PROFILES OF REACTIVITY TO BULLYING VICTIMIZATION: GENETIC AND FAMILY ENVIRONMENT PREDICTORS

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A dissertation submitted to the faculty at the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Health Behavior in the Gillings School of Global Public Health.

Chapel Hill
2016

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ABSTRACT

Meridith Lyn Eastman: Profiles of Reactivity to Bullying Victimization: Genetic and Family Environment Predictors
(Under the direction of Vangie Foshee)

This dissertation identified profiles of internalizing (anxiety and depression) and externalizing (delinquency and violence against peers) reactivity to bullying victimization (Aim 1) and then examined the influence of bullying characteristics (type—i.e., direct, indirect, dual—and frequency) (Aim 2), family characteristics (parental warmth and family conflict) (Aim 3), and selected genetic polymorphisms (*5-HTTLPR, BDNF,* and *MAOA*) (Aim 4) on membership in these profiles. The sample for addressing Aims 1-3 was 1,196 bullying victims who participated in the Context/Linkages Study in three North Carolina counties in Fall 2003 when they were in grades 8-10. The sample for addressing Aim 4 was a subset (n=281) of bullying victims who provided a biospecimen for genotyping. Five profiles were identified using latent profile analysis (Aim 1): a non-reactive profile and four profiles that captured combinations of internalizing and externalizing. Associations between bullying type and frequency on membership in these reactivity profiles were identified in Aim 2 using multinomial logistic regression. Direct victimization (i.e., physical violence, name calling) increased odds of membership in the high internalizers, high externalizers profile compared to all other profiles. Indirect victimization (i.e., damage to social relationships) increased odds of membership in the high internalizing profiles compared to the lower internalizing profiles. Dual (i.e., direct and indirect) victimization increased odds of membership in the high internalizers, high externalizers profile compared to

each other profile. More frequent victimization increased odds of membership in the two high internalizing reactivity profiles compared to the non-reactor profile. Aim 3 tested the stress-buffering effects of parental warmth and the exacerbating effects of family conflict using logistic regression. The effects of parental warmth were different for boys and girls, with girls disproportionately benefitting from parental warmth. Family conflict increased likelihood of membership in the high internalizing, high externalizing profile compared to all others. The buffering and exacerbating effects were the same regardless of the frequency of the victimization experienced. Binary logistic regression analysis used for Aim 4 revealed no association between reactivity profile membership and genotype for the three candidate genes. Implications for intervention include recognition of heterogeneity in response to bullying and inclusion of family members.

To my parents, Richard and Marilyn, for always believing in me.

ACKNOWLEDGEMENTS

This dissertation could not have been accomplished without the invaluable guidance and mentorship of Vangie Foshee. I am extremely grateful for the expert contributions of each of my committee members: Susan Ennett, Robert Faris, Kari North, H. Luz McNaughton Reyes, and Daniela Sotres-Alvarez. I am also appreciative of the programming and statistical technical assistance provided by Chris Wiesen at the Odum Institute. Lastly, I am grateful to friends and family, near and far, for providing social and emotional support throughout my educational journey.

This work was funded in part by a predoctoral fellowship provided by the National Institute of Child Health and Human Development (T32-HD07376) through the Center for Developmental Science, University of North Carolina at Chapel Hill and by a Dissertation Completion Fellowship from the Graduate School of the University of North Carolina at Chapel Hill.

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LIST OF ABBREVIATIONS

5-HTTLPR	Serotonin transporter linked polymorphic region
BDNF	Brain derived neurotrophic factor
fMRI	Functional magnetic resonance imaging
LPA	Latent profile analysis
MAOA	Monoamine oxidase-A
SCT	Social Cognitive Theory
SLT	Social Learning Theory

CHAPTER 1: SPECIFIC AIMS

Although often experienced in childhood and adolescence, bullying has far-reaching consequences that extend across the life course. Approximately 11% of US school children in grades 6-10 have been victims of bullying (Nansel et al., 2001) and between 20-40% of children experience bullying at least once during their school careers (Shetgiri, Lin, & Flores, 2013). Numerous studies have found that bullying victimization has negative effects in childhood and adolescence and, furthermore, evidence suggests that victims of bullying are at heightened risk for psychological maladjustment in adulthood (Arseneault, Bowes, & Shakoor, 2010; Copeland, Wolke, Angold, & Costello, 2013; Hawker & Boulton, 2000; Menard, 2002). Not all bullying victims, however, respond to bullying in the same way (Arseneault, Bowes, & Shakoor, 2010). For some, victimization may result in internalization; others may experience externalizing problems. Another set of victims may experience both simultaneously (Arseneault, Bowes, & Shakoor, 2010). These findings suggest that there are different typologies, or profiles, of reactivity to victimization. The purposes of this dissertation were to 1) identify profiles of reactivity to bullying victimization and 2) examine predictors of reactivity profile membership.

Latent profile analysis (LPA) was used in this dissertation to identify profiles of internalizing (depression and anxiety) and externalizing (peer violence perpetration and delinquency) reactivity reported by adolescent victims of bullying. LPA is a type of mixture modeling, a set of statistical models that assumes that qualitatively different subpopulations exist within a larger population (Nylund, Asparouhov, & Muthén, 2007). LPA can be used to

determine the number of subpopulations—or profiles—that exist for a given set of outcomes, with what probability each individual is in each profile, and determine which variables most strongly predict profile membership (Collins & Lanza, 2010). Further, the use of LPA allows researchers to consider multiple outcomes simultaneously in the identification of profiles. In this study, reactivity profiles that reflect internalizing *and* externalizing responses were identified. The predictors of reactivity profiles membership that were examined in this dissertation were characteristics of the bullying victimization, aspects of the victim's family environment, and the victim's genotype for a select group of genes that have been found to influence reactivity to peer rejection and exclusion.

Characteristics of the bullying victimization were examined as predictors of reactivity profile membership because previous research suggests that reactivity to being bullied may vary depending on the type of bullying experienced--direct or indirect--and on the frequency of the bullying. Direct bullying may include physical acts of aggression but also name calling—anything that requires a face-to-face interaction—whereas indirect consists of spreading rumors, attempts at social exclusion, and talking behind the victim's back (Arseneault, Bowes, & Shakoor, 2010; Dukes, Stein, & Zane, 2009). While research has found that all forms of bullying are damaging (Hampel, Manhal, & Hayer, 2009; Klomek et al., 2009), Hampel et al. (2009) found that direct bullying victimization was more strongly associated with antisocial behavior and anger control problems, whereas indirect victimization was more strongly associated with emotional distress and negative self- image (Hampel, Manhal, & Hayer, 2009). With regard to the effects of frequency, Champion and Clay (2007) found that anger and motivation to retaliate were associated with frequency of victimization (Champion & Clay, 2007).

This dissertation also examined the influence of family warmth and family conflict as predictors of reactivity profile membership. Previous work by Bowes and colleagues identified the importance of family warmth in minimizing internalizing responses to childhood bullying victimization (Bowes, Maughan, Caspi, Moffitt, & Arseneault, 2010). In a 2013 study on adolescent victims of frequently bullying, Sapouna and Wolke (2013) found that victims who reported low levels of family conflict were among the most resilient, reporting low levels of depression and delinquency (Sapouna & Wolke, 2013). No study, however, has examined the role of the family context in explaining patterns of reactivity among victims of bullying.

Studies also suggest that biological factors, such as one's genotype, may influence reactivity to bullying (Beaver, Mancini, DeLisi, & Vaughn, 2011). Several genes, including *5-HTTLPR*, *BDNF*, and *MAOA* have been found to influence sensitivity to peer rejection and peer exclusion. Eisenberger and colleagues (2007) conducted a study that simulated social exclusion (via a game of "cyberball") while comparing brain activity to participants' self-reported social distress (Eisenberger, Way, Taylor, Welch, & Lieberman, 2007). fMRI data showed that areas of the amygdala and dorsal anterior cingulate cortex that correspond to self-reported social exclusion-related distress were disproportionately active in test subjects who were carriers of the low-activity alleles of *MAOA* (Eisenberger, Way, Taylor, Welch, & Lieberman, 2007). Meta-analyses suggest that stressful life events may interact with *5-HTTLPR* genotype to produce anxiety and depression, such that carriers of the short as compared to the long allele are more likely to experience anxiety and depression after a stressful life event (Karg, Burmeister, Shedden, & Sen, 2011). *BDNF* genotype has also been found in meta-analyses to moderate response to stressful life events, with carriers of the Met allele as compared to carriers of the Val

allele of the Val66Met polymorphism being more likely to experience depression following a stressful life event (Hosang, Shiles, Tansey, McGuffin, & Uher, 2014).

In summary, this dissertation addressed the following specific research aims:

Aim 1) Identify profiles of reactivity to bullying victimization in a sample of adolescents who have been victims of bullying;

Aim 2) Quantify the influence of bullying characteristics (type and frequency) on reactivity profile membership;

Aim 3) Quantify the influence of the family environment (parental warmth and family conflict) on reactivity profile membership; and

Aim 4) Quantify the influence of genotype (*5-HTTLPR*, *BDNF*, and *MAOA)* on reactivity profile membership.

Data for addressing Aims 1-3 of this dissertation were from a seven-wave longitudinal study of adolescent health risk behaviors (the *Context/Linkages Study*). The analytic sample consisted of 1,196 adolescents who reported that they had been victims of bullying in the Fall 2003 study questionnaire. The sample for Aim 4 consisted of 281 adolescents who reported that they had been victims of bullying in the Fall 2003 study questionnaire and who also provided genotyped biospecimens in a follow up study (the *Genes in Context Study*).

