualifying schools were required to have an 11th grade and a school enrollment of 30 or more students. Subsequently, feeder schools for each high school were also identified. If high schools had multiple feeder schools, a feeder school was randomly selected based on a probability proportional to the feeder school's enrollment contribution to the high school. The final sample consists of 80 high schools and 52 feeder schools nested in 80 communities.

The current analysis utilizes the first and second waves of the Add Health survey including a Wave 1 in-school questionnaire (n=90,118), Wave 1 in-home interview (n=20,745), both conducted between September, 1994 and December, 1995, and Wave 2 in-home interview (n=14,738), conducted between April and August, 1996. Social network variables in this analysis are constructed using friendship nominations gathered from Wave 1 in-school questionnaire. Students in each school were asked to list up to five male friends and five female friends in the in-school questionnaire.

The sample selection for this analysis is first restricted by valid data on the dependent variable measured in the Wave 2 in-home interview (n=14,662) resulting in the elimination of 76 cases. The second restriction required cases to have valid Wave 1

network data including friendship nominations and school-level constructed network characteristics. In essence, this required students to be a part of a school that had at least a 50 percent completion rate for the Wave 1 in-school questionnaire. This restriction further limited our sample to 9,971 students nested within 121 schools. Due to multilevel nature of this study, we further limited our sample to schools with at least 30 students. Finally, we utilize Wave 2 sample weights for both the individual and school levels to account for the complex sampling design of the Add Health survey (cases with missing sample weights were eliminated). Our final sample consists of 9,580 students nested within 112 schools with a range from 30 to 836 students per school and an average of 85.5. Missing data on independent variables are imputed using multiple imputation methods in Stata SE 13 (StataCorp 2013).

## 2.6 Analytical Strategy

Given the multilevel nature of our hypotheses, we use a multilevel mixed effects model (Raudenbush and Bryk 2002). Statistically, this allows us to investigate two potential effects of the environment: 1) the effect of school on the average level of adolescent depressive symptoms and 2) the effect of school on the relationship between friends' depressive symptoms and the ego's. The former refers to variation in the intercept of the second level equation (random intercepts model) while the latter refers to variation in slopes of the second level equation (random slopes model). Additionally, it allows us to test cross-level interactions between individual and school-level variables in order to explain between-school variation in depressive contagion.

Secondly, we utilize longitudinal methods in predicting the level of depressive symptoms measured in Wave 2 using independent variables measured in the Wave 1 surveys while controlling for the level of depressive symptoms measured at Wave 1.

Finally, we address the complex sampling design of the Add Health survey by including sample weights throughout the analyses at both the individual and school level. This allows our sample to be representative of the overall U.S. population.

## 2.7 Measures

The outcome of interest in this analysis is respondents' level of depressive symptoms at the time of the Wave 2 in-home interview. The predictor variable in question is network depression, measured by the average level of depressive symptoms among alters in the ego's social network. We also control for prior levels of depressive symptoms, formal and informal social relationships, and basic demographic variables including age, sex, race, parent education, and parent income. Descriptive statistics of all variables included in the analyses can be seen in Table 2.1. The following sections describe the construction of each of these variables.

### 2.7.1 Dependent Variable

During the Wave 2 interview, respondents were presented with 19 statements roughly corresponding to the CES-D scale used in order to measure depressive symptoms in the general population (Radloff 1977). The original CES-D scale consists of 20 statements, 17 of which correspond to statements included in the Add Health data set. Perreira et al. (2005) provides a more in-depth discussion on the differences between the two depression scales.

In response to the statements, the adolescents were then asked to gauge how often they agreed with the statements in the past seven days. Answer choices included four options: never or rarely, sometimes, a lot of the time, and most of the time or all of the time. We code these responses from 0-3 respectively and a factor analysis indicates a single factor structure. The scores are thus aggregated to produce a final measure of

depressive symptoms ranging from 0 to 56 with an alpha level of .859. Due to the skewed nature of the CES-D scale, we log-transform the dependent variable in order to satisfy normality assumptions in regression analysis.

### 2.7.2 Network Context of Depressive Contagion

Measures of the adolescent social network and network depressive symptoms are constructed using the friendship nomination data gathered during the Wave 1 in-school questionnaire. Adolescents were given the opportunity to nominate up to five male friends and five female friends. The nominations utilized in these analyses are limited to friendship nominations sent to and/or received from alters who attend the same school as the ego. First, we construct the measure of network depressive symptoms using the average of all CES-D indices among alters. This includes alters nominated by the ego and alters that nominated the ego. The classification of individuals as alters does not require reciprocation in nominations. Then, if ego has two alter friendships with CES-D scores of 13 and 17, the ego's "friends' depressive symptoms" would be coded as 15.

In order to contextualize depressive contagion, we utilize several social network measures at both the microsocial and macrosocial levels (Dishion 2013). We operationalize the microsocial level as the immediate network of alters that are directly connected to the ego and the macrosocial level as the school-level network in which the ego attends. At the microsocial level, we include measures of network density, in-degree centrality, and out-degree centrality. Network density is measured as the number of dyads that exist within an ego network divided by the total possible number of dyads. In-degree centrality is measured as the number of nominations an ego received from other individuals in the school. Out-degree centrality is measured as the number of nominations the ego sent out to other individuals in the school. Each of these ego-centric measures are

standardized according to the means of each school. This enables our measures to capture ego-centric network characteristics in relation to each school's network potential.

At the macrosocial level, we include three measures: network size, density, and mutuality. First, network size is measured as the total number of students within the school. Second, we measure density as the number of dyads that exist in the school friendship network divided by the total of potential dyads that could possibly exist in the network. This provides an indication of connectedness within a given school network. A network with high density can mean that there is relatively little clustering of social ties in the network. Low density networks can signify either of two possibilities: a high level of clustering or a high level of isolation of individuals. Finally, network mutuality is measured using a mutuality index developed by Katz and Powell (1955). This index measures the tendency for nodes within the network to reciprocate nominations. Additionally, we include a non-network contextual variable of average level of depressive symptoms measured at the school-level.

**2.7.3 Formal and Informal Social Relationships**

In examining adolescent depressive symptoms, it is important to control for the effects of social relationships due to the potential support resources that can come from greater integration into the family, school, and community. We utilize three measures of formal social relationships: religious participation, extracurricular participation, and sports participation. We intend for these measures to account for the social resources these activities may provide in the same way that religious and volunteer organizations would provide social resources for adults.

First, we use a measure of athletic participation and non-athletic extracurricular participation in the school coded "0" for non-participating and "1" for participating in at

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least one activity. Second, we include a measure of religious participation to account for potential social resources coming from outside of the school. Students were asked how often they attended youth activities within a religious context such as youth groups, bible classes, or choir. Answers were coded as "0" for never, "1" for less than a month, "2" for once a month or more, and "3" for once a week or more.

To account for more informal social relationships, we utilize measures of household structure, parental attachment, school commitment, and grade point average. Household structure is measured using self-reported answers of whether students lived with their mother, father, both, or neither. Variables of single parent and other households are constructed using living with both parent as the reference category. To measure school commitment, we use the question asking students, "How hard do you try to do your school work well?" Answer choices were coded from 0 (never try at all) to 3 (try very hard).

Parental attachment is measured using an 8-item scale of questions asking students about their relationship with their parents. For example, students were asked whether they agreed with the statement, "Most of the time, your mother is warm and loving to you." The students were given the option to strongly disagree, disagree, neither agree nor disagree, agree, and strongly agree. We coded these answer choices from -2 to +2 respectively. Finally, to calculate respondents' GPA, self-reported grades for English, math, science, and history during the Wave 1 in-home interview were averaged together to generate a scale between 1-4 (the answer choices stopped at "D or lower").

### 2.7.4 Demographic Variables

Age is computed using suggestions by the Add Health codebook. Respondents were only asked for their birth month and year so therefore the 15th of each month was

Table 2.1 Descriptive Statistics of All Variables in the Analysis (N=9,580 in 112 Schools)

| Variables | Mean | S.D. | Minimum | Maximum |
|---|---|---|---|---|
| Wave 2 Depression[a] | 10.9 | 7.4 | 0 | 56 |
| Wave 1 Depression | 10.9 | 7.4 | 0 | 54 |
| Friends' Depression | 11.0 | 5.8 | 0 | 50 |
| School-level Context: | | | | |
|    Mean Depression | 11.0 | 1.4 | 6.3 | 14.8 |
|    Network Size | 1001.4 | 580.5 | 44 | 2559 |
|    Network Density | .01 | .02 | .01 | .13 |
|    Network Reciprocity | .38 | .05 | .23 | .53 |
| Ego-centric Context: | | | | |
|    Density[b] | .30 | .15 | .03 | 1 |
|    In-degree Centrality[b] | 4.5 | 3.7 | 0 | 30 |
|    Out-degree Centrality[b] | 4.4 | 3.0 | 0 | 10 |
| Informal Social Relationships: | | | | |
|    Single Parent Household | .24 | .42 | 0 | 1 |
|    Other Household | .05 | .22 | 0 | 1 |
|    Parent Attachment | 8.2 | 5.0 | -16 | 16 |
|    School Commitment | 2.3 | .67 | 0 | 3 |
|    Grade Point Average | 2.8 | .75 | 1 | 4 |
| Formal Social Relationships: | | | | |
|    Religious Youth Groups | 1.1 | 1.3 | 0 | 3 |
|    Extracurricular Activities | .52 | .50 | 0 | 1 |
|    Sports Team | .56 | .50 | 0 | 1 |
| Individual Level Controls: | | | | |
|    Age | 15.2 | 1.5 | 11 | 20 |
|    Female | .50 | .50 | 0 | 1 |
|    Black | .18 | .38 | 0 | 1 |
|    Asian | .06 | .23 | 0 | 1 |
|    Hispanic | .14 | .35 | 0 | 1 |
|    Other Race/Ethnicity | .01 | .12 | 0 | 1 |
|    Parent Income[a] | 46.5 | .54 | 0 | 999 |
|    Parent Education | 13.3 | .54 | 0 | 18 |
| School Level Controls: | | | | |
|    Private[c] | .02 | .14 | 0 | 1 |
|    Catholic[c] | .04 | .19 | 0 | 1 |
|    Rural[d] | .12 | .33 | 0 | 1 |
|    Suburban[d] | .59 | .49 | 0 | 1 |

[a]Logged in the analysis
[b]Within-school standardized in the analysis
[c]Public school as reference category
[d]Urban as reference category

Hispanic, or other using white as the reference category. During the in-home interview,

respondents were given the option to mark multiple racial and ethnic categories.

Therefore, to construct the race dummy variable, we followed Add Health's procedure

documented in the codebook for the constructed network variables data set. To achieve

mutual exclusivity, priority was given to Hispanic, Asian, black, white, and other in that

order.

Furthermore, Add Health also conducted an interview with one of the parents or

guardians during the Wave 1 in-home interview. Two measures, parent income and

parent education, were drawn from these interviews for this analysis. Parent income is

measured as annual income in thousands of dollars and logged in order to achieve

normality. Highest level of parent education is originally measured as a categorical

variable with responses such as "high school graduate," "some college," and "college

graduate." We recode "high school graduate" to 12, "some college" to 14, "college

graduate" to 16, and so on. In addition to demographic variables at the individual level,

we control for two measures at the school level indicating whether the school is public

(reference), private, or Catholic and whether the school is located in an urban (reference),

suburban, or rural location.