Three manuscripts were prepared for the dissertation. Manuscript 1 identified reactivity profiles and examined associations between characteristics of bullying and the reactivity profiles. Manuscript 2 examined associations between family characteristics and reactivity profile membership. Lastly, Manuscript 3 examined associations between three candidate genes (*5-HTTLPR*, *BDNF*, and *MAOA)* and reactivity profile membership. The next chapter, chapter 2,

discusses the definition of victims of bullying. Chapters 3-5 consist of the three manuscripts. Chapter 6 provides an overall summary and conclusions.

Identifying profiles of reactivity in this dissertation provided the opportunity to concurrently examine internalizing and externalizing outcomes associated with bullying victimization. Furthermore, understanding the family environment and genetic factors that contribute to membership in these profiles may provide the basis for interventions to mitigate the damaging effects of bullying victimization.

CHAPTER 2: DEFINITION OF BULLYING VICTIMIZATION

One of the most common definitions of bullying used in the literature is that of Dan Olweus, developed in 1978 (Olweus, 1978). Olweus distinguishes bullying from other forms of aggression because bullying 1) occurs between individuals of the same age group; 2) is characterized by an imbalance of power between the aggressor and the victim; and 3) occurs over a period of time (Olweus, 1978). The first part of this definition attempts to distinguish bullying from child maltreatment, which is when an adult acts aggressively toward a child. The second part of the definition highlights the difference between bullying and fighting that may occur in the context of a friendship or other more 'equal' peer relationship. The third characteristic underscores the importance of a pattern of behavior that establishes roles of perpetrator and victim. These roles unfold over time and become reinforced. Some researchers have challenged Olweus's definition. For example, Guerin and Hennessy (2002) note that even an action that is not intended by the perpetrator to cause harm may be interpreted by the victim as bullying (Guerin & Hennessy, 2002). They also note that the repetition of aggression may not need to be a criterion for bullying because one incident may cause the fear of repetition (Guerin & Hennessy, 2002). Additionally, Corvo and deLara (2010) suggest that measuring an imbalance of power between victim and perpetrator is unnecessary because children do not view a power differential as a dimension of bullying (Corvo & deLara, 2010). With these critiques in mind, this dissertation defines victims of bullying as those who report that at least one peer was mean to them or picked on them.

CHAPTER 3: LATENT PROFILES OF INTERNALIZING AND EXTERNALIZING REACTIVITY TO BULLYING AMONG ADOLESCENT VICTIMS (MANUSCRIPT #1)

Introduction

Bullying is a pervasive public health problem. Between 20-40% of children experience bullying at least once during their school careers (Shetgiri, Lin & Flores, 2013), and approximately 11% of US school children in grades 6-10 have been a victim of bullying (Nansel et al., 2001). The past 20 years have exhibited a change in how researchers and the general public view bullying (Piquero, Connell, Piquero, Farrington, & Jennings, 2013). Experiencing bullying was once considered a normal part of growing up, a rite of passage to be endured (Adams & Lawrence, 2011). However, media attention about suicides and acts of school violence, such as shootings at Columbine High School and Virginia Tech, which were attributed to retaliation for bullying, has called into question long-held lassez-faire attitudes about bullying (Dukes, Stein, & Zane, 2009). In response, educators, mental health practitioners, and researchers have increased their focus on understanding the negative impact that bullying can have on its victims. This study focused on identifying variation in reactivity to bullying and examined bullying characteristics associated with that variation. The term "reactivity" is being used based on longitudinal studies that have found that bullying victimization predicts the internalizing and externalizing attributes examined in this dissertation; however, longitudinal data needed to test for reactivity are not used in the present study.

Numerous studies have found that being bullied has negative effects in childhood and adolescence and, furthermore, evidence suggests that victims of bullying are at heightened risk

for psychological maladjustment in adulthood (Arseneault, Bowes, & Shakoor, 2010; Copeland, Wolke, Angold, & Costello, 2013; Hawker & Boulton, 2000; Menard, 2002). Not all bullying victims, however, respond to bullying in the same way (Arseneault, et al., 2010). For some, being bullied may result in internalizing problems (i.e., those that are harmful to self). These negative impacts include a range of deleterious mental health conditions including heightened social isolation, depression, and anxiety (Forero, McLellan, Rissel, & Bauman, 1999; Hawker & Boulton, 2000; Kaltiala-Heino, Rimpelä, Rantanen, & Rimpelä, 2000; Karatzias, Power, & Swanson, 2002; Nansel et al., 2001; Veenstra et al., 2005; Wolke, Woods, Bloomfield, & Karstadt, 2001).

Others may experience externalizing sequelae (i.e., those that are harmful to others) of bullying victimization. These include violent behavior towards others, carrying a weapon (Arseneault, Walsh, Trzesniewski, Newcombe, Caspi, Moffit, 2006; Kim, Leventhal, Koh, Hubbard, & Boyce, 2006; Liang, Flisher, & Lombard, 2007; Nansel, Overpeck, Haynie, Ruan, & Scheidt, 2003), and becoming a perpetrator of bullying behaviors (Barker, Arseneault, Brendgan, Fontain, & Maughan, 2008). Notably, in a sample of 3,932 adolescents in the Edinburgh Study of Youth Transitions and Crime, Barker et al. (2008) found that, in mid-adolescence, victimization increased the likelihood of bullying perpetration to a greater extent than bullying perpetration increased the likelihood of victimization (Barker, et al., 2008). This longitudinal study suggests that among bully/victims (i.e., students that are both bullies and victims of bullying), bullying perpetration is a response to victimization, rather than the other way around.

Another set of victims may experience both types of sequelae simultaneously (Arseneault, et al., 2010). For example, although they did not control for bullying perpetration, Hemphill and colleagues (2011) found that being victimized by bullying in grade 10 predicted a

twofold increased likelihood of depressive symptoms *as well as* increased likelihood of carrying a weapon, theft, and violent behavior in grade 11 (Hemphill et al., 2011). Similarly, in a cross-sectional study, Hampel et al (2009), measured antisocial behavior and anger control problems (externalizing) and distress and negative self-appraisal (internalizing) among 6-9th graders and found that victims experienced these outcomes (i.e., internalizing and externalizing) concurrently (Hampel, Manhal, & Hayer, 2009).

This variation in response to bullying victimization suggests that there may be different typologies, or profiles, of reactivity to victimization. Many studies examining the consequences of bullying victimization focus on either internalizing or externalizing responses. Such variable-centered approaches that focus on a single outcome ignore underlying heterogeneity in reactivity to bullying victimization and exclude complex reactions that incorporate internalizing and externalizing elements. By grouping individuals into categories based on similarity with one another and differences from those in other categories, person-centered approaches such as latent profile analysis can unmask this underlying heterogeneity to reveal group differences in patterns of responses to bullying (Muthén & Muthén, 2000; Laursen & Hoff, 2006). The first aim of this study was to determine whether there are different profiles of reactivity to bullying in a sample of adolescents who have been victims of bullying based on patterns of responses across a range of indicators of internalizing and externalizing behaviors.

LPA is a type of mixture modeling, a set of statistical models that assumes that qualitatively different subpopulations exist within a larger population (Nylund, Asparouhov, & Muthén, 2007). LPA can be used to determine the number of subpopulations—or profiles--that exist for a given set of indicators, with what probability each individual is in each profile, and which variables are most strongly associated with profile membership (Collins & Lanza, 2010).

In this study, we examined profiles of reactivity that emerged when examining two internalizing outcomes (anxiety and depression) and two externalizing outcomes (delinquency and physical violence against peers) among a sample of 8th-10th graders who self-identified as being a victim of bullying. Based on extant literature on varying responses to bullying we hypothesized (Hypothesis 1) that four profiles would be identified through LPA: one profile high on internalizing and low on externalizing, one profile low on internalizing and high on externalizing, one profile high on both internalizing and externalizing, and one profile low on both internalizing and externalizing.

Studies that have examined the influence of bullying victimization on different internalizing and externalizing outcomes have found that consequences differ depending on characteristics of victimization, including type of bullying experienced and frequency of victimization. The second aim of this study was to determine if characteristics of the bullying victimization (type and frequency) are associated with membership in reactivity profiles.

The terminology used to describe *types* of bullying varies across studies and intends to describe the mode through which harm is perpetrated against the victim. A common distinction made in types of bullying is between physical aggression and verbal aggression. The difference between the two is self-explanatory. Bullying behaviors have also been categorized into 'direct' and 'indirect' types wherein direct may include physical acts of aggression but also name calling—anything that requires direct interaction between bully and victim—and indirect comprises spreading rumors, attempts at social exclusion, and talking behind the victim's back (Arseneault et al., 2010; Dukes et al., 2009). 'Overt' and 'covert' bullying are often used as synonyms of direct and indirect bullying, respectively, and 'relational' bullying is also used as a synonym for indirect or covert bullying (Dukes, et al., 2009; Winsper, Lereya, Zanarini, &

Wolke, 2012). Although this study uses the terms direct and indirect to indicate the types of bullying examined, in the review of the literature that follows, we use the language of the respective study authors to summarize their findings about bullying type and internalizing and externalizing sequelae.

The relationship between bullying type and internalizing and externalizing outcomes was examined in a cross-sectional study of 6-9th graders in which Hampel et al. (2009) found that direct bullying victimization was more strongly associated with antisocial behavior and anger control problems, whereas indirect victimization was more strongly associated with emotional distress and negative self-image (Hampel et al.,2009). In a predominately African American sample of 8th graders, Sullivan and colleagues (2006), found that physical victimization was significantly related to perpetration of bullying and delinquent behaviors, whereas relational victimization was more strongly related to perpetration of relational bullying. Both associations were stronger for boys than girls (Sullivan, Farrell, & Kliewer, 2006). In a final example using a sample of 9th graders, Storch and colleagues (2005), found that relational victimization—but not overt victimization—predicted social phobia in both genders one year following victimization (Storch, Masia-Warner, Crisp, & Klein, 2005). These studies suggest that type of bullying experienced plays a role in the type of internalizing and/or externalizing response displayed by the victim. The present study examined whether experiencing direct bullying (physical and verbal overt forms of bullying), indirect bullying (actions to harm social relationships), or dual victimization (i.e., both direct and indirect) were associated with reactivity profile membership.

We hypothesized (Hypothesis 2) that adolescents who experienced any *direct* victimization would have a greater likelihood of membership in profiles characterized by high externalizing reactivity than in profiles not characterized by high externalizing reactivity. This

hypothesis is supported by the empirical literature summarized above, but also by Social Cognitive Theory (SCT) (Bandura, 1986). SCT posits that individuals model behavior they witness and experience in their social contexts. If an adolescent experiences direct bullying, SCT suggests that he/she may copy this behavior and respond by victimizing his/her peers or by externalizing in some other way. In contrast, we hypothesized (Hypothesis 3) that adolescents who experienced any *indirect* victimization would have greater likelihood of membership in profiles characterized by high internalizing reactivity than in profiles characterized by low internalizing reactivity. This hypothesis is rooted in the empirical literature cited above, but also reflects the subtle nature of indirect bullying. Because the harm against the victim is perpetrated not via direct attack, but rather through manipulation of social relationships, an effective external target for response may be difficult to identify. Direct confrontation with the perpetrator, for example, would not necessarily be effective in extinguishing a socially harmful rumor. Without an effective external target, frustrated victims may internalize the experience, leading to depression and anxiety. Further, we hypothesized (Hypothesis 4) that adolescents who experienced *dual* victimization would have greater likelihood of membership in the profile characterized by high internalizing and high externalizing than in the other profiles, thereby exhibiting characteristics of victims of both direct and indirect bullying.

Frequency of bullying is the number of times a person has been bullied over a particular reference period of time. Regarding the influence of frequency of victimization on negative sequelae of bullying, Penning et al. (2010) found that frequency of being bullied (no distinction made between bullying types) was associated with higher mean scores on five trauma subscales (anxiety, depression, posttraumatic stress, dissociation, and anger) of the Trauma Symptom Checklist for Children (TSCC) in a sample of 12-18 year old boys in South Africa. Similarly,

Klomek and colleagues (2009) found in a mixed-gender sample of 9-12 graders in New York State, that the more frequent involvement in bullying (either as a victim or a perpetrator; no distinction made between bullying types), the more likely an individual was to be depressed, to have serious suicide ideation, or to have attempted suicide (Klomek et al., 2009). Champion and Clay (2007) also found that more frequently victimized children responded to victimization (overt and relational victimization analyzed together) with more intense feelings of anger, more motivation to retaliate, less motivation to improve the situation, and more frequent intentions to aggress in a sample of Midwestern 4-6 graders (Champion & Clay, 2007). Taken together, these studies suggest that greater frequency of victimization has the potential to intensify the victimization experience regardless of bullying type, therefore intensifying both internalizing *and* externalizing reactivity among victims. We hypothesized (Hypothesis 5) that greater frequency of victimization would be associated with greater likelihood of membership in the higher reactivity profiles than in the non-reactive profile.

As noted above, sex differences have been observed in reactivity to bullying victimization. Therefore the third aim of this study was to examine whether the hypothesized associations between bullying type and frequency and reactivity profiles varied by sex of the victim.

Method

Data were from a seven-wave longitudinal study of adolescent health risk behaviors (Ennett et al. 2008; Foshee et al. 2011). Adolescents eligible for that study were those in grades 6 to 8 in the public school systems of three primarily rural counties in North Carolina, except for those who were unable to complete the survey in English (1-4 students per wave), in special education programs (.04% to .05% of students), or who were in long-term suspension or

expulsion (1-4 students per wave). Response rates in this study were high, ranging from 89% at wave 1 to 73% at wave 7. Parents had the opportunity to refuse consent for their child's participation by returning a written form or by calling a telephone number. Trained data collectors administered the questionnaires in classrooms. Assent was obtained from adolescents immediately prior to the survey administration from students whose parents had consented. The Institutional Review Board for the University of North Carolina at Chapel Hill approved the study.

Analytic Sample

The current study used the fourth wave of data, which was collected from the adolescents in Fall 2003, when they were in grades 8 to 10. This wave of data was used for the current analyses because assessments of bullying were introduced at this wave and the greatest number of students reported being a victim of bullying during this wave, thereby maximizing sample size for the present study. Despite the availability longitudinal data, use of multiple waves (e.g., victimization status at wave 4 to create profiles of internalizing and externalizing at wave 5) would not capture a full history of victimization—only victimization in the prior 3 months could be used in developing profiles. Additionally, latent profile analysis does not allow for the controlling of prior waves' levels of internalizing and externalizing. Therefore, wave 4 was used for the cross-sectional latent profile analysis. A total of 5,017 adolescents, from 8 middle schools, 2 K-8 schools, 3 alternative schools, and 6 high schools (19 schools total), completed the wave 4 questionnaire (79.1% of those eligible). At the time of the questionnaire administration, data collectors provided each student with a Student Directory that alphabetically listed enrolled students along with a unique four-digit peer identification number for each student. Bullying victimization was assessed in the questionnaire by asking students to identify

up to five peers *who had been mean to them or who had picked on them in the past 3 months* (i.e., bullied them). The analytic sample for the current study was limited to the 1,196 adolescents (23.8% of those who completed questionnaires) who indicated that any school peer had bullied them. The sample was 59.8% female. 56.9% reported their race as White, 27.5% Black or African-American, 4.1% Hispanic or Latino, 2.4% American Indian or Native American, 1.3% Asian or Pacific Islander, 4.7% Multiracial (mixed race), and 1.7% Other (total 41.7% Non-white).

Measures

Indicators for Latent Profile Analysis.

Internalizing. Internalizing symptoms were assessed with items from the Revised Children's Manifest Anxiety Scale (Reynolds & Richmond, 1979) and the Short Mood and Feelings Questionnaire (Angold, Costello, Messer, & Pickles, 1995). Four items from the Revised Children's Manifest Anxiety Scale assessed anxiety (e.g., "I worried about what was going to happen" and "I worried when I went to bed at night") within the past three months and four items from the Short Mood and Feelings Questionnaire assessed feelings of depression (e.g., "I did everything wrong" and "I was tired a lot"). Responses ranged from 0 ("strongly disagree") to 4 ("strongly agree"). Responses for the four anxiety items were summed to create the **anxiety score** (alpha =.86, *M*=8.34, *SD*=4.95, range=0-16); responses for the four depression items were summed to create the **depression score** (alpha = .86, *M*=6.47, *SD*=4.96, range=0-16). The distribution of both of these internalizing indicators was approximately normal; no transformation of these variables was required for analysis.

Externalizing. Two subtypes of externalizing were examined: delinquency and physical violence against peers. Delinquency was measured with four items that captured the frequency

with which the respondent skipped school, damaged property, threatened a teacher, or threatened someone with a weapon (Farrell, Kung, White, & Valois, 2000). Response options were: 0=none; 1=1-2 times; 2=3-5 times; 3=6-9 times; and 4=10 times or more. Responses to these items were summed to create a composite **delinquent behaviors score** (alpha=.80, *M*=1.16, *SD*=2.77, range=0-16). Physical violence against peers was measured with six items that captured how often in the past 3 months the respondent pushed, grabbed, shoved, or kicked a peer; slapped or scratched a peer; twisted a peer's arm or bent back a peer's fingers; hit a peer with a fist or with something else hard; beat up a peer; or assaulted a peer with a knife. Response options were: 0=none; 1=1-2 times; 2=3-5 times; 3=6-9 times; and 4=10 times or more. These six items were summed to create a composite **physical violence against peers score** (alpha=0.88, *M*=1.93, *SD*=4.18, range=0-24). The distributions of these two externalizing indicators were heavily right-skewed, violating the assumption of normality required for LPA. Thus, each of these externalizing outcomes was trichotomized after reviewing univariate statistics such that 0=none, 1=some, and 2=a lot of externalization. Cutoffs for the categories were based on univariate statistics so that the "a lot" category captured individuals at approximately the 90th percentile for the outcome and above, the "none" category consisted of individuals reporting no externalization, and individuals with scores between 0 and the approximate 90th percentile cutoff fell into the "some" category. Table 1.1 shows the composite score ranges and the percentile cutoffs for each of the three categories of each variable, as well as the number of adolescents in each category of each variable.

Table 1.1 Distributions of the trichotomized delinquency and violence against peers scales

	Score range	Percentile	N
Delinquency			
None	0	0-63rd	775
Some	1-2	64th -88th	283
A lot	3-16	89th-100th	138

	Score range	Percentile	N
Violence against peers			
None	0	0-60th	753
Some	2-4	61th-88th	313
A lot	5-24	89th-100th	130

Bullying Characteristics

***Bullying type*.** Adolescents were asked to indicate whether each student that they nominated as someone who had been mean to them or who picked on them in the past 3 months had "physically attack[ed] you in any way (hitting, shoving, tripping)?", "ma[de] fun of you or call[ed] you names to your face," and or "talk[ed] badly about you behind your back or tr[ied] to get others not to be friends with you". A **dichotomous direct bullying type variable** was created such that 1 indicated that a peer had physically attacked them in some way or made fun of them or called them names to their face and 0 indicated that a peer had not done these things to them. A **dichotomous indirect bullying type variable** was created such that 1 indicated that a peer had talked badly about them behind their back or tried to get others not to be friends with them and 0 indicated that a peer had not done these things to them. A **dichotomous dual victimization type variable** was created where 1 indicated that the adolescent was both directly and indirectly bullied and 0 indicated that the adolescent experienced only one type of victimization (either direct or indirect).