**2.8 Results**

**2.8.1 Depressive Contagion**

As a preliminary step, we investigate the effect of Wave 1 network depressive

symptoms on Wave 2 ego depressive symptoms using a random intercepts model. Results

in Model 1 of Table 2.2 show a contagion effect indicating that the average level of

depressive symptoms among alters is significantly associated with ego depressive

symptoms above and beyond the effects of control variables and social relationship

variables. Models 2-4 present a step-wise introduction of macrosocial and microsocial

Table 2.2 Mixed Effects Regression: Influence of Peer Depression on Adolescent Ego Depression

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|---|---|---|---|---|---|---|---|---|
| | β | SE | β | SE | β | SE | β | SE |
| Intercept | 1.548*** | .122 | 1.662*** | .154 | 1.683*** | .155 | 2.053*** | .181 |
| Friends' Depression | .005*** | .002 | .005*** | .002 | .005*** | .002 | .008* | .003 |
| School-level Context: | | | | | | | | |
|   Mean Depression | | | -.006 | .010 | -.005 | .010 | -.007 | .009 |
|   Network Size | | | .001 | .003 | .001 | .003 | .001 | .003 |
|   Network Density | | | -.708 | .450 | -.729 | .456 | -.291 | .333 |
|   Network Reciprocity | | | -.112 | .229 | -.112 | .231 | -.178 | .231 |
| Ego-centric Context: | | | | | | | | |
|   Density[a] | | | | | -.004 | .009 | -.004 | .009 |
|   In-degree Centrality[a] | | | | | .020* | .010 | .022* | .010 |
|   Out-degree Centrality[a] | | | | | -.040*** | .010 | -.034*** | .010 |
| Informal Social Relationships: | | | | | | | | |
|   Single Parent Household | | | | | | | .002 | .022 |
|   Other Household | | | | | | | .023 | .039 |
|   Parent Attachment | | | | | | | -.014*** | .002 |
|   School Commitment | | | | | | | -.015 | .014 |
|   Grade Point Average | | | | | | | -.046*** | .014 |
| Formal Social Relationships: | | | | | | | | |
|   Religious Youth Groups | | | | | | | -.015 | .009 |
|   Extracurricular Activities | | | | | | | -.014 | .019 |
|   Sports Team | | | | | | | -.004 | .017 |
| Individual Level Controls: | | | | | | | | |
|   Wave 1 Depression | .052*** | .002 | .052*** | .002 | .051*** | .002 | .047*** | .002 |
|   Age | .016* | .006 | .016* | .007 | .013 | .007 | .007 | .007 |
|   Female | .063*** | .017 | .063*** | .017 | .069*** | .018 | .080*** | .020 |
|   Black | .058* | .026 | .057* | .027 | .050 | .026 | .056 | .029 |
|   Asian | .138* | .055 | .137* | .054 | .134* | .053 | .163** | .059 |
|   Hispanic | .118*** | .031 | .118*** | .031 | .114*** | .031 | .110*** | .032 |
|   Other Race/Ethnicity | .153* | .074 | .153* | .074 | .160* | .074 | .178* | .071 |
|   Parent Income (logged) | -.047*** | .012 | -.047*** | .012 | -.046*** | .012 | -.027* | .012 |
|   Parent Education | -.005 | .004 | -.005 | .004 | -.005 | .004 | -.003 | .004 |
| School Level Controls: | | | | | | | | |
|   Private | .049 | .050 | .065 | .055 | .064 | .055 | .091 | .053 |
|   Catholic | .022 | .022 | .053* | .026 | .054* | .026 | .029 | .022 |
|   Rural | -.064 | .033 | -.047 | .035 | -.047 | .035 | -.031 | .034 |
|   Suburban | -.023 | .023 | -.014 | .023 | -.015 | .023 | -.014 | .026 |
| Random Effects: | | | | | | | | |
|   var(Network Depression) | | | | | | | .004** | .002 |
|   var(Intercept) | .007*** | .001 | .007*** | .001 | .007*** | .001 | .05*** | .02 |
|   var(Residual) | .367*** | .011 | .367*** | .011 | .366*** | .011 | .360*** | .011 |

Notes: *p<.05, **p<.01, ***p<.001 (two-tailed)
School level N=112, Individual level N=9,580
[a] Within-school standardized

Table 2.3 Mixed Effects Regression Model: Examining Ego-centric and School-level Contextual Moderators of Depressive Contagion

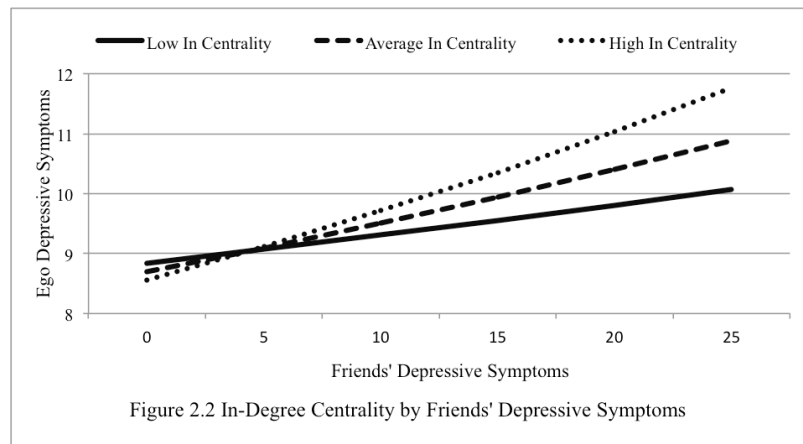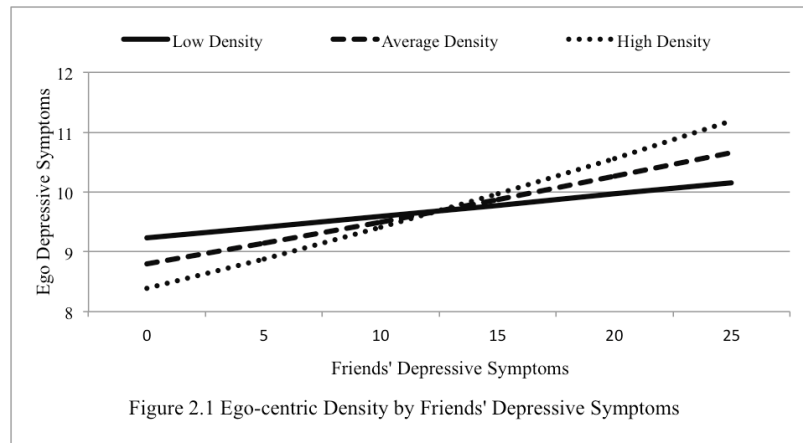| | Model 5 | Model 6 | Model 7 | Model 8 | Model 9 | Model 10 | Model 11 | Model 12 | Model 13 | Model 14 |
|---|---|---|---|---|---|---|---|---|---|---|
| **Main Effects** | | | | | | | | | | |
| Friends' Depression | .008* | .009** | .008* | .009** | .049** | .014* | -.003 | -.047* | -.033 | -.033 |
| | (.003) | (.003) | (.003) | (.003) | (.018) | (.007) | (.003) | (.019) | (.024) | (.024) |
| Microsocial Context: | | | | | | | | | | |
| Ego-centric Density | -.032 | -.003 | -.004 | -.053** | -.004 | -.004 | -.003 | -.003 | -.003 | -.053** |
| | (.019) | (.009) | (.009) | (.019) | (.009) | (.009) | (.009) | (.009) | (.009) | (.019) |
| In-degree Centrality | .021* | -.016 | .022* | -.032 | .022* | .022* | .022* | .022* | .022* | -.032 |
| | (.010) | (.023) | (.010) | (.025) | (.010) | (.010) | (.010) | (.010) | (.010) | (.025) |
| Out-degree Centrality | -.034*** | -.034*** | -.039* | -.042* | -.034*** | -.034*** | -.034*** | -.034*** | -.034*** | -.042* |
| | (.010) | (.010) | (.018) | (.020) | (.010) | (.010) | (.010) | (.010) | (.010) | (.020) |
| Macrosocial Context: | | | | | | | | | | |
| Mean Depression | -.006 | -.008 | -.007 | -.007 | .035 | -.007 | -.009 | -.008 | -.004 | -.004 |
| | (.009) | (.009) | (.009) | (.009) | (.020) | (.009) | (.010) | (.009) | (.021) | (.021) |
| Network Size | .000 | .000 | .000 | .000 | .000 | .015 | .001 | .001 | -.000 | -.008 |
| | (.003) | (.003) | (.002) | (.003) | (.003) | (.009) | (.003) | (.003) | (.000) | (.007) |
| Network Density | -.251 | -.289 | -.271 | -.274 | -.189 | -.195 | -5.26* | -.179 | -5.52* | -5.49* |
| | (.314) | (.324) | (.319) | (.319) | (.355) | (.312) | (2.17) | (.333) | (2.55) | (2.52) |
| Network Reciprocity | -.194 | -.209 | -.200 | -.200 | -.177 | -.198 | -.170 | -1.75** | -1.01* | -1.05* |
| | (.231) | (.232) | (.230) | (.234) | (.232) | (.232) | (.240) | (.651) | (.480) | (.480) |
| **Interaction with Friends' Depression** | | | | | | | | | | |
| Microsocial Context: | | | | | | | | | | |
| Ego-centric Density | .003* | | | .005*** | | | | | | .005*** |
| | (.001) | | | (.001) | | | | | | (.001) |
| In-degree Centrality | | .004* | | .005* | | | | | | .005* |
| | | (.002) | | (.002) | | | | | | (.002) |
| Out-degree Centrality | | | .000 | .001 | | | | | | .001 |
| | | | (.001) | (.002) | | | | | | (.002) |
| Macrosocial Context: | | | | | | | | | | |
| Mean Depression | | | | | -.004* | | | | -.000 | -.001 |
| | | | | | (.002) | | | | (.002) | (.002) |
| Network Size | | | | | | -.002* | | | .001 | .001 |
| | | | | | | (.001) | | | (.001) | (.001) |
| Network Density | | | | | | | .504** | | .530* | .528* |
| | | | | | | | (.191) | | (.225) | (.222) |
| Network Reciprocity | | | | | | | | .144** | .078* | .082* |
| | | | | | | | | (.055) | (.038) | (.038) |

Notes: *p<.05, **p<.01, ***p<.001 (two-tailed)

context variables. Several findings are of note in these models. First, friends' depressive symptoms remain a significant predictor of adolescent depressive symptoms even after adjusting for the main effects of microsocial and macrosocial variables. Second, none of the macrosocial variables are significant predictors of adolescent depressive symptoms. In fact, even after entering these school-level variables, there is no statistical reduction in the variance of the intercept (.007 in both Model 1 and 2). However, microsocial contextual variables of in-degree centrality and out-degree centrality are significant predictors of adolescent depressive symptoms.

In interpreting the coefficients in Table 2.2, recall that the dependent variable, adolescent depressive symptoms, in this analysis is log transformed. Therefore, according to Model 4, a 1 unit increase in friends' level of depressive symptoms results in a .8 percent increase in adolescent level of depressive symptoms after adjusting for sociodemographic controls, social relationship, and contextual variables. Alternatively, a one standard deviation increase in friends' level of depressive symptoms (SD=5.8), adolescent level of depressive symptoms increases by almost 5 percent. This finding suggests significant support for the presence of depressive contagion in adolescent peer networks.

Other notable significant effects include in-degree and out-degree centrality and sociodemographic variables. First, adolescents who sent more nominations are less depressed, but adolescents who received more nominations are more depressed. As received nominations is often used a measure of popularity in adolescent networks, it is interesting that popular individuals are more depressed than unpopular individuals. This finding can potentially be explained using Falci and McNeely's (2009) research suggesting that despite the common belief that social isolation causes emotional distress,

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having too many friends can also be harmful for adolescent mental health. Too many

friends can result in role strain and role conflict. Additionally, results in Model 4 suggest

large effect sizes for female, Asian, and Hispanic adolescents: roughly 8% increase, 16%

increase, and 11% increase in adolescent level of depressive symptoms, respectively.



Figure 2.1 Ego-centric Density by Friends' Depressive Symptoms



Figure 2.2 In-Degree Centrality by Friends' Depressive Symptoms

### 2.8.2 Contextual Moderators

The results in Table 2.3 concern the primary analysis in this study investigating

whether the effect of friends' level of depressive symptoms on adolescent level of

depressive symptoms varies dependent upon microsocial and macrosocial contexts. To

examine this, we run a series of interaction models shown in Table 2.3. Models 5-7 enter

microsocial density, in-degree centrality, and out-degree centrality one at a time to

examine whether each of these measures significantly moderates the effect of depressive

contagion. Model 8 examines the microsocial interactions simultaneously. Similarly,

Models 9-11 enter macrosocial factors of school mean level of depressive symptoms,

network size, density and mutuality, respectively while Model 12 examines the

macrosocial interactions simultaneously. Finally, Model 13 examines whether each of

these interaction effects remains significant when combined into a single model.

Findings in Table 2.3 suggest that of the three microsocial moderators tested, ego-

centric density and in-degree centrality are significant moderators of depressive

contagion. We illustrate these results in Figures 2.1 and 2.2 showing the effects of

friends' level of depressive symptoms on ego level of depressive symptoms at varying

levels of ego-centric density and in-degree centrality. These effects are shown while

holding all other covariates at their overall means. High levels of microsocial context are

operationalized as 1 standard deviation above the mean and low levels as 1 standard

deviations below the mean. Moreover, we set the x-axis in these to include only values

within approximately two standard deviations of the average level of depressive

symptoms among adolescents' friends (mean=11, SD=5.8). In other words,

approximately 95% of adolescents in the sample are represented within the figures.

As shown in Figure 2.1, the effect of network depressive symptoms is exacerbated

when embedded in dense, cohesive peer networks. Although the statistical results

indicate a moderation of microsocial density, Figure 2.1 shows that the difference in the

effect of depressive contagion between an adolescent embedded in a high density peer

group and a low density peer group is relatively modest. For instance, as friends' level of

depressive symptoms increases from 0 to 25, more than 4 standard deviations, adolescent

depression in low density networks increases by .93 compared to adolescent level of

depressive symptoms in high density networks which increases by approximately 2.8. In

relative terms, adolescents embedded in high density networks are three times more susceptible to depressive contagion. However, in response to a four standard deviation change in friends' depression, the response for high density networks only represents less than half a standard deviation in adolescent's own level of depressive symptoms.

In contrast to the modest moderating effects of microsocial density, the results in Model 6 show that in-degree centrality is a significant contextual moderator of depressive contagion. This finding is illustrated in Figure 2.2. According to the finding, students with high in-degree centrality are especially susceptible to depressive contagion. At low levels of friends' level of depressive symptoms, there is essentially no difference in levels of depressive symptoms between adolescents with low and high in-degree centrality. However, as friends' level of depressive symptoms increases, popular students experience higher levels of depressive symptoms while less popular students experience no real change. More specifically, the effect size of depressive contagion for popular adolescents is five times as large as that for unpopular adolescents.

The next analysis examines the variation of depressive contagion dependent upon macrosocial contextual moderators operationalized as school-level characteristics. The first step is to test whether the effect of network level of depressive symptoms varies according to school. We do this by entering the variable network level of depressive symptoms as a random effect in Model 4 on Table 2.2. To examine the significance of the random effect, we perform two statistical tests. First, a Wald statistic is generated by dividing the variance estimate by its associated standard error. As shown in Model 4, this statistic is significant at the .01 level. Second, we conduct a likelihood ratio (LR) test of whether there is a statistically difference between Model 4 with random coefficients and Model 4 without random coefficients. The LR test indicates that the random coefficients

model is statistically significant at the .001 level (LR chi2(2)=100.97, p<.000). The next

step is to explain this variation through a series of cross-level interactions between

school-level network characteristics and network level of depressive symptoms. Models

9-12 show the results to these interaction effects.



Figure 2.3 School Network Density by Friends' Depressive Symptoms



Figure 2.4 School Network Mutuality by Friends' Depressive Symptoms
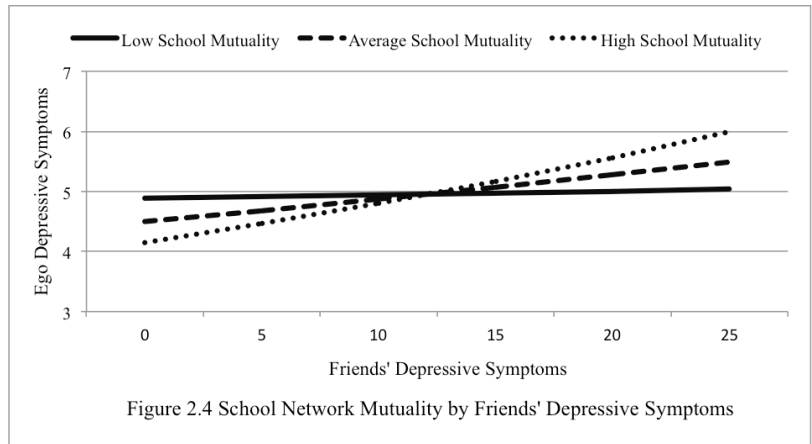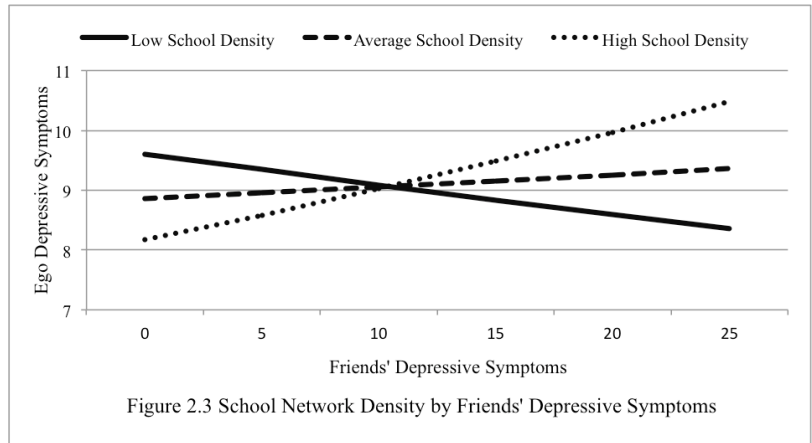
Models 9 and 10 examine the roles of schools' mean level of depressive

symptoms and network size as moderating effects of depressive contagion. Although

these models each reveal a statistically significant interaction term, both terms become

null after entering all macrosocial moderators simultaneously in Model 13. The two

macrosocial interaction coefficients that remain significant in Model 13 are network

density and network mutuality. These effects are illustrated in Figures 2.3 and 2.4. As

shown in Figure 2.3, friends' level of depressive symptoms increases adolescent

depression in high density schools. In average density schools, there does not appear to be any real effect of depressive contagion. However, most interestingly, friends' level of depressive symptoms in low density schools actually decreases adolescents' own level of depressive symptoms. This finding suggests important implications for future research as it demonstrates a significantly differing effect of depressive contagion dependent on a higher level contextual measure. For instance, the only other empirical study of contextual moderators of depressive contagion focuses only on peer group contexts. This finding in particular, indicates the importance of examining contextual moderators at multiple nested levels.

The final macrosocial moderator tested is network mutuality shown in Model 12. Results show that network mutuality exerts a significant moderating effect on depressive contagion. To better illustrate the effect, Figure 2.4 shows that depressive contagion is essentially non-existent in schools with relatively low mutuality indices (holding all other variables at their means). However, the effect of depressive contagion is significantly greater and positive in high mutuality schools. Implications of these results are discussed in the next section.

**2.9 Discussion**

The literature on the association between social relationships and health has examined relational mechanisms that lead to both positive and negative health outcomes. Researchers in support of the former cite greater social integration and social control from risky behaviors as resources provided by having social relationships (House et al. 1988; Umberson and Montez 2010). Through these relationships, individuals are provided opportunities to socially learn (Akers et al. 1979) healthy behaviors. However, in this same process where healthy phenomena travel through the pipelines of social

relationships, unhealthy phenomena can do the same. This hypothesized contagion effect where peer behaviors influence one's own behavior has been historically studied in the branches of deviance (Sutherland 1947; Akers et al. 1979) and can even theoretically be traced back to Durkheim's discussion of imitation in his famous work *Suicide* ([1897] 1951).

In this study, we examine contextual moderators of adolescent depressive contagion. We operationalize contextual moderators using two separate levels: the microsocial and macrosocial. The results of this analysis demonstrate support for the effect of depressive contagion in adolescent networks. This finding demonstrates the robustness of the adolescent depressive contagion thesis suggested in other empirical studies (e.g. Hogue and Steinberg 1995; Prinstein 2007; Conway et al. 2011; Cheadle and Goosby 2012). Although our study utilizes a longitudinal design to control for prior level of depressive symptoms and a multilevel design to control for environmental main effects, evidence of depressive contagion does not imply the absence of a selection effect. In fact, multiple studies using various methodologies have indicated that depression is a significant quality when selecting or deselecting friends (e.g. Zalk et al. 2010b; Schaefer et al. 2011; Cheadle and Goosby 2012). However, many of these studies also indicate evidence of depressive contagion above and beyond homophilous effects (Shalizi and Thomas 2011). Moreover, the primary objective of this study is not to engage in whether contagion or homophily represents greater effect sizes for the network autocorrelation of depression. Rather, our study aims at shedding light on the significance of context and furthermore, context at multiple nested levels.

At the microsocial level, our results show that adolescents are more susceptible to depressive contagion when embedded in dense peer networks. Dense networks are

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characterized by not only the direct ties between ego and the ego's friends but many indirect ties. These indirect ties can serve two primary purposes. First, the prevalence of indirect ties allow for more pathways through which depressive influence can travel. Second, indirect ties and consequently dense networks are more susceptible to the creation of small-group cultures where depressive behavior can be normalized (Conway et al. 2011). These norms, created through the interactions among ego and its alters, are subsequently internalized by the ego. It is no surprise then that this process is strengthened in conjunction with many social interactions occurring between not simply ego and alter but also alter and alters.

Furthermore, at the microsocial level, popularity plays an important role in moderating depressive contagion. Our findings suggest that popular students are more susceptible to the effects of depressive contagion. This finding may be indicative of the intrinsic personality traits of popular adolescents, or target-oriented factors (Prinstein 2007), rather than the network structure of individuals who receiving more friendship nominations. Nonetheless, popular students may be inherently more vulnerable to emotional and behavioral changes of their peer environment. Moreover, these same students may be more likely to engage in and be influenced by peer pressure and adolescent social norms. Prior research shows differing results for the moderating role of popularity in depression. Particularly, Conway et al. (2011) find that less popular students are more susceptible to depressive contagion, a directly opposite result from our study. Conway et al. (2011) suggest that popular students may experience less pressure for conformity. However, other contagion research suggests that popular students are more likely to conform potentially due to greater source exposures and status motivation (Aloise-Young et al. 1994; Urberg et al. 2003).

At the macrosocial level, our findings suggest a significant variation in the effect of depressive contagion contingent upon school characteristics. To explain this variation, we examined four school-level measures: mean level of depressive symptoms, network size, density, and mutuality. We find that after considering all four measures simultaneously, school network density and network mutuality significantly moderates the effect of depressive contagion. This finding is conceivable for two reasons. Since network density is measured as the number of observed social ties divided by the total number of potential ties, a high density network should serve as a particularly vulnerable social environment for depressive contagion. In addition, mutuality accounts for the directionality of these ties. Networks with greater tendencies toward mutuality in friendship nominations should also serve as vulnerable contexts for depressive contagion.

Overall, our findings indicate the importance in examining contextual moderators of depressive contagion. Although this was theorized in Prinstein's (2007) research almost a decade ago, empirical investigations of contextual moderators are limited (see Conway et al. 2011). Nonetheless, if we are to assume that behaviors or emotions are capable of spreading through social ties, then we must also understand that the structure of these social ties matter. In the same way that a telephone tree is considered an efficient way of diffusing information to a group of individuals, certain network structures are more susceptible to the social contagion of behaviors, emotions, etc. Certain network structures may be particularly vulnerable to depressive contagion.

**2.9.1 Implications**

Our findings present several implications for the literature on depressive contagion and more broadly on contagion research in general. First, our findings demonstrate the robustness of the depressive contagion thesis. Despite the increasing use

of actor-based models to simultaneously investigate homophily and contagion, future research must move further in order to examine factors that predict varying degrees in the effect of peer influence or selection. Second, our findings contribute to the contagion literature by providing evidence for the significance of macrosocial context. The results suggest that depressive contagion is moderated both at the peer network level and school level. Therefore, future research must view shared environments as a significant contextualizing factor for depressive contagion.

## 2.9.2 Limitations

Although the findings presented in this study provide significant contributions to the contagion literature, we acknowledge two limitations in our analyses. First, our study examines specifically peer contagion utilizing friendship nominations among adolescents. It is possible that influence comes from sources in addition to peers such as family, relatives, or etc. Second, the friendship nominations are limited to nominations to alters that attend the same school as the ego. As a result, the analysis does not capture the effects from alters that do not attend the same school. This boundary limitation is a common issue in social network research.

Despite these limitations, this study presents a valuable contribution to research on adolescent mental health and social contagion. Adolescence presents a unique stage in life where multiple biological, social, and psychological processes intersect. As individuals navigate through adolescence, sources of influence begin to shift from parental guidance and control to peer-group influences due the growth in peer network sizes and increasing time spent within the school. This shift coincides with the salience of social and psychological identity formation and biological outcomes of puberty. The

findings in this study suggest the importance in studying adolescent health in combination with social contextual factors.

## Chapter 3: Race and Ethnic Differences in Depressive Contagion:
## The Role of Racial Homophily

### 3.1 Introduction

Much of prior research on adolescent depression focuses on psychosocial and behavioral risk factors at the individual level and contextual risk factors at the parental level. However, more recent literature suggests the importance of peer groups at the social interactional level documenting a peer-influence of depressive symptoms, often referred to as the social contagion of depression. For instance, Rosenquist et al. (2011) use longitudinal data from the Framingham Heart Study and find that evidence for the contagion of depression within a social network of over 12,000 adults. Evidence for depressive contagion is also found in samples of the adolescent population (Prinstein 2007; Conway et al. 2011; Reynolds and Crea 2015; Guan and Kamo forthcoming).

Explanations for depressive contagion revolve around behavioral and cognitive mechanisms suggesting that individuals can be susceptible to peer-influence of emotions, behaviors, and ideas through emotional mimicry (Hatfield et al. 1994) and norm salience (Rosenquist et al. 2011). However, the simple identification of social contagion of adolescent depression is not enough to aid public health officials and intervention strategies in curbing depression levels within the adolescent population. Rather, researchers need to ask what factors can amplify an individual's resilience or vulnerability to depressive contagion. For instance, at the individual level, researchers can examine psychosocial measures such as coping mechanisms and social support frameworks in buffering the effects of depressive contagion.