Bullying frequency. Each respondent was asked to indicate the frequency with which each nominated peer was mean to or picked on them. Response categories included: 5=6 or more times per week, 4=3 to 5 times per week, 3=1 to 2 times per week, 2=1 to 2 times per month, 1=2 times in the past 3 months. Frequency of bullying victimization was calculated by summing the frequency of victimization across all nominated peers (up to five). The bullying victimization frequency of the sample ranged from 1 to 25; *M*=8.72, SD=6.75.

Control Variables

To control for potential confounding effects, control variables used in analyses assessing associations between the bullying characteristics and reactivity profiles were respondent race, grade in school, and parental education. Respondent race was coded as 0=White, 1=Non-white, where non-white represented Black or African-American, Hispanic or Latino, American Indian or Native American, Asian or Pacific Islander, Multiracial (mixed race), and Other. The grade variable captured whether the student was in grade 8, 9, or 10 at the time of the assessment. Parental education was coded as 0=Did not graduate from high school, 1= Graduated from high school, 2=Some college or tech school, 3=Graduated from community college or tech school, 4= Graduated from college, 5=Graduate or professional school. Sex was treated as a moderator variable and was coded female=0, male=1.

Analysis Strategy

The analytic approach undertaken consisted of three major steps. First, latent profile analysis was conducted to identify the profiles of reactivity to bullying from anxiety, depression, delinquency, and physical violence against peers and to assign participants to profiles based on the highest probability of membership. The latent profile analysis was conducted in Mplus 7 using the expectation maximization algorithm with the robust maximum likelihood (MLR) estimator for the indicators of anxiety, depression, delinquency, and physical violence against peers. In these analyses, missing data were handled using full information maximum likelihood (FIML). 9.8% of observations were missing on anxiety, 10.3% on depression, 4.1% on delinquency, and 6.9% on violence against peers. A one-profile model was estimated first, followed by a two-profile model, and additional profiles were added sequentially until there was no improvement in model fit. Several criteria were used to evaluate the fit of latent profile

models: Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), bootstrapped likelihood ratio test (BLRT), and entropy. AIC (Akaike, 1974) and BIC (Schwarz, 1978) are relative fit statistics where lower numbers indicate improved model fit as compared to higher numbers. These statistics are based upon the log likelihood—the logarithm of the likelihood ratio--which expresses how many times more likely the data fit under a *k* profile model than a *k-1* profile model. The *p* value for the bootstrapped likelihood ratio test (BLRT; McLachlan & Peel, 2000) represents the results of a test that assesses whether a model of *k* profiles represents a better fit for the data than a model of *k*-1 profiles. *P* values of <.05 indicate that the *k* profile model better suits the data than a model of *k*-1 profiles. Lastly, entropy is a criterion that measures classification certainty and can range from 0-1 (Celeux & Soromenho, 1996). Higher values indicate that profiles have good separation; that is, that profiles are more distinct from one another. An entropy value of .80 represents good separation between the profiles (Ramaswamy et al., 1993). After the number and nature of the reactivity profiles were identified, individuals were assigned to their most likely profile based on their vector of posterior probabilities (that is, the set of values describing the likelihood of being assigned to that profile, given the data). Profile membership was subsequently used as the outcome in multinomial logistic regression models, described further below in step three, to quantify the association between type (direct, indirect, or dual) and frequency of bullying victimization and profile membership.

The second step was to conduct descriptive analyses of the distribution of bullying characteristics (type and frequency) by the identified reactivity profiles in SAS 9.4. Chi-square tests were used to determine if the distributions of the dichotomous bullying characteristics (direct, indirect, or dual) varied significantly across reactivity profiles. Post hoc Tukey-type

multiple comparisons of proportions developed by Zar (1999) were conducted using the *compprop* macro written by Elliott and Reisch (2006). One-way analysis of variance (ANOVA) tested for significant differences in mean levels of bullying frequency across identified reactivity profiles. Tukey's test was used to identify significant differences in mean levels of frequency between each reactivity profile. Listwise deletion was used so that only observations with complete data on bullying characteristics were included in the analysis.

The third step in the analysis was to conduct a series of multinomial logistic regressions using SAS v9.4 to test the hypotheses related to associations between bullying type (direct, indirect, or dual) and frequency with reactivity profile membership and whether these associations differed by sex. Note that type and frequency were not tested in the same models due to multicollinearity and also because models required different comparison groups depending upon the hypothesis being tested. We first tested for significance of the interactive effects of sex and bullying characteristics (direct, indirect, dual, and frequency) on reactivity profile membership. In all cases, interactions were found to be non-significant; therefore, interaction terms were dropped from subsequent models. We then tested for main effects of bullying characteristics and sex on reactivity profile membership controlling for the demographic characteristics. Rather than selecting a single reference category (i.e., the reactivity profile used for comparison) for all models, such as the largest profile, for example, the reference category for the logistic regression models necessarily varied according to the hypothesis being tested (see Results). In all cases, resulting odds ratios represent the likelihood of membership in each profile relative to the specified reference category.

Missing data for the covariates used in multinomial logistic regression were imputed using PROC MI, and PROC MIANALYZE was used to pool the results from the logistic

regression models fit on the imputed datasets. 18.1% of observations were missing on parental education, 9.1% were missing on race, and 3.5% were missing on bullying frequency. There were no missing data on gender, grade, bullying type, or latent profile. The multiple imputation models included all variables (including interactions) that were included in the logistic regression models as recommended by Allison (2002). With the exception of the nominal variable representing the latent profile, all variables in the multiple imputation models were quantitative in nature (either binary, ordered categorical, or continuous). Dummy variables representing each level of the nominal latent profile variable were created to ensure that the imputation procedure appropriately recognized the latent profiles as categorical. Minimum and maximum values were specified to ensure that plausible values were imputed for all variables. Twenty imputations were run based on recommendations by Graham and colleagues (2007).

Results

Latent Profile Analysis of Internalizing and Externalizing Reactivity to Bullying Victimization

Table 1.2 shows parameters of model fit for 1-6 profile models that were tested. Because the AIC, BIC, and BLRT results for the two-profile model indicated improved fit over the one-profile model, a three-profile model was estimated. AIC, BIC, and the BLRT for the three-profile model indicated improved fit over the two-profile model. A four-profile model showed further improved fit and, subsequently, so did the five- and six-profile models. Note, however, that the entropy value was worse in the six-profile model as compared to the five-profile model despite improved AIC, BIC, and BLRT results. This suggests that the five-profile model had better separation between profiles than the six-profile model. The five-profile model also demonstrated superior interpretability; the six-profile model did not provide conceptually meaningful

distinctions because it produced multiple profiles with very similar moderate amounts of internalizing and externalizing reactivity. Taking together the parameters of fit and these conceptual considerations, the five-profile model was determined to be the best representation of the data, rather than the four profiles that we hypothesized.

Table 1.2 Parameters of fit for 1-6 profile solutions for reactivity to bullying victimization

# of Profiles	AIC	BIC	LL	*p*-value for BLRT	Entropy
1	17204.014	17244.708	-8594.007	--	--
2	16623.936	16700.237	-8296.968	0.0000	.678
3	16266.324	16378.232	-8111.162	0.0000	.741
4	16134.471	16281.986	-8038.235	0.0000	.751
5	**15925.759**	**16108.881**	**-7926.879**	**0.0000**	**.843**
6	15878.925	16097.655	-7896.463	0.0000	.808

AIC=Aikake Information Criterion; BIC=Bayesian Information Criterion; LL=log likelihood; BLRT=Bootstrap Likelihood Ratio Test
Note: Best fitting model in bold

The five profiles showed distinct characteristics (See Table 1.3). Profile 1, named the "non-reactors," consisting of 27% of the sample, had the lowest mean levels of anxiety (*M*=2.75) and depression (*M*=0.38), and its members had the greatest probability of reporting no externalizing behaviors (76% reported no delinquency and 71% reported no violence). Profile 2, comprising 14% of the sample, consisted of adolescents reporting high levels of anxiety (*M*=11.69) and depression (*M*=11.26) and who also had an appreciable probability of reporting "some" or "a lot" of externalizing behaviors. Among these "high internalizers, moderate externalizers" there was a combined 0.44 probability of reporting "some" or "a lot" for both delinquency and violence against peers. Profile 3 was the smallest, consisting of 11.7% of the sample. This profile includes adolescents with the highest mean levels of anxiety (*M*=13.76) and depression (*M*=15.40) and also the highest probabilities of reporting "a lot" of delinquency

(0.31) and violence against peers (0.19). Profile 3, therefore, was named the "high internalizers, high externalizers". Probabilities of reporting "some" externalizing behaviors for this profile were 0.23 for delinquency and 0.19 for violence against peers. Profile 4 was the second largest profile, consisting of 24.8% of the sample. This profile showed moderately high levels of anxiety (*M*=8.11), yet comparatively low levels of depression (*M*=3.76). These adolescents, the "moderately anxious, moderate externalizers", had medium probabilities of reporting "some" delinquency and violence against peers (0.26 and 0.30, respectively) and low probabilities of reporting "a lot" of externalizing behaviors (0.08 for delinquency and 0.09 for violence against peers). Lastly, Profile 5 consisted of 22.4% of the sample and reflects internalizing scores that are in the middle when compared to other profiles (anxiety *M*=9.16; depression *M*=7.80) and levels of externalizing behaviors similar to Profile 4. These "moderate internalizers, moderate externalizers" reported medium mean levels of depression and anxiety (*M* =7.80 and 9.16, respectively) and medium probabilities of reporting "some" and "a lot" of delinquency and violence against peers (0.11 and 0.23 for delinquency; 0.12 and .24 for violence against peers).

Table 1.3 Profile prevalences, means (for internalizing reactivity), item response probabilities (for externalizing reactivity), and classification probabilities for the 5 profiles of reactivity to bullying victimization

	Profile 1: Non-reactors	Profile 2: High internalizers, moderate externalizers	Profile 3: High internalizers, high externalizers	Profile 4: Moderately anxious, moderate externalizers	Profile 5: Moderate internalizers, moderate externalizers
N(%)	323 (27.0)	167(14.0)	140(11.7)	297(24.8)	269 (22.4)
Anxiety (mean)	2.75	11.69	13.76	8.11	9.16
Depression (mean)	.38	11.26	15.40	3.76	7.80
Delinquency					
None	.76	.57	.46	.66	.66
Some	.16	.34	.23	.26	.23
A lot	.08	.10	.31	.08	.11
Violence					
None	.71	.56	.57	.62	.64
Some	.23	.31	.25	.30	.24
A lot	.07	.13	.19	.09	.12

	Profile 1: Non-reactors	Profile 2: High internalizers, moderate externalizers	Profile 3: High internalizers, high externalizers	Profile 4: Moderately anxious, moderate externalizers	Profile 5: Moderate internalizers, moderate externalizers
Classification probability	.95	.88	.92	.86	.86

Analytic Sample and Reactivity Profile Descriptive Statistics

Results from the bivariate descriptive statistics indicated that indirect victimization (X^2 (4, N=1196)=9.91, p=.0420), dual victimization (X^2 (4, N=1196)=19.61, p=.0006), and frequency of victimization (F(4,1149)=9.07, p=<.0001) were significantly associated with membership profile, whereas direct bullying was not associated with reactivity profile (X^2 (4, N=1196)=8.01, p=.0913) (See Table 1.4).

The post hoc Tukey-type test for pairwise comparisons indicated that the proportion of those in Profile 3 (high internalizers, high externalizers) who experienced indirect bullying (84.29%) was significantly higher than the proportion of those in Profile 1 (non-reactors) (74.68%) and in Profile 4 (moderately anxious, moderate externalizers) (72.05%) who experienced indirect bullying. No other differences in proportions experiencing indirect bullying were significant.

A significant association between dual victimization and reactivity profile was evident. The post hoc Tukey-type test for pairwise comparisons found that the proportion of those in Profile 3 (high internalizers, high externalizers) who experienced dual victimization (78.57%) was significantly higher than the proportion of those in Profile 1 (non-reactors) (61.92%), Profile 4 (moderately anxious, moderate externalizers) (58.25%), and Profile 5 (moderate internalizers, moderate externalizers) (60.97%) who experienced dual victimization. No other differences in proportions experiencing dual victimization were significant.

Post hoc analysis of the ANOVA using Tukey's test indicated that the average frequency of bullying experienced by victims in Profile 3 (high internalizing, high externalizing) (M=11.73) was significantly higher than that experienced in all other reactivity profiles (Profile 1 M =7.86; Profile 2 M=9.18; Profile 4 M=8.11; Profile 5 M=8.55).

Table 1.4. Distributions of bullying type and frequency across profiles

Profile	Descriptor	Total	Direct N(% of row total)		Indirect N(% of row total)		Dual N(% of row total)		Frequency M(SD)
			Yes	No	Yes	No	Yes	No	
1	Non-reactors	323	285(88.24)	38(11.76)	238(73.68)	85(26.32)	200(61.92)	123(38.08)	7.86(6.46)
2	High internalizers, moderate externalizers	167	147(88.02)	20(11.98)	133(79.64)	34(20.36)	113(67.66)	54(32.34)	9.18(6.80)
3	High internalizers, high externalizers	140	132(94.29)	8(5.71)	**118(84.29)[§]**	**22(15.71)[§]**	**110(78.57)[¶]**	**30(21.43)[¶]**	**11.73 (7.52)[†]**
4	Moderately anxious, moderate externalizers	297	256(86.20)	41(13.80)	214(72.05)	83(27.95)	173(58.25)	124(41.75)	8.11(6.11)
5	Moderate internalizers, moderate externalizers	269	229(85.13)	40(14.87)	204(75.84)	65(24.16)	164(60.97)	105(39.03)	8.55(6.90)
	Total (across all Profiles)	1196	1049(87.71)	147(12.29)	907(75.84)	289(24.16)	760 (63.55)	436(36.45)	8.72 (6.75)
	Significance test		8.01 [A] (*p*=.0913)		**9.91 [A] (*p*=.0420)**		**19.61 [A] (*p*=.0006)**		**9.07[B] (*p*=<.0001)**

A Results of Chi square test (df=4)

B Results of ANOVA F test (df=4,1149)

† Tukey's test indicated significantly higher mean than all other Profiles, alpha=0.05

§ Post hoc Tukey-type test indicated significant difference of proportions between Profile 3 vs. Profile 1 and 4

¶ Post hoc Tukey-type test indicated significant difference of proportions between Profile 3 vs. Profile 1, 4, and 5

Tests of Hypothesized Associations between Bullying Type and Frequency with Reactivity Profiles

Direct victimization. Our first set of multinomial logistic regression models tested Hypothesis 2 that adolescents who experienced any *direct* victimization would have a greater likelihood of membership in profiles characterized by high externalizing reactivity than in profiles not characterized by high externalizing reactivity. Reflecting the LPA results, this hypothesis required comparing Profile 3 (high internalizers, high externalizers) against each of the other profiles. Profile 3, therefore, was used as the reference category in the multinomial logistic regression models testing hypothesis 2.

Table 1.5 displays the results of the test of association between direct bullying victimization and reactivity profile membership, adjusting for demographic variables and controlling for indirect bullying. The comparisons between Profile 1 (non-reactors) and Profile 3 (high internalizers, high externalizers; OR=.30, 95% CI:.13,.68), Profile 2 (high internalizers, moderate externalizers) and Profile 3 (OR=.41, 95% CI: .17, .97), Profile 4 (moderately anxious, high externalizers) and Profile 3(OR=.28, 95% CI: .13,.63), and Profile 5 (moderate internalizers, moderate externalizers) and Profile 3 (OR: .27; 95% CI: .12, .60) were all significant. Thus, we found support for Hypothesis 2.

Table 1.5. Adjusted associations† (OR and 95% CI) between direct bullying victimization and reactivity profile membership

	Profile 1 vs. Profile 3		Profile 2 vs. Profile 3		Profile 4 vs. Profile 3		Profile 5 vs. Profile 3	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Direct Victimization	**.30**	**(.13,.68)****	**.41**	**(.17,.97)***	**.28**	**(.13,.63)****	**.27**	**(.12,.60)****
Indirect Victimization§	.51	(.30,.87)*	.64	(.35,1.17)	.42	(.25,.72)	.52	(.30,.90)
Sex (M vs F)	2.07	(1.36, 3.14)	.68	(.41,1.11)	1.06	(.69,1.62)	1.47	(.96,2.26)
Race (white vs nonwhite)	.88	(.57,1.37)	.66	(.40,1.08)	.79	(.51,1.44)	.92	(.59,1.44)
Parent Education	1.04	(.89,1.20)	.95	(.80,1.12)	1.03	(.89,1.20)	.99	(.85,1.14)
Grade	**.67**	**(.52,.85)****	.94	(.72,1.24)	.80	(.63,1.02)	.92	(.71,1.17)

Note: Profile 1= Non-reactors; Profile 2= High internalizers, moderate externalizers; Profile 3=High internalizers, high externalizers (reference category); Profile 4= Moderately anxious, moderate externalizers; Profile 5=Moderate internalizers, moderate externalizers.

†Adjusted odds ratios (OR) estimated from a multinomial logistic regression of reactivity to bullying profile membership on direct bullying victimization adjusting for covariates shown in the table.

§ Indirect victimization is used as a control variable in this model. Results of hypothesis tests regarding indirect victimization are reported in Tables 6 and 7.

*p<.05

**p<.01

Indirect victimization. The next set of logistic regression models tested Hypothesis 3 that adolescents who experienced any *indirect* victimization would have greater likelihood of membership in profiles characterized by high internalizing reactivity than in profiles characterized by low internalizing reactivity. LPA identified two profiles with higher internalizing reactivity: Profile 2 (high internalizers, moderate externalizers) and Profile 3 (high internalizers, high externalizers). Three profiles have lower internalizing reactivity: Profile 1 (non-reactors), Profile 4 (moderately anxious, moderate externalizers), and Profile 5 (moderate internalizers, moderate externalizers). Our multinomial logistic regression, therefore, required two sets of contrasts: Profile 2 compared to Profile 1, 4, and 5 and Profile 3 compared to Profile 1, 4, and 5. We begin our presentation of results for this hypothesis with the multinomial logistic regression models that used Profile 2 as the reference category in comparison to Profile 1, 4, and 5. These results are presented in Table 1.6.

Table 1.6 shows that, contrary to Hypothesis 3, indirect bullying victimization was not significantly associated with membership in any of the lower internalizing reactivity profiles when compared to Profile 2.

Table 1.6. Adjusted associations[†] (OR and 95% CI) between indirect bullying victimization and reactivity profile membership with Profile 2 (high internalizers, moderate externalizers) as the reference category

	Profile 1 vs. Profile 2		Profile 4 vs. Profile 2		Profile 5 vs. Profile 2	
	OR	95% CI	OR	95% CI	OR	95% CI
Indirect Victimization	.80	(.50,1.28)	.66	(.42,1.06)	.81	(.50,1.32)
Direct Victimization[§]	.74	(.40,1.36)	.70	(.38,1.27)	.66	(.36,1.20)
Sex (M vs F)	**3.05**	**(2.01,4.62)*** **	**1.56**	**(1.02,2.38)***	**2.16**	**(1.41,3.32)****
Race (white vs nonwhite)	1.34	(.88,2.05)	1.20	(.79,1.83)	1.40	(.91,2.15)
Parent Education	1.10	(.95,1.26)	1.09	(.95,1.25)	1.04	(.91,1.20)
Grade	**.71**	**(.56,.89)****	.85	(.67,1.07)	.97	(.77,1.23)

Note: Profile 1= Non-reactors; Profile 2= High internalizers, moderate externalizers (reference category); Profile 3=High internalizers, high externalizers; Profile 4= Moderately anxious, moderate externalizers; Profile 5=Moderate internalizers, moderate externalizers

†Adjusted odds ratios (OR) estimated from a multinomial logistic regression of reactivity to bullying profile membership on direct bullying victimization adjusting for covariates shown in the table.