Although prior research examines demographic variation in the effect of depressive contagion, only one study examines group differences based on racial/ethnic

identity. Reynolds and Crea (2015) find that minorities are more resilient to depressive contagion. However, racial/ethnic identity is not the primary measure of interest in this study. As a result, the authors use a dichotomous measure aggregating all non-white identities into a single "minority" category eliminating any potential for examining variation among minority groups. Furthermore, there is no empirical literature documenting the moderating effects of racial/ethnic composition of the peer network.

Based on these limitations of prior research, there are two purposes to this study. First, I examine potential variation in the effect of depressive contagion within minority groups. Second, I investigate the role of racial homophily in the depressive contagion process. This study aims to further existing research by establishing race as a significant moderator of depressive contagion.

## 3.2 Theory of Depressive Contagion

There are two primary perspectives used to explain the social contagion of depression: cognitive and behavioral. Rooted in the social psychological theories of Gabriel Tarde (1903), cognitive perspectives suggest a contagion of emotions, moods, and behaviors operating through two mechanisms. First, contagion can occur through the mimicry or imitation of the emotions of significant others (Hatfield et al. 1994). Second, it can also occur as a result of an individual's inclusion of close others within their self-evaluation. That is, an individual evaluates and internalizes the emotional state of his/her friends (Joiner and Katz 1999).

On the other hand, behavioral explanations focus on the interpersonal environment created by having depressed friends (Joiner and Katz 1999). Goffman's interpersonal reality (1959 [1974]) proves helpful in understanding this perspective. Norms of emotions, behaviors, and attitudes are collectively created by individuals,

consciously or subconsciously, which then has a subsequent feedback effect on the actors themselves. These norms become a potential source for contagion whereby actors imitate not the behavior of specific actors but instead enact the collectively agreed upon acceptable behaviors (norms). In the case of depressive contagion, the interpersonal environment within a depressed peer network can be characterized by having low levels of positive reinforcement and high levels of negative affect and sense of burdenedness (Coyne 1976a; 1976b; Joiner and Katz 1999). Furthermore, this social environment can become a source of stress for network members thereby perpetuating distress and depression (Coyne 1976a; Coyne 1976b).

## 3.3 Moderators of Depressive Contagion

A fruitful extension to understanding the process of depressive contagion is recognizing that the effect is not the same for all individuals. Certain characteristics of individuals and/or their environment may act as moderators that enhance resilience or vulnerability to depressive contagion. Prinstein (2007) first recognized the lack of research on moderating factors of depressive contagion. As a result, he identified four classes of moderators for future research that may increase individuals' resilience or vulnerability to depressive contagion: target, prototype, relationship, and contextual-oriented moderators.

Target moderators refer to the attributes of the individual being influenced while prototype moderators refer to the attributes of the individual applying influence (Prinstein 2007). These moderators can include basic demographic characteristics such as race, age, and gender or it can include psychosocial measures such as self-esteem and coping mechanisms. Relationship moderators are attributes of the relationship such as the

strength of relationship or type of relationship. Finally, contextual moderators refer to attributes of the context within which the peer network is embedded.

Subsequent research following Prinstein's (2007) call show a variety of factors that moderate the effect of depressive contagion. The most studied moderator regards the gender of the target and prototype. The results are mixed however. For instance, Conway et al.'s (2011) find that adolescent girls are more reactive to their friends' depression than adolescent boys. However, this finding is contradicted by other studies that find males to be more susceptible (Hogue and Steinberg 1995; Cheadle and Goosby 2012) and a meta-analytic review (Joiner and Katz 1999) predating Prinstein's call that suggests no significant difference between genders for either target or prototype.

Other research suggests that psychosocial measures such as reassurance seeking (Katz and Joiner 1999), failure anticipation (Zalk et al. 2010b), and social anxiety (Prinstein 2007) can be important moderators of depressive contagion. Moreover, studies also show that network embeddedness plays an important role in the social contagion of depression (Conway et al. 2011; Reynolds and Crea 2015). For instance, Guan and Kamo (forthcoming) find that popular students and students embedded in dense networks are significantly more susceptible to depressive contagion.

## 3.4 Race and Ethnic Group Differences

Despite this encouraging response to Prinstein's call, racial/ethnic group differences in depressive contagion have received little attention. For instance, in an otherwise thorough discussion of target-oriented moderators of peer contagion, Brechwald and Prinstein (2011) fail to mention race and ethnicity despite recognizing the importance of other demographic variables such as gender and age. This tendency is

reflected also in Joiner and Katz's (1999) meta-analytic review of more than 40 studies on depressive contagion.

This is a surprising oversight given the overwhelming literature on race and ethnic group differences in the rates of depression and mental health disorders (Vega and Rumbaut 1991; Williams et al. 1997). Reynolds and Crea (2015) is the only study that addresses race and ethnicity as an important moderator of depressive contagion. The authors find that adolescents self-reporting racial/ethnic minority status are more resilient to depressive contagion. Reynolds and Crea (2015) pose three explanations for their findings. They suggest that adolescents from the minority category may already be less embedded within social networks, have lower rates of depression, or actually be resilient to depressive contagion. Based on their results, I hypothesize that relative to white adolescents, depressive contagion is weaker for adolescents from the minority groups of black, Asian, and Hispanic.

$H_1$: Black, Asian, and Hispanic adolescents are more resilient to depressive contagion than white adolescents.

Unfortunately however, Reynolds and Crea's (2015) study is limited in that the authors aggregate racial/ethnic group identifications into a single "minority status" category. This method eliminates the ability to investigate group variations among minorities such as blacks, Asians, and Hispanics. Prior literature documents significant group differences within minorities in the rate of mental health (Vega and Rumbaut 1991; Williams et al. 1997), network embeddedness (Lin 2000), and the costs and benefits of having social ties (Umberson and Montez 2010). As a result, it is important to consider group variations in the social contagion of depression.

Moreover, much of this literature is based on assimilation theories suggesting that the minority experience of the Asian and Hispanic population significantly differs from that of the black population. This can be traced back to the former groups' recent and voluntary immigration status (Quillian and Campbell 2003) and significant differences in population size (Blau and Schwartz 1984). Therefore, it would be reasonable to expect that the salience of depressive contagion is significantly different between 1) black adolescents and Hispanic adolescents and 2) black adolescents and Asian adolescents.

$H_2$: The salience of depressive contagion is different between black adolescents and Asian adolescents

$H_3$: The salience of depressive contagion is different between black adolescents and Hispanic adolescents.

## 3.5 Racial Homophily and Depressive Contagion

In addition to investigating racial and ethnic group differences of depressive contagion, research can benefit by incorporating the concept of racial homophily. Racial homophily is essentially one type of friendship segregation, defined as the relationship between a characteristic of a group of people and their friendship choices (Moody 2001). The degree to which friendships are segregated is directly related to the degree of restriction in friendship choices based on this particular characteristic.

There are two primary reasons for investigating the role of racial homophily in the study of depressive contagion. First, homophily is always present in the formation of peer networks (McPherson et al. 2001; Moody 2001). Using the Add Health study, Moody (2001) finds that same-race friendships are 1.8 times more likely than cross-race friendships regardless of frequency and opportunity of interracial contact. This finding is supported by various other empirical studies (e.g. Hallinan and Williams 1989; Quillan

and Campbell 2003). Second, with regard to Prinstein's (2007) typology of contagion moderators, incorporating racial homophily shifts race from simply a target moderator to also a prototype moderator of depressive contagion. No empirical research to the best of my knowledge has studied this potential relationship.

Nevertheless, existing research on racial homophily suggests reasonable expectations that the racial composition of an individual's friendship network can express significant moderating effects on depressive contagion. Several studies show that same-race friendships are characterized by stronger emotional closeness (Hansell 1984; Schneider et al. 2007), greater number of shared activities (Kao and Joyner 2004), and greater stability of friendship overtime (Hansell 1984). In other words, same-race friendships are characterized by dense, strong, and stable network ties. These findings can be traced back to social psychological theories of friendship stability. For instance, both cognitive organization (Heider 1958) and balance (Newcomb 1961) theories suggest that relationships are more stable when characteristics of the individuals closely align with one another.

Therefore, for individuals embedded in racially homophilous peer networks, they are more likely to be 1) highly invested in the emotional welfare of their peers, 2) sensitive to the emotions, moods, and behaviors of peers and subsequent mimicry or imitation of these outcomes, and 3) embedded in peer networks with high levels of behavioral and emotional norm salience. As a result, I hypothesize that individuals embedded in racially homophilous peer networks are more susceptible to depressive contagion.

$H_4$: Depressive contagion is more salient for individuals with a high degree of racial homophily.

However, I also test the potential for this moderation effect of racial homophily to vary among racial and ethnic groups. For instance, racial homophily among black and white students is often understood through the historical framework of black-white relations including racial oppression, current socioeconomic inequality, systemic discrimination, and racial resentment (Quillian and Campbell 2003). However, for the growing minority populations of Asians and Hispanics, the implications of racial homophily are much less clear. Based on traditional assimilation theory (Warner and Srole 1945; Gordon 1964), one perspective suggests that Asians and Hispanics have a greater tendency toward assimilation given their voluntary immigration to America (Quillian and Campbell 2003). Furthermore, prior literature suggests that Asians aim to assimilate into the dominant culture and perceive racial homophily in friendship networks as a sign of failure (Lee 1994; Shih 1998; Ying et al. 2001).

$H_5$: The moderation of racial homophily on depressive contagion varies among racial/ethnic groups.

**3.6 Data**

Data used in this study are from the Wave 1 in-school questionnaire, Wave 1 in-home interview, and Wave 2 in-home interview of the National Longitudinal Study of Adolescent Health (Add Health). Adolescents included in the Add Health survey were selected using a stratified sampling process. First, a nationally representative sample of high schools was chosen based on region, urbanicity, school size, school type, and racial/ethnic composition. The Wave 1 in-school questionnaire was administered to every student within 132 schools (n=90,118). During this questionnaire, students were asked to nominate up to five female friends and five male friends. These nominations were used to construct egocentric network data used in this analysis.

Additionally, this study uses demographic, behavioral, and health data gathered from the Wave 1 in-home interview (n=20,745) and Wave 2 in-home interview (n=14,738). Data restrictions include participation in all three surveys and valid data on Wave 2 depression and sampling weights (n=13,499). Additionally, I eliminate 191 adolescents who reported a race or ethnicity other than white, black, Asian, or Hispanic due to the small sample size within these other groups (final n=13,308). Multiple imputation methods in Stata SE 13 (StataCorp 2013) are used to address missing data on variables other than Wave 2 depressive symptoms.

## 3.7 Measures

### 3.7.1 Dependent Variable

Adolescent depression in this study is measured using a 19-item symptomatic scale that corresponds to the Center for Epidemiologic Studies Depression (CES-D) scale (Radloff 1977). During the Wave 2 in-home interview, students were asked how often they experienced 19 different symptoms. Example symptoms include, "I was tired all the time," "My appetite was poor," and "I felt that people disliked me." Answers to these statements were coded as 0 for "rarely or none of the time," 1 for "sometimes," 2 for "a lot of the time," and 3 for "most or all of the time." The scores are then aggregated to produce a single depressive symptoms scale ranging from 0 to 56 with an alpha level of .877. As a result of respondents reporting significantly more low CES-D scores and few high CES-D scores, this measure is log transformed to reduce positive-skew and satisfy the normality assumption of OLS regression.

### 3.7.2 Independent Variables

The primary independent variables in this study are friends' depression, racial homophily, and adolescent racial/ethnic identity. First, racial/ethnic identity is coded

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according to five categories: white, black, Asian, Hispanic, and other. However, the Add Health survey allowed for adolescents to mark multiple responses. Therefore to achieve mutual exclusivity, I code adolescents as Hispanic if they marked Hispanic regardless of any additional racial/ethnic category. I follow this coding procedure for Asian, black, and white respectively.

The second set of independent variables utilizes the peer networks constructed from the friendship nominations during the Add Health in-school questionnaire. Recall that during this questionnaire, adolescents were asked to nominate up to five male and five female friends. Therefore, adolescent peer networks are created using both sent friendship nominations and received friendship nominations.

As a result, friends' depressive symptoms is measured using the average CES-D score among friends within an adolescent's peer network. Additionally, racial homophily is measured as the proportion of same-race friendships within an adolescent's peer network. In other words, if the adolescent identifies as Asian, racial homophily measures the proportion of Asian friends within his or her peer network.