§ Direct victimization is used as a control variable in this model. Results of hypothesis tests regarding direct victimization are reported in Table 5.

*p<.05

**p<.01

***p<.0001

Table 1.7 presents the results of multinomial logistic regression of reactivity profile membership on indirect bullying victimization with Profile 3 (high internalizers, high externalizers) as the reference category. The results show that, consistent with Hypothesis 3, there was a significant association between indirect victimization and membership in the lower internalizing profiles (Profile 1, non-reactors; Profile 4, moderately anxious, moderate externalizers; and Profile 5, moderate internalizers, moderate externalizers) as compared to Profile 3 and controlling for direct bullying victimization and demographic variables. Adolescents who experienced indirect victimization had 49% lower odds of membership in Profile 1 (OR=.51, 95% CI: .30,.87), 58% lower odds of membership in Profile 4 (OR=.42, 95% CI:.25,.72), and 48% lower odds of membership in Profile 5 (OR=.52, CI: .30, .90) compared to Profile 3.

Table 1.7. Adjusted associations† (OR and 95% CI) between indirect bullying victimization and reactivity profile with Profile 3 (high internalizing, high externalizing) as the reference category

	Profile 1 vs. Profile 3		Profile 4 vs. Profile 3		Profile 5 vs. Profile 3	
	OR	95% CI	OR	95% CI	OR	95% CI
Indirect Victimization	**.51**	**(.30,.87)***	**.42**	**(.25,.72)****	**.52**	**(.30,.90)***
Direct Victimization§	**.30**	**(.13,.68)****	**.28**	**(.13,.63)****	**.27**	**(.12,.60)****
Sex (M vs F)	**2.07**	**(1.36,3.14)****	1.06	(.69,1.62)	1.47	(.95,2.26)
Race (white vs	.88	(.57,1.37)	.79	(.51,1.23)	.92	(.59,1.44)

	Profile 1 vs. Profile 3		Profile 4 vs. Profile 3		Profile 5 vs. Profile 3	
	OR	95% CI	OR	95% CI	OR	95% CI
nonwhite)						
Parent Education	1.04	(.89,1.20)	1.03	(.89,1.20)	.99	(.85,1.14)
Grade	**.67**	**(.52,.85)****	.80	(.63,1.02)	.92	(.71,1.17)

Note: Profile 1= Non-reactors; Profile 2= High internalizers, moderate externalizers; Profile 3=High internalizers, high externalizers (reference category); Profile 4= Moderately anxious, moderate externalizers; Profile 5=Moderate internalizers, moderate externalizers.
†Adjusted odds ratios (OR) estimated from a multinomial logistic regression of reactivity to bullying profile membership on direct bullying victimization adjusting for covariates shown in the table.
§ Direct victimization is used as a control variable in this model. Results of hypothesis tests regarding direct victimization are reported in Table 5.
*p<.05
**p<.01

Taking together the results of the multinomial logistic regression contrasts using Profile 2 (high internalizers, moderate externalizers) and then Profile 3 (high internalizers, high externalizers) as the reference category, we conclude that our hypothesis regarding indirect bullying victimization was only partially supported. Significant associations between indirect bullying victimization and reactivity profile membership, independent of direct bullying victimization, were found only when Profile 3 was the referent high internalizing profile.

Dual victimization. Importantly, 63.6% of our sample reported experiencing both direct and indirect types of bullying; therefore, our next set of multinomial logistic regression models tested Hypothesis 4, which was that adolescents who experienced *both* direct and indirect victimization would have greater likelihood of membership in the profile characterized by high internalizing and high externalizing (Profile 3) than in the other profiles. To do so, we fit a set of models with Profile 3 as the reference profile. The results from these models are presented in Table 1.8.

Table 1.8. Adjusted associations† (OR and 95% CI) between dual bullying victimization and reactivity profile membership

	Profile 1 vs. Profile 3		Profile 2 vs. Profile 3		Profile 4 vs. Profile 3		Profile 5 vs. Profile 3	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Dual Victimization	**.43**	**(.27,.69)****	**.56**	**(.33,.94)***	**.37**	**(.23,.60)*****	**.42**	**(.26,.68)****
Sex (M vs F)	**2.01**	**(1.33,3.04)****	.66	(.41,1.08)	1.04	(.68,1.59)	1.40	(.92,2.15)
Race (white vs nonwhite)	.90	(.58,1.39)	.67	(.41,1.09)	.80	(.51,1.24)	.94	(.61,1.47)
Parent Education	1.04	(.89,1.21)	.95	(.80,1.12)	1.03	(.89,1.20)	.99	(.85,1.15)
Grade	**.67**	**(.52, .86)****	.95	(.72,1.25)	.80	(.63,1.03)	.93	(.72,1.19)

Note: Profile 1= Non-reactors; Profile 2= High internalizers, moderate externalizers; Profile 3=High internalizers, high externalizers (reference category); Profile 4= Moderately anxious, moderate externalizers; Profile 5=Moderate internalizers, moderate externalizers.

†Adjusted odds ratios (OR) estimated from a multinomial logistic regression of reactivity to bullying profile membership on direct bullying victimization adjusting for covariates shown in the table.

*p<.05

**p<.01

***p<.0001

Consistent with Hypothesis 4, adolescents who experienced both types of victimization were indeed more likely to be in Profile 3 (high internalizing, high externalizing) than in any of the other reactivity profiles. They had 57% lower odds of membership in Profile 1 (non-reactors) as compared to Profile 3 (OR=.43, 95% CI: .27,.69), 44% lower odds of membership in Profile 2 compared to Profile 3 (OR=.56, 95% CI: .33,.94), 63% lower odds of membership in Profile 4 compared to Profile 3 (OR:.37, 95% CI: .23,.60), and 58% lower odds of membership in Profile 5 compared to Profile 3 (OR=.42, 95% CI: .26,.68).

Frequency of victimization. Our last set of multinomial logistic regression models tested Hypothesis 5 that greater frequency of victimization would be associated with greater likelihood of membership in the higher reactivity profiles than in the non-reactor profile. To test this hypothesis, Profile 1(non-reactors) was used as the reference category against which membership in each other profile was compared. Table 1.9 summarizes the results of the multinomial logistic regression testing this hypothesis. We found that frequency of victimization was significantly associated with membership in Profiles 2 and 3 (the high internalizing profiles), with each one unit increase in bullying frequency being equivalent to a 4% (OR=1.04, 95% CI: 1.01,1.07) and a 9% (OR=1.09, 95% CI: 1.05, 1.12) respective increase in odds of profile membership compared to Profile 1. Hypothesis 5, then, was only partially supported. Frequency of bullying victimization did not increase odds of profile membership for all reactivity profiles but, rather, the significance of its effects was limited to the two high internalizing profiles (Profiles 2 and 3) compared to the non-reactors (Profile 1).

Table 1.9. Adjusted associations† (OR and 95% CI) between frequency of bullying victimization and reactivity profile membership

	Profile 2 vs. Profile 1		Profile 3 vs. Profile 1		Profile 4 vs. Profile 1		Profile 5 vs. Profile 1	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Frequency	**1.04**	**(1.01,1.07)****	**1.09**	**(1.06,1.12)*****	1.01	(.99,1.04)	1.02	(.99,1.05)
Sex (M vs F)	**.32**	**(.21,.48)*****	**.44**	**(.29,.67)****	**.51**	**(.37,.70)*****	**.69**	**(.49,.95)***
Race (white vs nonwhite)	.72	(.47,1.09)	1.01	(.65,1.57)	.89	(.62,1.25)	1.05	(.73,1.50)
Parent Education	.93	(.81,1.06)	.98	(.84,1.13)	1.00	(.89,1.13)	.96	(.85,1.08)
Grade	**1.41**	**(1.12,1.79)****	**1.50**	**(1.17,1.93)****	1.20	(.99,1.46)	**1.38**	**(1.13,1.69)****

Note: Profile 1= Non-reactors (reference category); Profile 2= High internalizers, moderate externalizers; Profile 3=High internalizers, high externalizers; Profile 4= Moderately anxious, moderate externalizers; Profile 5=Moderate internalizers, moderate externalizers.

†Adjusted odds ratios (OR) estimated from a multinomial logistic regression of reactivity to bullying profile membership on direct bullying victimization adjusting for covariates shown in the table.

*p<.05

**p<.01

***p<.0001

Associations between demographic variables and reactivity profiles. Associations between a set of demographic variables (sex, race, parental education, and grade) and reactivity profiles were tested in all models, with varying profiles used as the comparison profile and adjusting for varying bullying characteristics. Race and parental education were not associated with reactivity profile membership in any of the models, but there were significant associations between sex and reactivity profile membership and grade and reactivity profile membership in nearly all models.