### 3.7.3 Covariates

Regression models in the current study adjust for prior levels of depressive symptoms and six other covariates to control for potential selection effects. Full and sub-sample means of all covariates are shown in Table 3.1. Prior depressive symptoms is measured using adolescents' Wave 1 CES-D scores. Attachment to parents is constructed using an eight-item scale gauging the strength of students' relationships to each of their parents. Grade point average is calculated as the average score of Wave 1 self-reported grades for English, math, science, and history.

Parent education and parent income are measured using a parent interview during the Wave 1 survey. Parent education is coded according to the number of completed school years. For instance, high school graduates are coded as 12, some college as 14, and college graduate as 16. Parent income is measured as annual income in dollars and subsequently log-transformed to achieve normality.

## 3.8 Analytical Strategy

The analysis in this study utilizes lagged-effect OLS regression models predicting Wave 2 adolescent depressive symptoms while adjusting for Wave 1 adolescent depressive symptoms. Due to the positive skew of the Wave 2 CES-D scale, Wave 2 depressive symptoms is log-transformed to satisfy normality assumptions of regression analysis. Additionally, the models in this study use appropriate sampling weights to account for the complex sampling design of the Add Health survey.

First, I present a model estimating the effect of depressive contagion by including a measure of friends' depressive symptoms in predicting adolescent depressive symptoms. Second, I test the hypotheses of racial/ethnic variation in depressive contagion by entering interaction terms of racial/ethnic category × friends' depressive symptoms. After examining racial/ethnic group differences in depressive contagion, I investigate the moderating role of racial homophily by entering a racial homophily × friends' depressive symptoms interaction term. Finally, I utilize sub-sample analyses to estimate whether the moderating role of racial homophily varies according to the adolescent's racial/ethnic identity. To do this, I use pair-wise t-tests to compare the slope coefficients of the racial homophily × friends' depressive symptoms interaction term between each racial/ethnic group.

Table 3.1. Descriptive Statistics of All Variables in the Analysis: Full and Sub-sample

| Variables | Full Model N=13,308 | White N=7,214 | Black N=2,853 | Asian N=964 | Hispanic N=2,277 |
|---|---|---|---|---|---|
| Wave 2 Depression | 11.2* | 10.2[BAH] | 11.6[WAH] | 13.1[WB] | 12.6[WB] |
| | (7.5) | (7.4) | (7.5) | (7.5) | (7.5) |
| Wave 1 Depression | 11.3* | 10.4 [BAH] | 11.8 [WAH] | 13.0 [WB] | 12.6 [WB] |
| | (7.5) | (7.3) | (7.7) | (7.2) | (7.8) |
| Friends' Depression | 11.1* | 10.2 [BAH] | 11.4 [WAH] | 12.3 [WBH] | 13.1 [WBA] |
| | (5.7) | (5.4) | (5.6) | (5.6) | (6.3) |
| Racial Homophily | .66* | .73 [BAH] | .63 [WAH] | .54 [WB] | .51 [WB] |
| | (n/a) | (n/a) | (n/a) | (n/a) | (n/a) |
| Age | 15.3* | 15.2[AH] | 15.3[AH] | 15.7 [WB] | 15.6 [WB] |
| | (1.6) | (1.6) | (1.6) | (1.5) | (1.6) |
| Female | .51* | .51 | .54[A] | .48[B] | .50 |
| | (n/a) | (n/a) | (n/a) | (n/a) | (n/a) |
| Parent Income | 46.7* | 53.2 [BH] | 35.3 [WA] | 52.4 [BH] | 35.5 [WA] |
| | (53.5) | (58.8) | (34.1) | (47.6) | (51.3) |
| Parent Education | 13.2* | 13.6 [AH] | 13.5 [AH] | 14.1 [WBH] | 11.4 [WBA] |
| | (2.6) | (2.3) | (2.5) | (2.8) | (2.9) |
| Parent Attachment | 8.0* | 8.3 [BH] | 7.7 [W] | 7.9 | 7.5 [W] |
| | (5.1) | (5.1) | (4.8) | (5.3) | (5.1) |
| Grade Point Average | 2.8* | 2.9 [BAH] | 2.6 [WAH] | 3.0 [WBH] | 2.5 [WBA] |
| | (.77) | (.78) | (.71) | (.77) | (.76) |

*<.05
[W] p<.05 different from white
[B] p<.05 different from black
[A] p<.05 different from Asian
[H] p<.05 different from Hispanic

## 3.9 Results

Table 3.1 shows descriptive statistics of the variables included in this study. First, I use an ANOVA test to examine whether each variable varies according to race and ethnicity. The results show that there is variation for all variables in the study. Second, I conduct post-hoc analyses using a Scheff test to further analyze pair-wise differences within racial and ethnic categories.

According to the results, depressive symptoms measured during the Wave 1 and Wave 2 interviews are significantly different for all racial/ethnic pairs except between Asians and Hispanics. These two groups have the highest CES-D scores followed by

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blacks and whites, respectively. The pattern is similar in regards to friends' depression with only one difference. Hispanic adolescents have more depressed peer networks than Asian adolescents.

Additionally, peer networks of white and black adolescents are significantly more racially homophilous than that of Asian and Hispanic adolescents. The finding that racial homophily among black adolescents is different from both Hispanics and Asians follows expectations of assimilation theory. Hispanics and Asians have a higher tendency for cross-race friendships than blacks due to differences in their experiences of discrimination and other aspects of minority identity (Quillian and Campbell 2003).

Table 3.2 presents a lagged-effect regression model predicting adolescent depressive symptoms using a log transformation of the CES-D scale. In the first model, I test the main effects of friends' depressive symptoms and racial homophily on adolescent depressive symptoms while adjusting for prior Wave 1 depressive symptoms and basic socio-demographic measures. Results in Model 1 indicate a significant effect of friends' depressive symptoms on adolescent depressive symptoms and no effect from racial homophily. A one standard deviation increase in friends' depressive symptoms (5.7) results in an approximately 1.7% increase in adolescent depressive symptoms.

Additionally, being black, Asian, or Hispanic relative to being white has a significantly positive effect on adolescent depressive symptoms (5.7%, 15.6%, and 11.3% increase respectively). Female adolescents are also likely to have greater numbers of depressive symptoms than male adolescents. Other findings in Model 1 suggest a negative effect of parent education, attachment to parents, and grade point average on adolescent depressive symptoms.

Table 3.2 OLS Regression Predicting Wave 2 Depressive Symptoms

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|---|---|---|---|---|---|---|---|---|
| | β | SE | β | SE | β | SE | β | SE |
| Intercept | 2.015*** | .091 | 2.008*** | .092 | 2.095*** | .094 | 2.144*** | .095 |
| Main Effects: | | | | | | | | |
| Friends' Depressive Symptoms | .003** | .001 | .003* | .002 | .001 | .003 | -.002 | .003 |
| Racial Homophily | -.005 | .020 | -.004 | .021 | -.004 | .021 | -.121** | .043 |
| White | - Ref - | | - Ref - | | -.087* | .038 | - Ref - | |
| Black | .057** | .018 | .087* | .038 | - Ref - | | .056** | .018 |
| Asian | .156*** | .031 | .173** | .059 | .086 | .065 | .151*** | .031 |
| Hispanic | .113*** | .022 | .132* | .053 | .045 | .059 | .111*** | .022 |
| Interactions with Friends' Depressive Symptoms: | | | | | | | | |
| Racial Homophily | -- | -- | -- | -- | -- | -- | .010*** | .003 |
| White | -- | -- | - Ref - | | .003 | .003 | -- | -- |
| Black | -- | -- | -.003 | .003 | - Ref - | | -- | -- |
| Asian | -- | -- | -.002 | .005 | .001 | .005 | -- | -- |
| Hispanic | -- | -- | -.002 | .004 | .001 | .004 | -- | -- |
| Controls: | | | | | | | | |
| Wave 1 Depression | .047*** | .001 | .047*** | .001 | .047*** | .001 | .047*** | .001 |
| Age | .006 | .005 | .006 | .005 | .006 | .005 | .006 | .005 |
| Female | .080*** | .015 | .080*** | .015 | .080*** | .015 | .080*** | .015 |
| Parent Income (logged) | -.013 | .009 | -.013 | .009 | -.013 | .009 | -.013 | .009 |
| Parent Education | -.010*** | .003 | -.010*** | .003 | -.010*** | .003 | -.010*** | .003 |
| Attachment to Parents | -.014*** | .001 | -.014*** | .001 | -.014*** | .001 | -.014*** | .001 |
| Grade Point Average | -.066*** | .010 | -.066*** | .010 | -.066*** | .010 | -.066*** | .010 |

Notes: *p<.05, **p<.01, ***p<.001 (two-tailed)
N=13,308

Table 3.3 Racial/Ethnic Group Differences: Sub-sample Analysis of the Moderating Effect of Racial Homophily on Depressive Contagion (Model 4)

| | White | | Black | | Asian | | Hispanic | |
|---|---|---|---|---|---|---|---|---|
| | β | SE | β | SE | β | SE | β | SE |
| Intercept | 2.031* | .128 | 2.136* | .179 | 2.029* | .353 | 2.367* | .206 |
| Friends' Depression | .000 | .004 | -.002 | .005 | -.011 | .009 | -.002 | .005 |
| Racial Homophily | -.045 | .060 | -.067 | .086 | -.065 | .149 | -.191* | .096 |
| Interaction Effect | .007 | .005 | .001[AH] | .006 | .028*[B] | .011 | .020*[B] | .007 |
| Sample | N=7,214 | | N=2,853 | | N=964 | | N=2,277 | |

Notes: *p<.05
[W] p<.05 different from white
[B] p<.05 different from black
[A] p<.05 different from Asian
[H] p<.05 different from Hispanic

### 3.9.1 Racial/Ethnic Differences of Depressive Contagion

Model 2 investigates the hypothesis that the minority statuses of black, Asian, and Hispanic exacerbate an adolescent's susceptibility to depressive contagion ($H_1$). To examine this effect, I include three two-way interaction terms between friends' depression and black, Asian, and Hispanic. These interaction terms essentially test whether the effect of depressive contagion is different between white adolescents and each minority group. The results show no statistical significance in any of the interaction terms. This finding provides evidence that being black, Asian, or Hispanic relative to being white does not make an adolescent more vulnerable to depressive contagion.

However, I also hypothesize a within-minority variation in depressive contagion. This hypothesis is based on prior research demonstrating significant differences in social network structure and experience among racial/ethnic minorities (Vega and Rumbaut 1991; Williams et al. 1997; Umberson and Montez 2010). Therefore, I predict that the effect of depressive contagion is different between black and Asian adolescents ($H_2$) and between black and Hispanic adolescents ($H_3$). To test both hypotheses, I include

interaction terms between friends' depression and racial/ethnic categories while setting black as the reference category. The results in Model 3 once again show no statistical significance in the interaction between race/ethnicity and friends' depression.

Taken together, the results in Models 2 and 3 demonstrate that adolescent depressive contagion does not vary according to racial/ethnic identification. This finding directly contradicts the results in Reynolds and Crea's (2015) study suggesting that racial/ethnic minorities are more susceptible to depressive contagion. This contradiction can be a consequence of their study's method of aggregating all racial/ethnic groups into one single minority category. Therefore, the average effect of depressive contagion among minorities may actually be significantly different than the white reference category.
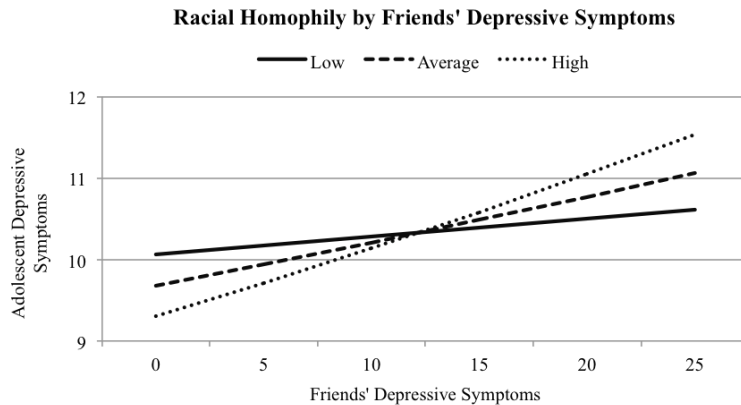
**Racial Homophily by Friends' Depressive Symptoms**



Figure 3.1 Results from Model 4 of Table 2 indicate that adolescents embedded in racially homophilous peer networks are more susceptible to depressive contagion. The top figure shows the effect of friends' depressive symptoms (x-axis) on adolescent depressive symptoms (y-axis). This effect is partitioned into three separate lines indicating low (-1 SD), average (mean), and high (+1 SD) racial homophily.

### 3.9.2 The Role of Racial Homophily

The final model in Table 3.2 examines the role of racial homophily in the study of depressive contagion. Based on prior research indicating stronger social ties in same-race friendships, I hypothesized that depressive contagion is more salient in racially

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homophilous peer networks ($H_4$). The results in Model 4 show a statistically significant interaction between friends' depressive symptoms and racial homophily. To better explain this finding, I graph the results shown in Figure 3.1.

Figure 3.1 illustrates the effect of friends' depressive symptoms on adolescent depressive symptoms. This effect is split into three lines: high racial homophily (+1SD), average racial homophily (mean), and low racial homophily (-1SD). As shown in Figure 3.1, depressive contagion is exacerbated when embedded in racially homophilous peer networks. Moreover, the effect of friends' depression on adolescent depression is statistically insignificant in peer networks characterized by low racial homophily.

Although racial homophily is shown to significantly moderate depressive contagion, this effect may only exist for certain racial/ethnic groups given the different implications of racial homophily for different racial/ethnic groups ($H_5$). Therefore, to test this hypothesis, I utilize a sub-sample analysis shown in Table 3.3 where the final regression model (Model 4) is run for each of the racial/ethnic groups. The results in Table 3.3 show a statistically significant interaction effect between friends' depression and racial homophily within the Asian and Hispanic sub-samples. However, this does not necessarily indicate a difference in the moderating effect of racial homophily on depressive contagion. Therefore, I utilize pair-wise t-test comparisons to analyze whether the coefficient estimates within each sub-sample model significantly differ from each other. The results show a statistically significant difference between the interaction coefficient of the black sub-sample and the Asian and Hispanic sub-samples. Moreover, Figure 3.2 shows the moderating effect of racial homophily within each sub-sample. The potential implications of these findings are discussed in the next section.
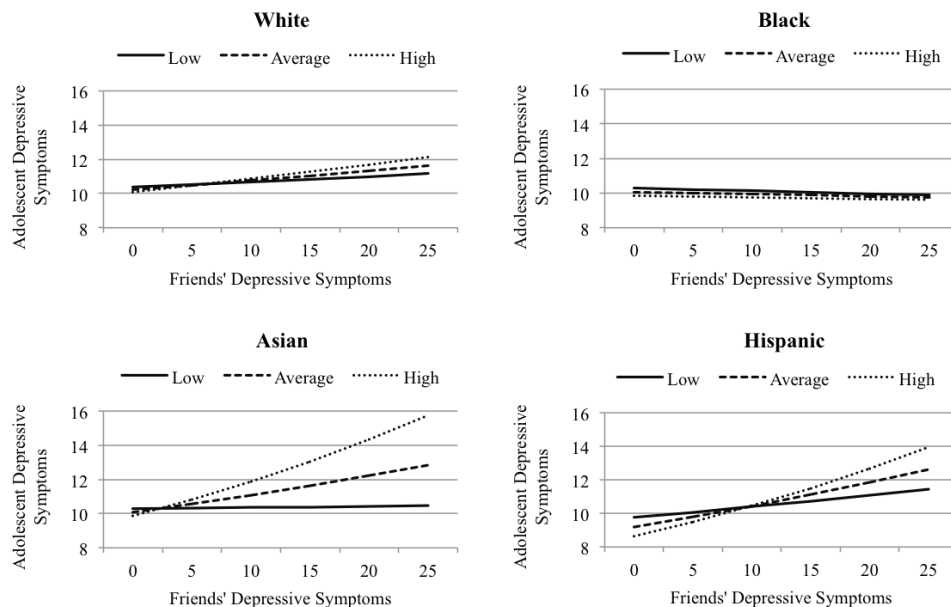
Figure 3.2 Sub-sample analysis results show that the moderating effect of racial homophily on depressive contagion varies dependent on adolescent racial/ethnic identity. The separate lines in the figure represent low, average, and high degrees of racial homophily operationalized as -1 SD, mean, and +1 SD. The interaction coefficients between racial homophily and friends' depression for Asian and Hispanic are found to be significantly different from the interaction coefficients for White and Black.

## 3.10 Discussion

The purpose of this study was to examine the role of racial and ethnic identities in the social contagion of adolescent depression. Based on prior research (Reynolds and Crea 2015) that finds a difference in the effect of depressive contagion between white and minority adolescents, I hypothesized that depressive contagion is different between white adolescents and black, Asian, and Hispanic adolescents ($H_1$). However, I found no evidence to support this hypothesis. Therefore, the results in this study appear to contradict the findings in Reynold and Crea's (2015) study, despite both using the Add Health study.

There are several potential explanations for the difference in the findings above and beyond coding differences. First, Reynolds and Crea (2015) utilize only 15 symptoms of the 19 symptom CES-D scale. Second, there are methodological differences

in that the authors choose to use a random effects model (survey waves nested within respondents) while the current study utilizes a longitudinal lagged-effect model. Finally, the current study adjusts for sampling weights using the *svy* command in Stata SE (StataCorp 2013). As a result, further research using other data samples may shed light on whether minorities are indeed more resilient to depressive contagion.

In addition to differences between whites and minority racial/ethnic groups, I hypothesized a within-minority variation suggesting that depressive contagion be different between black adolescents and Asian and Hispanic adolescents ($H_2$, $H_3$). However, the results show no significant difference within minorities. Moreover, further analysis (not shown) suggests no significant difference among all racial/ethnic groups included in this study.

The second focus of this study investigates racial/ethnic identity as a prototype moderator as opposed to a target moderator. Much of prior research investigating demographic differences in contagion fail to incorporate demographics of the peer network. As a result, I hypothesized that the racial composition, more specifically racial homophily, increases an adolescent's susceptibility to depressive contagion ($H_4$). This hypothesis is based on prior research that suggests that racial homophily plays an important role in friendship formation (Moody 2001). Moreover, same-race friendships are on average stronger and more stable (Hansell 1984; Kao and Joyner 2004; Schneider et al. 2007) leading to greater likelihood of emotional and behavioral influence within the peer network.

The results in this study suggest an exacerbation effect of racial homophily on depressive contagion. Adolescents embedded in peer networks with high racial homophily are more vulnerable to depressive contagion. However, prior research

suggests that the meaning of racial homophily may differ among racial/ethnic groups. For instance, among the black population, racial homophily is viewed as racial solidarity (Quillian and Campbell 2003) while Asians perceive racial homophily as a failure to assimilate into the dominant culture. Therefore, I hypothesized that the importance of racial homophily may differ among the racial/ethnic groups included in this study. After employing a sub-sample analysis of the interaction effect between friends' depression and racial homophily, I find that the exacerbation effect of racial homophily on depressive contagion exists predominantly for Asian and Hispanic adolescents.

The findings in this study have significant theoretical implications. Although the results show no racial/ethnic group differences in depressive contagion, high levels of racial homophily exacerbate the effects of depressive contagion. Moreover, I find that this relationship affects specifically Asian and Hispanic adolescents. This finding is surprising because I hypothesized that this exacerbation effect would affect specifically black adolescents. This hypothesis was based on prior literature suggesting that 1) racial homophily is important for the black population as it represents racial solidarity (Quillian and Campbell 2003) and 2) contagion is more salient within strong and dense social networks (Guan and Kamo forthcoming).

The results in this study however, indicate the direct opposite. Although, the original hypothesis was correct in predicting a racial/ethnic variation in the moderating effect of racial homophily, it was wrong in suggesting that racial homophily conditions depressive contagion for black adolescents but not Asian and Hispanic adolescents. Therefore, rather than focusing on racial solidarity and subsequent strengthening of social ties, the question is why is depressive contagion exacerbated for Asians and Hispanics embedded in racially homophilous peer networks. One possible explanation is that

although Asians and Hispanics value assimilation into the majority culture, intraracial social relationships remain stronger than interracial social relationships given their relatively recent immigration. Future research incorporating generation differences could provide answers to this question.

**3.11 Conclusion**

In regards to depressive contagion, only one prior study (Reynolds and Crea 2015) investigates the moderating role of racial/ethnic identity. Moreover, no research has examined race and ethnicity as a prototype moderator. As a result, this study presents significant contributions to the literature on depressive contagion and more broader social contagion research. The results of this study indicate that an adolescent's racial/ethnic identity may not be important as a target-oriented moderator of depressive contagion, alone. Rather, the importance is in the relationship between the target's race/ethnicity and the prototypes' race/ethnicity, otherwise conceptualized as racial homophily in this study.

In addition, the findings in this study present valuable information for public health initiatives on curbing adolescent depression. Depression represents the number one cause of illness among adolescents worldwide (World Health Organization 2014). Although recent research has brought social contagion in focus as a risk factor of depression, it has also led to the potential for drawing the unfortunate and misled conclusion that adolescents should avoid friendships with depressed individuals. As a result, I suggest that the growing research on moderators of social contagion can significantly curb this tendency.

Rather than simply identifying the existence of depressive contagion, research on moderators can elucidate the factors that may exacerbate vulnerability or resilience. In other words, research on moderators are trying to understand why certain individuals are

particularly receptive to depressive contagion. Finding the answers to this question

empowers intervention strategists with the appropriate information to allocate resources

to individuals or social contexts that are more susceptible to depressive contagion. For

example, a prior study on substance use prevention strategies indicate that network-

driven intervention strategies may be more effective than traditional intervention

treatments (Valente et al. 2007). As a result, the findings in this study present major

contributions in aiding public health researchers in designing network-driven intervention

strategies with racial and ethnic identities in mind.

## 3.12 Limitations

There are two pervasive challenges in the research on social contagion. First,

empirical studies investigating social contagion must always attend to teasing out the

effects of social contagion from the effects of homophily and shared environments

(Christakis and Fowler 2013). Second, social contagion research relies on the availability

of social network data that includes longitudinal measures. The limitations of the current

study are byproducts of these two challenges. Although I control for a lagged effect of

prior depressive symptoms at the time of the measured friendship network composition,

there remains potential effects of homophily and shared environments. However, based

on prior research utilizing SIENA models that simultaneously model homophily and

contagion effects (Cheadle and Goosby 2012), depressive contagion effects remain

significant despite "adequately" adjusting for homophily effects.

Additionally, the current study is limited to the use of the Wave 1 and Wave 2

interviews of the Add Health survey which were conducted two decades ago. Therefore,

this study is limited in that the results of this research may not adequately reflect the

current population of adolescents. Unfortunately, the Add Health survey is the only

nationally representative survey that includes information on adolescent social network

compositions and longitudinal measures of the health and illness of adolescents. Despite

these limitations, the current study makes significant contributions to social contagion

research and demonstrates novel and important empirical avenues for future research.

## Chapter 4: The Social Contagion of Adolescent Depression: Moderation by the 5-HTTLPR Polymorphism in the SLC6A4 Gene

### 4.1 Introduction

The gene-environment interaction (GxE) paradigm is inherently an interdisciplinary approach between biological and social science research. The analytical approach of GxE regards examining variation in health outcomes based on the interplay between individual genotype and environmental exposures. It is no surprise then that this perspective has heavily adopted the social stress model as the predominant method of measuring environmental exposure, the "E" component of the GxE paradigm.

Much of this research however, suffers from a shallow interpretation of the social stress perspective. More specifically, empirical studies on GxE prefer the operationalization of environmental exposure as the "number of stressful life events" for the sake of empirical replication (Uddin et al. 2010). Although replication is an important component of scientific research, this preference disregards several aspects of social stress theory including the differentiation of acute and chronic stress and the role of social relationships (Pearlin et al. 1981; Pearlin 1989). The present study focuses on the latter regarding potential negative outcomes of social relationships with mentally distressed individuals.

This phenomenon, referred to as "social contagion," suggests that depression can spread through social networks by way of social ties (Christakis and Fowler 2013). In terms of depressive contagion, individuals can become depressed as a result of being embedded within peer networks of depressed friends. In addition, subsequent research on social contagion has applied a differential susceptibility model to depressive contagion. That is, certain individuals can be particularly vulnerable to depressive contagion while

others are more resilient (Prinstein 2007). Although research has investigated factors such as gender (Hogue and Steinberg 1995; Conway et al. 2011; Cheadle and Goosby 2012), popularity (Conway et al. 2011; Guan and Kamo forthcoming), social anxiety (Prinstein 2007), and network structure (Reynolds and Crea 2015; Guan and Kamo forthcoming), no existing study has examined genotypic variation in depressive contagion. In other words, are certain individuals more susceptible to depressive contagion due to their genetic composition?

To answer this question, the present study utilizes a measure of a functional polymorphism (5-HTTLPR) in the serotonin transporter gene (SLC6A4) that has been shown in prior studies (e.g. Caspi et al. 2003; Eley et al. 2004; Reinelt et al. 2015) to moderate the effect of environmental stress on depression. The evidence suggests that individuals carrying one or two copies of the S allele are more susceptible to the effect of environmental stressors on individual depression. In the following sections, I review literature on the social contagion of depression and GxE research on 5-HTTLPR. Additionally, I explain the theoretical and practical significance of investigating genotypic variation of depressive contagion.