In our test of Hypothesis 3 regarding indirect bullying, using Profile 2 (high internalizers, moderate externalizers) as the reference category, we found a significant association between sex and reactivity to bullying profile membership. Compared to girls, boys had approximately three times the odds of membership in Profile 1 (non-reactors) compared to Profile 2 (OR=3.05, 95% CI: 2.01, 4.62), 1.56 times the odds of membership in Profile 4 (moderately anxious, moderate externalizers) compared to Profile 2 (OR=1.56, 95% CI: 1.02,2.38) and over two times the odds of membership in Profile 5 (moderate internalizers, moderate externalizers) compared to Profile 2 (OR=2.16, 95% CI:1.41,3.32). Significant associations between sex and reactivity profile membership were also found when we used Profile 3 (high internalizers, high externalizers) as the reference category to test Hypothesis 3. In this model, boys had higher odds than girls of being in Profile 1(non-reactors) as compared to Profile 3 (OR=2.07, 95% CI:1.36, 3.14). Similarly, when testing Hypothesis 4 regarding dual victimization and reactivity profile membership with Profile 3 (high internalizers, high externalizers) as the reference category, boys had approximately two times the odds of girls of being in Profile 1 (non-reactors) compared to Profile 3 (OR=2.01, 95% CI: 1.33,3.04). With regard to the association between frequency of victimization and reactivity profile membership

(Hypothesis 5) we found that, compared to girls, boys had 68% lower odds of membership in Profile 2 compared to Profile 1 (OR=.32, 95% CI: .21,.48), 56% lower odds of membership in Profile 3 compared to Profile 1 (OR=.44, 95% CI: 29.,.67), 49% lower odds of membership in Profile 4 compared to Profile 1 (OR=.51, 95% CI:.37,.70), and 31% lower odds of membership in Profile 5 compared to Profile 1 (OR=.69, 95% CI:.49, .95). Taken together, the significant associations between sex and reactivity profile membership across these models suggest that girls are at higher risk for membership in all reactivity profiles (compared to the non-reactive profile) than boys.

We also found a significant association between grade and reactivity profile membership in all models. In testing the hypothesis between direct bullying victimization and reactivity profile membership (Hypothesis 2) using Profile 3 (high internalizers, high externalizers) as the reference category, we found a significant association between grade and reactivity profile membership (OR=.67; 95% CI: .52, .85), with each one-year increase in grade being associated with 33% decreased odds of membership in the non-reactors profile (Profile 1) as compared to the high internalizers, high externalizers profile (Profile 3). There was also a significant association between grade and reactivity profile in our test of the relationship between indirect bullying victimization and reactivity profile membership (Hypothesis 3) using Profile 2 as the reference category, with each one year increase in grade associated with 29% lower odds of being in Profile 1 compared to Profile 2 (OR=.71, 95% CI:.56,.89). When using Profile 3 as the reference category to test Hypothesis 3, we found each one year increase in grade to be associated with 33% lower odds of being in Profile 1 compared to Profile 3 (OR=.67, 95% CI:.52,.85). Similarly, in our test of Hypothesis 4 with Profile 3 as the reference category, each one year increase in grade was also associated with 33% lower odds of being in Profile 1

compared to Profile 3 (OR=.67, 95% CI:.52,.86). With regard to frequency of victimization and reactivity profile membership (Hypothesis 5), we found that, for each 1 year increase in grade level, students had a 41% increase in odds of membership in Profile 2 compared to Profile 1 (OR=1.41, 95% CI: 1.12,1.79), 50% increase in odds of membership in Profile 3 compared to Profile 1 (OR=1.50, 95% CI:1.17,1.93) and 38% increase in odds of membership in Profile 5 compared to Profile 1 (OR=1.38, 95% CI:1.13,1.68). On the whole, results across all models suggest that the risk for internalizing and externalizing reactivity to bullying victimization increases as adolescents advance from grade 8-10.

Discussion

We expected to find variation in reactivity to bullying victimization with LPA and our results confirmed the presence of subgroups of reactivity within our sample. Our first hypothesis was that four profiles of reactivity would be identified through LPA: one group high on internalizing and low on externalizing, one group low on internalizing and high on externalizing, one group high on both internalizing and externalizing, and one group low on both internalizing and externalizing. In contrast to the proposed profiles, the data supported a five-profile model consisting of a profile low on internalizing and externalizing (Profile 1, the non-reactors), a profile of high internalizers and moderate externalizers (Profile 2), a profile of high internalizers and high externalizers (Profile 3), a profile of moderately anxious, moderate externalizers (Profile 4), and a profile characterized by moderate levels of internalizing and externalizing (Profile 5). The profiles that emerged from the data reflect a more nuanced picture of reactivity than the hypothesized four profiles, allowing for identification of more moderate levels of internalizing and externalizing responses and in one case (Profile 4, the moderately anxious, moderate externalizers), distinguishing between the two types of internalizing responses

measured in this study: anxiety and depression. Our person-centered approach enabled the detection of the co-existence of internalizing and externalizing reactivity in our profiles, revealing more than could have been learned in a single outcome, variable-centered study. Of note, internalizing and externalizing problems coexist in all of the reactivity profiles, underscoring the importance of secondary interventions that address both types of responses among victims of bullying victimization.

Kretschmer and colleagues (2015) identified four profiles of maladjustment along dimensions of internalizing (withdrawal, somatic complaints, anxiety) and externalizing (delinquency, aggression) in early adolescence. These profiles consisted of Low [maladjustment], Internalizing, Externalizing, and Comorbid groups (Kretschmer et al., 2015). The present study and Kretschmer et al. used different measures to identify profiles, but readers of Kretschmer et al. there is some degree of comorbidity present even in the Internalizing and Externalizing profiles.

The descriptive analysis detected important distinctions among the profiles in terms of bullying characteristics experienced. Of note, Profile 3 (high internalizers, high externalizers) had the highest proportion of members who experienced indirect bullying and dual victimization and also had a mean frequency of victimization that was significantly higher than that in all other profiles. In testing our hypotheses, we further examined the relationship between bullying characteristics and reactivity profile membership.

Supporting Hypothesis 2 and in harmony with findings by Hampel et al.(2009) and Sullivan et al., (2006) we found that direct bullying victimization was associated with membership in the profile characterized by high externalizing reactivity (Profile 3) as compared to each of the other reactivity profiles. It is possible that when the harm is direct, retaliation may

be an attractive method for dealing with victimization for some. Such bully/victims or "aggressive victims" (Pellegrini, Bartini, & Brooks, 1999) may be captured in our high internalizing, high externalizing profile as well as all of the profiles that capture some externalizing. Due to the cross-sectional nature of our analysis, we cannot determine whether the externalizing behaviors in our profiles preceded or followed the bullying victimization; however, as noted previously, work by Barker and colleagues (2008) suggests that victimization precedes perpetration rather than the other way around. Equivalent longitudinal research on the temporality of victimization and delinquency is lacking. Work by Hampel and colleagues (2009) identified anger control problems and negative self-appraisal as potential psychological adjustment factors that may mediate the relationship between direct victimization and externalizing reactivity. Longitudinal analyses are needed to test the significance of these psychological adjustment factors and their respective roles in the experience of direct victimization and externalizing reactivity.

Our hypothesis regarding indirect bullying victimization (Hypothesis 3) was only partially supported. Significant associations between indirect bullying victimization and profile membership were found only when Profile 3 (high internalizers, high externalizers) was the referent high internalizing profile for comparisons against lower internalizing groups (Profiles 1,4, and 5). Researchers (e.g., Baldry & Winkel, 2008; Rigby & Slee, 1999) have suggested that, because of the covert nature of indirect victimization, victims may not report incidents of bullying to teachers and parents, leading to feelings of frustration and powerlessness. Additionally, as we noted in the introduction, an effective target for retaliation may be difficult to identify when the damage is not done via direct attack but, rather, through damage to social relationships. For these reasons, victims of indirect bullying may be less likely than victims of

direct bullying to act out against peers in retaliation. The present study, however, suggests that this may not be the case. Victims of indirect bullying that were higher internalizers (compared to lower internalizing groups) also reported high levels of externalizing behaviors.

Adolescents who experienced *both* types of bullying victimization were also more likely than those who experienced only one type of victimization to be in profile 3 (high internalizers, high externalizers) compared to any other profile. While either type of victimization is damaging, dual victimization appears to be particularly deleterious. Our findings are consistent with those of Hampel and colleagues (2009) who found that although both types of victimization in their study were associated with poor coping and emotional and behavioral problems, students who were direct *and* indirect/relational victims displayed the most unfavorable pattern of maladaptive coping and emotional and behavioral problems.

We found that frequency of bullying victimization was significantly associated with reactivity profile, with increased frequency associated with increased likelihood of membership in Profile 2 (high internalizers, moderate externalizers) and Profile 3 (high internalizers, high externalizers) than in the non-reactor profile (Profile 1). This finding is consistent with Penning and colleagues' work (2010) which found that increased frequency of bullying led to an increase in trauma symptoms and with Champion & Clay (2007), who found frequency to be associated with intense anger and motivation to retaliate. Repeated victimization, therefore, seems to have an intensifying effect on both internalizing symptoms and externalizing behaviors among victims of bullying. This is supported in the descriptive analyses by the finding that adolescents in Profile 3 (high internalizing, high externalizing) had significantly higher mean frequency of victimization than any of the other profiles. Multinomial logistic regression found that membership in both of the high internalizing profiles was associated with increased frequency of

victimization, perhaps suggesting that internalizing symptoms are particularly sensitive to repetitive victimization.

Taking together findings from all of our multinomial logistic regression models, it appears that girls are at heightened risk for membership in all reactivity profiles (vs. the non-reactor profile) compared to boys when controlling for type and frequency of bullying victimization, race, parent education, and grade. While studies show that the prevalence of bullying victimization is the same for girls and boys (Kochenderfer-Ladd, Ladd, & Kochel, 2009), our results suggest that the experience of victimization may be more damaging for girls. One potential explanation is that girls—more so than boys--are socialized to maintain and protect social relationships (Carbone-Lopez, Esbensen, & Brick, 2010). The social failure represented by bullying victimization may, therefore, be felt more acutely by girls than boys.