## 4.2 Social Contagion of Depression

Recent research on depression shows evidence of a contagion effect suggesting that individuals become more depressed when surrounded by depressed peers (Hogue and Steinberg 1995; Stevens and Prinstein 2005; Prinstein 2007; Zalk et al. 2010a; 2010b; Conway et al. 2011; Cheadle and Goosby 2012). The social contagion process is often explained using two theoretical perspectives: cognitive and behavioral. Cognitive perspectives (Hatfield et al. 1994) suggest that contagion occurs through the inclusion of the emotional and behavioral state among significant others within an individual's self-

concept. This leads to the individual evaluating this emotional state as his/her own and therefore, subsequently internalizing emotions and behaviors of significant others(Joiner and Katz 1999). On the other hand, behavioral perspectives suggest that peer groups consciously and subconsciously develop interpersonal realities (Goffman 1959 [1974]) that subsequently enforce norms of behaviors, emotions, and attitudes. These norms are created and enforced by the collective consciousness of the individuals embedded within the peer network.

An extension of both cognitive and behavioral perspectives on social contagion is the understanding that the effect of social contagion is not uniform among all individuals. Prinstein (2007) explains that certain characteristics of individuals or the environment can exacerbate resilience or enhance vulnerability to depressive contagion. For instance, prior research shows that the psychosocial measures of reassurance seeking (Katz and Joiner 1999) and social anxiety (Prinstein 2007) can significantly moderate depressive contagion. Additionally, Guan and Kamo (forthcoming) find evidence suggesting that popular students are more susceptible to depressive contagion.

As a result, the research on moderators of contagion present significant implications for practical application. Rather than framing contagion as simply the spread of behaviors or emotions through social networks, research on moderators enable intervention strategists to focus in on populations and environments that are shown to be significantly more vulnerable to depressive contagion. Unfortunately however, no research has incorporated genetic composition as a factor in determining differential susceptibility to depressive contagion. This limitation is surprising given prior research documenting the moderating effect of genetic variation on the relationship between stress and depression (Caspi et al. 2003).

**4.3 Gene-Environment Interactions of the 5-HTTLPR Polymorphism on Depression**

In an empirical study that has since been cited over 6,000 times, Caspi et al. (2003) apply a differential susceptibility model to depression based on the interaction between a functional polymorphism (5-HTTLPR) in the serotonin transporter system (SLC6A4) and environmental exposure. Their findings suggest that individuals carrying one or two S alleles are more susceptible to the effects of stressful life events on individual depression. The empirical design of Caspi et al.'s (2003) study has since been replicated by dozens of other studies. Two meta-analytical analyses (Risch et al. 2009; Munafo et al. 2009) find no evidence for a GxE effect between 5-HTTLPR and stressful life events. However, a more recent meta-analytical review by Karg et al. (2011) suggests that the studies of Risch et al. (2009) and Munafo et al. (2009) are not entirely exhaustive of all studies. As such, the literature suggests that the moderating role of 5-HTTLPR remains unclear.

A fruitful direction for future research may be evident in recent studies applying alternative operationalizations of environmental exposures. Stressful life events are but one type of environmental stressor that can interact with genotypic variations in predicting depression. For instance, although most studies cite Caspi et al.'s (2003) findings for stressful life events, their results also show a significant GxE effect for 5-HTTLPR on childhood maltreatment and depression. Other measures of environmental exposure include county-level economic deprivation (Uddin et al. 2010), friends and family suicidal behavior (Watts 2015), and maternal parenting (Zhang et al. 2015). As a result, examining genotypic moderation of depressive contagion can present meaningful contributions to the broader GxE research paradigm.

**4.4 Present Study**

The present study examines the functional polymorphism (5-HTTLPR) in the serotonin transporter gene as a moderator of the social contagion of adolescent depression. Although prior research shows no evidence of a direct effect of 5-HTTLPR on depression, several studies document a moderating effect on the relationship between environmental stressors and depression. Therefore, I apply a differential susceptibility model in hypothesizing that individuals carrying one or two copies of the S allele are more vulnerable to the social contagion of depression. The following analysis tests this hypothesis using a longitudinal and nationally representative sample of adolescents.

$H_1$: Adolescents with one or two S alleles in the 5-HTTLPR are more susceptible to depressive contagion than adolescents with two L alleles.

This study presents several contributions to the existing literature. First, this study merges the GxE paradigm with the vast literature on the health effects of social relationships. Second, this study utilizes depressive contagion as an alternative operationalization of the "E" component of the GxE hypothesis. This avoids the tendency of GxE research to focus predominantly on stressful life events as the proxy for environmental exposures. Finally, this paper demonstrates the potential importance of genetic composition within the broader literature on moderators of social contagion.

**4.5 Data**

Data used in this paper are from the National Longitudinal Study of Adolescent Health (Add Health). The Add Health study follows a cohort of individuals from adolescence to adulthood. Currently, four waves of interviews have been conducted. Respondents were chosen based on a stratified sampling strategy. First, approximately 140 nationally representative schools were chosen based on each school's region,

urbanicity, size, public/private status, and ethnic composition. During the first wave of data collection, an initial in-school questionnaire was conducted for every student attending each of these schools. Subsequently, a sample of students from each school was probabilistically chosen for the following four waves of in-home interviews.

This analysis utilizes a measure of depressive symptoms collected during the first two waves of the Add Health study. Demographic covariates are from the Wave 1 in-school questionnaire. Additionally, two unique components of the Add Health study are utilized for this study. First, I use network data gathered through friendship nominations during the Wave 1 in-school questionnaire. Each student within the 140 nationally representative schools was asked to nominate up to five male friends and five female friends. Since the in-school questionnaire was an exhaustive survey, researchers are able to construct peer and school social networks based on friendship ties. This study utilizes a measure of the level of depressive symptoms within adolescents' peer networks. Second, this paper uses genetic data gathered during the Wave IV in-home interview. During the Wave IV interview, 14,560 respondents agreed to provide DNA samples via Oragene or other buccal cell DNA collection methods. For further details on the Add Health design, see Harris et al. (2009).

I restrict the sample used in this analysis based on valid data on Wave 1 and Wave 2 depression measures, friendship network, and genetic information (n=6,397). Additionally, I utilize Wave 2 sampling weights to account for the complex sampling design of the Add Health survey. This further eliminates 146 cases. As a result, the final sample size is 6,251.

**4.6 Measures**

**4.6.1 Depression**

Depression is operationalized in this analysis as the level of depressive symptoms using a 19-item scale collected during the Wave 1 and Wave 2 in-home interviews of the Add Health study. This scale roughly corresponds to the Center for the Epidemiological Study of Depression (CES-D) scale used in other health surveys (Radloff 1977). The CES-D scale consists of 20 statements such as, "I felt depressed," "I felt lonely," and "I could not get going." Seventeen of these statements are included in the Add Health survey. For further details on similarities and differences in the two scales, see Pereira et al. (2005).

Adolescents were asked how often they experienced each of the 19 depressive symptoms: never or rarely, sometimes, a lot of the time, or most of the time. Responses are coded from 0-3, respectively. Subsequently, I combine these responses into a single scale measuring adolescents' level of depressive symptoms (range 0-56, alpha=.859). Three measures in this analysis utilize this depressive symptoms scale. First, the primary dependent variable in this analysis uses adolescent depressive symptoms collected during the Wave 2 in-home interview. Second, in order to adjust for potential selection or spurious effects, I include a lagged dependent variable, Wave 1 depressive symptoms, in the regression model. Finally, to measure depressive contagion, I use the average of each adolescents' friends' depressive symptoms.

**4.6.2 5-HTTLPR**

In the following analysis, I follow a two-step coding procedure advised by the Add Health study on the measurement of the functional polymorphism 5-HTTLPR in the serotonin transporter gene. First, I follow prior research suggesting that the less common

$L_G$ allele (as opposed to the more common $L_A$ allele) is no more efficient in transcription than the S allele (Hu et al. 2006). Therefore, $L_G$ and S alleles are coded as S' and the $L_A$ allele is coded as L'. Second, adolescents are categorized into three groups in order to conduct a triallelic analysis of 5-HTTLPR: S'/S' if they carry two copies of the S allele, S'/L' if they carry one copy of the S allele, and L'/L' if they two copies of the l allele. For more information on the Add Health genotyping methodology, see Smolen et al. (2012).

### 4.6.3 Covariates

Adolescent depression is shown to be strongly correlated with various demographics (Avison and McAlpine 1992; Williams et al. 1997). Therefore, I control for measures of gender, race, and age in all of the following regression models. Race is measured using a self-reported question asking respondents to select all racial/ethnic categories with which they identify. Due to sample size limitations within each category, I utilize the four largest racial/ethnic categories: white, black, Asian, and Hispanic. All other choices are coded as "other race." Additionally, in order to achieve mutual exclusivity in these five categories, I give priority to Hispanics, Asian, black, white, and other race, respectively.

### 4.7 Analytical Strategy

The following analysis includes a preliminary evaluation of the hypothesized gene-interaction effect between friends' depressive symptoms and the 5-HTTLPR polymorphism on adolescent CES-D score. I do this using a cross-tabulation analysis of 5-HTTLPR genotypes and a quartile measure of friends' depressive symptoms. The results of this analysis are shown in Table 4.2 and discussed in the next section.

Table 4.1 Descriptive Statistics of All Variables in the Analysis

| Variables | Mean (Proportion) | S.D. | Minimum | Maximum |
|---|---|---|---|---|
| Wave 2 Depression | 11.3 | 7.4 | 0 | 50 |
| Wave 1 Depression | 11.2 | 7.5 | 0 | 54 |
| Friends' Depression | 11.1 | 5.7 | 0 | 46 |
| 5HTTLPR—S'/S' | .31 | n/a | 0 | 1 |
| 5HTTLPR—S'/L' | .43 | n/a | 0 | 1 |
| 5HTTLPR—L'/L'* | .26 | n/a | 0 | 1 |
| Male* | .45 | n/a | 0 | 1 |
| Female | .55 | n/a | 0 | 1 |
| Age | 15.3 | 1.6 | 12 | 20 |
| White* | .48 | n/a | 0 | 1 |
| Black | .15 | n/a | 0 | 1 |
| Asian | .07 | n/a | 0 | 1 |
| Hispanic | .15 | n/a | 0 | 1 |
| Other Race | .16 | n/a | 0 | 1 |

*Reference category in the analysis
N=6,251

The second and primary investigation of this study utilizes a negative binomial regression model predicting adolescent depressive symptoms. Negative binomial models are often used for count data with overdispersion (Long and Freese 2006) and therefore appropriate for the dependent variable in this analysis (mean=11.3, variance=55.3). Therefore, the negative binomial coefficients in the models represent the change in the log of adolescent depressive symptoms in response to a one unit change in the predictor variable.

The results of the negative binomial regression models are shown in Table 4.3. Significance tests of all regression coefficients are based on two-tailed tests. Model 1 shows regression results for the main effects of friends' depressive symptoms and 5-HTTLPR genotype while adjusting for demographic covariates. Model 2 builds on the first model by entering the lagged dependent variable, Wave 1 depressive symptoms.

Table 4.2 Mean Wave 2 Depressive Symptoms by Friends' Depressive Symptoms and
5-HTTLPR

| 5-HTTLPR | Friends' Depressive Symptoms | | | | |
|---|---|---|---|---|---|
| | Quartile 1 (0-7) | Quartile 2 (7-10) | Quartile 3 (10-14) | Quartile 4 (14-46) | Q4 – Q1 |
| S'/S' | 9.3 (463) | 10.4 (481) | 12.2 (507) | 13.7 (496) | 4.4*** |
| S'/L' | 8.9 (701) | 9.8 (646) | 11.3 (682) | 12.8 (630) | 3.9*** |
| L'/L' | 9.1 (402) | 9.7 (430) | 10.4 (430) | 10.9 (383) | 1.8 |

Notes: Numbers in parenthesis within the quartile cells indicate range of friends' depressive symptoms in the respective quartile. Numbers within each cross-tabulation cell refer to the average Wave 2 depressive symptoms. Sample sizes are included in parenthesis. Q4-Q1 represents the interquartile range of mean Wave 2 depressive symptoms within each genotype row. An ANOVA test was used to test whether there is a statistically significant difference among quartiles within each genotype.

Finally, Model 3 includes interaction effects between peer network level of depressive symptoms and 5-HTTLPR genotype.

**4.8 Results**

Table 4.1 presents descriptive statistics for all variables included in this study. Average levels of depressive symptoms among adolescents are similar between Wave 1 (mean=11.2) and Wave 2 (mean=11.3). The triallelic coding procedure of this analysis results in a prevalence of individuals carrying the S'/L' genotype (43%) as compared to S'/S' (31%) and L'/L' (26%). Additionally, the demographic composition of this sample consists of 55% females, 48% white, 15% black, 7% Asian, and 15% Hispanic. The average age of the sample at Wave 1 is 15 with a range of 12 to 20.

First, I conduct a preliminary investigation into the primary hypothesis of this study suggesting that adolescents carrying one or two S alleles are more susceptible to depressive contagion. To examine this hypothesis, Table 4.2 displays a cross-tabulation

Table 4.3 Negative Binomial Model Predicting Wave 2 Depressive Symptoms with GxE of Friends' Depressive Symptoms and 5-HTTLPR

|  | Model 1 | | Model 2 | | Model 3 | |
|---|---|---|---|---|---|---|
|  | β | SE | β | SE | β | SE |
| Friends' Depressive Symptoms | .014*** | .002 | .008*** | .002 | .006* | .003 |
| 5-HTTLPR—S'/S' | .040 | .032 | .013 | .027 | .046 | .052 |
| 5-HTTLPR—S'/L' | .047 | .029 | .030 | .025 | -.069 | .046 |
|  |  |  |  |  |  |  |
| Friends' Depressive Symptoms x S'/S' | -- | -- | -- | -- | .011** | .004 |
| Friends' Depressive Symptoms x S'/L' | -- | -- | -- | -- | .009* | .004 |
|  |  |  |  |  |  |  |
| Wave 1 Depressive Symptoms | -- | -- | .050*** | .001 | .050*** | .001 |
| Female | .190*** | .024 | .086*** | .021 | .086*** | .021 |
| Age | .050*** | .008 | .017** | .006 | .017** | .006 |
| Black | .140*** | .033 | .086** | .030 | .089** | .030 |
| Asian | .188*** | .049 | .097* | .041 | .106* | .042 |
| Hispanic | .225*** | .035 | .114*** | .031 | .111*** | .031 |
| Other Race | .149*** | .031 | .085** | .029 | .085** | .029 |
| Intercept | 1.320*** | .118 | 1.348*** | .095 | 1.375*** | .100 |

Notes: *p<.05, **p<.01, ***p<.001 (two-tailed)
N=6,251

analysis of the interaction between quartile categories of friends depressive symptoms and the triallelic categorization of 5-HTTLPR in predicting Wave 2 depressive symptoms. The effect of interest in this table is the change in Wave 2 depressive symptoms in response to change from Quartile 1 to Quartile 4 of friends' depressive symptoms. This effect is shown through a measure of the interquartile range (Q4-Q1) within all three genotypes of 5-HTTLPR. The results show that increases in Wave 2 depression are significantly greater among adolescents carrying the S'/S' genotype (IQR=4.4) and S'/L' genotype (IQR=3.9) as compared to the L'/L' genotype (IQR=1.8). To statistically test this effect, I utilize an ANOVA test to examine whether there is statistically significant variation in Wave 2 depressive symptoms among quartiles within each genotypic category. Table 4.2 reports ANOVA results showing significant variation among quartiles within S'/S' and S'/L' genotypes.

Although Table 4.2 demonstrates evidence of a GxE effect, an ANOVA test is limited in that it does not adjust for demographic covariates and is unable to directly compare the effect of depressive contagion among genotypic categories. As a result, I utilize a negative binomial regression to test the main effects and a potential interaction effect of genotypic variation in 5-HTTLPR and friends' depressive symptoms. The results are shown in Table 4.3.

According to the findings in Model 1, friends' depressive symptoms are significantly associated with adolescent depressive symptoms even after controlling for demographic covariates of gender, race, and age. The direct effect of genotypic variation in 5-HTTLPR is also tested but shown to be statistically insignificant. This finding parallels findings from prior research suggesting only a moderating role for 5-HTTLPR. In regards to demographics, females have approximately 19% greater levels of depressive symptoms than males. Blacks, Asians, and Hispanics are likely to be more depressed than their white counterparts. Additionally, adolescents experience more depressive symptoms as they become older, approximately 5% for every one year increase in age.

Although Model 1 shows evidence for depressive contagion, previous research (Christakis and Fowler 2013) suggests potential selection or spurious effects whereby individuals become friends as a result of similarity in characteristics such as depressive levels (homophily) or individuals become depressed as a result of being embedded in a vulnerable environment (shared environments). Therefore, to attend to this concern, Model 2 enters a lagged dependent variable effectively adjusting for prior levels of depressive symptoms measured during the Wave 1 in-home interview. Even after controlling for the lagged effect, friends' depressive symptoms remain statistically significant in predicting adolescent depressive symptoms. Moreover, there remains no

evidence for a direct effect of genotypic variation while all demographic variables retain statistical significance.

Finally, Model 3 tests the primary hypothesis of this study suggesting that a polymorphism in the serotonin transporter significantly moderates the effect of depressive contagion. The model enters interaction terms of friends' depressive symptoms by S'/S' and S'/L' genotypes with L'/L' as the reference category. Significance of these interaction terms would indicate that the slope effect of friends' depressive symptoms for adolescents carrying the S'/S' or S'/L' genotypes significantly differ from adolescents carrying the L'/L' genotype. The results show a statistically significant coefficient for both interaction terms.



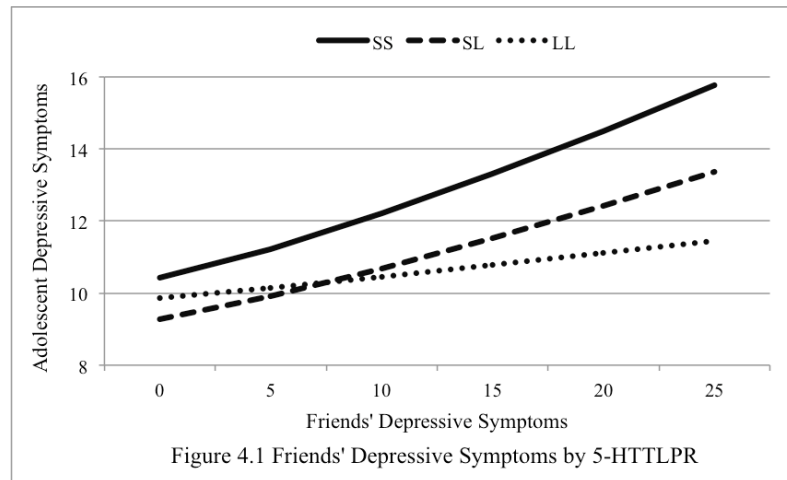Figure 4.1 Friends' Depressive Symptoms by 5-HTTLPR

Figure 4.1 provides an illustration of the interaction effect shown in Model 3. The figure depicts the relationship between friends' depressive symptoms and adolescent depressive symptoms. In other words, a positive slope would indicate evidence of the social contagion of depression. However, the present study hypothesizes that the effect of depressive contagion differs depending on a genotypic variation in 5-HTTLPR. Therefore, to illustrate this slope variation in depressive contagion, Figure 4.1 depicts separate lines for adolescents carrying the S'/S', S'/L', and L'/L' genotypes. As shown,

adolescents carrying one or two S alleles in 5-HTTLPR are significantly more susceptible to depressive contagion, thereby confirming the primary hypothesis of this study. Implications of these findings will be discussed in the next sections.

## 4.9 Discussion

Based on prior research utilizing a differential susceptibility model in both the GxE and social contagion literature, I hypothesized that adolescents carrying one or two S alleles in the 5-HTTLPR polymorphism would be more susceptible to the social contagion of depression. The findings confirmed the original hypothesis showing a significant moderation effect of genotypic variation in 5-HTTLPR on depressive contagion. Additionally, the results showed a significant main effect for friends' depressive symptoms in predicting adolescent depressive symptoms. No direct effect on depression was shown for a genotypic variation in 5-HTTLPR of the serotonin transporter region.

The purpose of this study was to integrate research on the social contagion of depression with GxE literature suggesting that genetic variation can moderate the effect of environmental stressors on depression. More specifically, research on depressive contagion is limited in that no prior study has incorporated genetic composition as a moderating effect despite the growing research on moderators of depressive contagion (Prinstein 2007). Moreover, empirical studies aimed at finding evidence for GxE effects are limited in its narrow operationalization of environmental exposures. Most research in this area adopts the social stress model and therefore operationalizes environmental stress as simply stressful life events. Unfortunately, this methodological tendency presents a significant limitation in fully examining the potential for GxE effects. This study attends to both prior limitations.

In this analysis, I examined an alternative operationalization of environmental exposure in the form of depressive contagion. The depressive contagion process asserts that individuals are susceptible to depression when embedded in peer networks characterized by high depressive levels. As a result, it is of particular interest that this significantly different alternative in measuring environmental exposure shows similar GxE results as prior research (e.g. Caspi et al. 2003; Eley et al. 2004) utilizing stressful life events. Although the findings in this study remain to be confirmed through the scientific process of replication, this study presents meaningful contributions. First, the results in this study offer support for the role of 5-HTTLPR in applying a differential susceptibility model to the relationship between environmental stressors and depression. Second, although prior research has documented individual and environmental factors that can enhance an individual's susceptibility or resilience to depressive contagion, this study is the first to examine variation in depressive contagion based on individuals' genotype.

**4.10 Implications**

As a result, the finding of a GxE effect between depressive contagion and 5-HTTLPR suggests fruitful opportunities for future research. First and foremost, replications of the present study should be the primary priority. Second, although replication is important, future research must continue to analyze alternative operationalizations of environmental exposure in the GxE perspective given the variety of social risk factors to chronic diseases such as depression. Finally, social contagion research would benefit in incorporating genetic composition in understanding variation in resilience and susceptibility to the contagion effect.

## Chapter 5: Conclusion

On August 17, 2015, National Public Radio (NPR) published an article suggesting that recent research on the social contagion of mental illness has produced two outcomes. First, the author suggests that the "beliefs" that mental illness can spread from one to another are "almost certainly false." Second, the author suggests that this research further stigmatizes people who suffer from mental illness. The research in this dissertation presents meaningful responses to both of these concerns. In response to the first, prior research on depressive contagion (Hogue and Steinberg 1995; Prinstein 2007; Conway et al. 2011; Cheadle and Goosby 2012) in addition to the three studies included in this dissertation present significant evidence for the social contagion of depression. This finding is replicated using various data samples and analytical methodologies to control for the effects of homophily and shared environments. In fact, Cheadle and Goosby (2012) utilize SIENA modeling methods to simultaneously estimate and compare the effects of homophily and contagion in determining network autocorrelation of depression. The authors find that contagion remains a significant predictor of depressive autocorrelation even after directly adjusting for homophily effects. As a result, the social contagion of depression represents one robust scientific finding in support of the contagion of mental illness.

Regarding the second and well-intentioned concern suggesting that research on mental health contagion can stigmatize and thereby socially isolate those with mental illnesses, the research in this dissertation presents a potential remedy. The evidence for depressive contagion is but the first step of research aimed at aiding public health researchers in designing network-driven intervention strategies in reducing rates of depression. The second and more important step is to analyze features of individuals and

environments that render certain people more susceptible to depressive contagion. This can include looking at psychosocial factors such as coping mechanisms or social support systems that may enhance resilience. This can also include applying demographic analyses to determine whether certain demographic populations are particularly more vulnerable to depressive contagion. Moreover, contagion does not occur within a vacuum. Examining structural features such as social network arrangements within which contagion is embedded can present fruitful knowledge for public health intervention strategists.

This dissertation represents the latter of the two steps in providing public health researchers with empirical studies documenting characteristics that can render individuals more susceptible to depressive contagion. In Chapter 2, I find that the effect of depressive contagion depends on the network context within which it is embedded. At the peer network level, popular adolescents and adolescents who are part of dense peer groups are more vulnerable to depressive contagion. At the school social network level, depressive contagion is more salient in schools characterized by high reciprocity in social ties and dense social networks. These findings demonstrate the importance of integrating multilevel understandings with the differential susceptibility model of social contagion.

Results in Chapter 3 show that race can play an important moderating role of depressive contagion. Adolescents embedded in racially homophilous networks are more susceptible to depressive contagion. However, this moderation effect is shown to significantly differ between black adolescents and Hispanic and Asian adolescents. I explain this finding using prior literature suggesting that the meaning of racial homophily can be significantly different among racial/ethnic groups. According to Quillian and Campbell (2003), racial homophily is viewed as a sense of racial solidarity given their

long history of racial oppression. Among Asians however, racial homophily can come to represent a failure to actively assimilate into the dominant culture.

Chapter 4 integrates the gene-environment interaction (GxE) paradigm in analyzing variation in the effect of depressive contagion. Results in this study show that individuals carrying one or two short alleles in a functional polymorphism (5-HTTLPR) in the serotonin transporter system (SLC6A4) are more susceptible to depressive contagion. This finding demonstrates the importance of 1) integrating genetic composition into social contagion research and 2) examining alternative operationalizations of environmental exposures, the "E" component of the GxE effect.

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