Interestingly, although previous research has reported that rates of bullying tend to decrease as children advance through high school/secondary school (Smith, Madsen, & Moody, 1999; Sourander Helstela, Helenius, Piha, 2000), the present study suggests that the risk for internalizing and externalizing reactivity increases as adolescents advance from grade 8-10. Maccoby (1988) postulated that adolescents are more upset than younger children when peer relationships are damaged (e.g., because of bullying) due to the concomitant loss of emotional support. It is possible that the increasing importance of peer relationships as adolescents advance in grades makes bullying victimization more damaging, even though it may occur less frequently. Another possibility for the increased reactivity in later grades is that those in higher grades may have experienced more victimization over time. Due to the cross-sectional nature of this study, we cannot examine whether the association between grade and membership in higher reactivity profiles as compared with the non-reactor profile is a cumulative effect of years of

persistent bullying victimization or whether it reflects other changes—psychological or environmental—for the bullying victim over time.

This study has several strengths. First, our large sample size (n=1196) enhances confidence in our ability to identify latent profiles of reactivity. Second, we used previously validated measures of reactivity with high internal consistency, as reflected by high Cronbach alphas to measure anxiety, depression, delinquency, and violence against peers. Third, we used a person-centered approach, LPA, which enabled us to holistically and simultaneously examine internalizing and externalizing responses to bullying victimization. In doing so, we were able to identify four subtypes of reactivity (and one non-reactor profile) that may be overlooked in variable-centered, single outcome studies. In all reactivity subtypes, internalizing and externalizing responses co-exist. It seems that adolescents are not *either* internalizers *or* externalizers, a finding that has implications for intervention with victims of bullying.

Despite these strengths, some limitations must be noted. In particular, we cannot attribute the internalizing and externalizing behaviors solely to the experience of bullying victimization. It is possible that the victims in our dataset were anxious, depressed, delinquent, or violent prior to their reported victimization. Our profiles represent emotional and behavioral states of victims of bullying, but they cannot be said to be *caused* by bullying victimization. Our use of the term "reactivity" must be considered with the caveat that our investigation was cross-sectional; indicators of internalizing and externalizing and bullying characteristics were measured simultaneously. Multicollinearity precluded us from testing the influence of bullying type and frequency on reactivity profile membership in the same multinomial logistic regression models. Additionally, skewness of our delinquency and violence against peers measures necessitated trichotomization, leading to some loss of information which could have influenced our ability to

identify the number and nature of latent profiles of reactivity. Lastly, although the inclusion of indicators for two types of outcomes (internalizing and externalizing) represents a significant contribution to the literature, the profiles identified here cannot be said to fully encapsulate all responses to bullying victimization. Types of reactivity not easily captured in the framework of internalizing symptoms and externalizing behavior, such as substance use, eating disorders, and suicidality are beyond the scope of this study. Their inclusion in future research may lead to identification of even more complex typologies of reactivity.

It must also be noted that mixture models, including LPA, are not without methodological and substantive controversy. For example, Bauer and Curran (2003) note that overextraction of discrete classes or profiles is likely when data are non-normal. We have guarded against this possibility by ensuring use of either normal or categorical indicators in the identification of our profiles. In addition to this methodological concern, critics of mixture modeling question whether true clustering of people along behavioral and psychological phenomena exists or whether the identification of such clusters is a statistical artifact using arbitrary cut points (e.g., see Eysenck, 1986, Meehl 1992, and Pickles & Angold, 2003 for more on this debate). We acknowledge this controversy and recognize that the profiles that emerged from our data are based on probability and membership in each is subject to error; however, we believe that unmasking and describing subtypes of internalizing and externalizing reactivity to bullying victimization has both theoretical and practical utility to understanding sequelae of bullying victimization.

While the largest profile in our sample consisted of adolescents who reported little internalizing or externalizing reactivity following bullying victimization (Profile 1, 27%), the majority of our sample fell into one of the four other profiles of reactivity, demonstrating varying

levels of internalizing and externalizing responses. Further research is needed to identify factors beyond bullying characteristics that may influence membership in these more reactive profiles. Examination of associations between characteristics of the family environment and genetic susceptibility to the effects of social exclusion and bullying reactivity are particularly promising factors of many that could be examined.

CHAPTER 4: FAMILY CHARACTERISTICS ASSOCIATED WITH LATENT PROFILES OF EXTERNALIZING AND INTERNALIZING AMONG ADOLESCENT VICTIMS OF BULLYING (MANUSCRIPT #2)

Introduction

Approximately 22% of US adolescents in grades 6-12 experience bullying during the school year (National Center for Education Statistics, 2015). Numerous studies have documented detrimental negative effects of bullying for children and adolescents and, furthermore, research indicates that victims of bullying are at heightened risk for psychological maladjustment into adulthood (Arseneault, Bowes, & Shakoor, 2010; Copeland, Wolke, Angold, & Costello, 2013; Hawker & Boulton, 2000; Menard, 2002). Although peer relationships become increasingly important in adolescence, parent and family relationships remain influential (Ledwell & King, 2013). It follows then, that examination of family characteristics, such as parental warmth and family conflict, may inform our understanding of variation in reactivity to bullying victimization among adolescents.

Adolescents have been found to vary in their responses to being bullied. For some, bullying victimization leads to internalizing (i.e., harmful to self) responses, such as increased social isolation, depression, and anxiety (Forero, McLellan, Rissel, & Bauman, 1999; Hawker & Boulton, 2000; Kaltiala-Heino, Rimpelä, Rantanen, & Rimpelä, 2000; Karatzias, Power, & Swanson, 2002; Nansel et al., 2001; Veenstra et al., 2005; Wolke, Woods, Bloomfield, & Karstadt, 2001). Other victims of bullying exhibit externalizing (i.e., harmful to others) responses, such as violent behavior towards others, carrying a weapon (Arseneault, Walsh, Trzesniewski, Newcombe, Caspi, Moffit, 2006; Kim, Leventhal, Koh, Hubbard, & Boyce, 2006;

Liang, Flisher, & Lombard, 2007; Nansel, Overpeck, Haynie, Ruan, & Scheidt, 2003), and becoming a perpetrator of bullying behaviors (Barker, Arseneault, Brendgan, Fontain, & Maughan, 2008). Additionally, some victims exhibit both internalizing and externalizing reactivity (Arseneault, Bowes, & Shakoor, 2010; Eastman et al., unpublished; Hampel, Manhal, & Hayer, 2009; Hemphill et al, 2011; Kretschmer, Barker, Dijkstra, Oldehinkel,& Veenstra, 2015). Noting this variation in responses to bullying, previous research used latent profile analysis to identify profiles of reactivity to bullying based on levels of internalizing (anxiety and depression) and externalizing (delinquency and violence against peers) (Eastman et al, unpublished). Five profiles were identified in this research: non-reactors (Profile 1), high internalizers, moderate externalizers (Profile 2), high internalizers, high externalizers (Profile 3), moderately anxious, moderate externalizers (Profile 4), and moderate internalizers, moderate externalizers (Profile 5). The present study builds upon these findings to examine whether characteristics of the family, specifically parental warmth and family conflict, are associated with adolescent reactivity to bullying.

Parental Warmth

Warm and supportive relationships with parents have been found to buffer against negative outcomes related to interactions at home and school (Laursen & Collins, 2009), and parental social support, communication, and warmth have been associated with reduction in reactivity, specifically, to bullying victimization (Bowes, Maughan, Caspi, Moffitt, & Arseneault, 2010; Coohey, Renner, & Sabri, 2013; Davidson & Demaray, 2007; Holt & Espelage, 2007; Ledwell & King, 2013).

Parental warmth may be conceived of as a particular type of social support—emotional social support, specifically—which is the provision of empathy, concern, caring, love, and

acceptance (Langford, Bowsher, Maloney, & Lillis, 1997). In their seminal work on stress and social support, Cohen and Wills (1985) described the stress-buffering potential of social support. According to their conceptualization, social support is particularly influential on well-being when individuals are experiencing stress. It is the potential stress-buffering effect of *parental warmth*, in particular, that will be the focus of this paper.

Consistent with the stress-buffering conceptualization of social support, parental warmth is thought to be particularly important for adolescents experiencing bullying (Malecki & Demaray, 2004). The buffering is thought to occur in two ways. First, belief in, or knowledge of, the availability of parental warmth before a stressor occurs may increase the adolescent's belief in his/her own ability to cope with a crisis when it occurs (Davidson & Demaray, 2007). Second, parental warmth may mitigate the harmful consequences (e.g., internalizing and externalizing reactivity) of the stressor after the fact by reducing negative appraisals of the situation (Davidson & Demaray, 2007). To test the stress-buffering hypothesis of parental warmth, we hypothesized (Hypothesis 1) that adolescent victims of bullying who experienced parental warmth as compared to those who did not would have a greater likelihood of membership in Profile 1 (non-reactors) compared to the other reactivity profiles. Furthermore, because the severity of bullying victimization has been found to be associated with reactivity (Penning et al, 2010) and the stress-buffering conceptualization of social support suggests that the buffering effects of social support may be even greater in higher stress situations (Cohen and Wills, 1985), we hypothesized (Hypothesis 2) that parental warmth would moderate the association between frequency of bullying victimization (an indicator of the severity of the bullying victimization) and reactivity profile such that more frequent victims of bullying would disproportionately benefit from the buffering effects of parental warmth. That is, more frequent victims of bullying

will have greater likelihood of membership in Profile 1 (non-reactors) compared to the other reactivity profiles when they are in environments characterized by higher, as opposed to lower, levels of parental warmth. Support of either hypothesis would be consistent with the stress-buffering conceptualization of social support.

Research is mixed on whether there are or are not sex differences in the buffering effect of parental warmth on type of reactivity (internalizing or externalizing) demonstrated by the bullying victim. For example, Davidson and Demaray (2007), found that high parental social support was associated with reduced internalizing symptoms for girls who had experienced bullying victimization, but not for boys, and that parental social support did not protect against externalizing reactivity in response to bullying victimization for either sex (Davidson & Demaray, 2007). Ledwell and King (2013), examined only internalizing symptoms, but found that the buffering effect of parental communication was the same for boys and girls (Ledwell & King, 2013). In contrast, Tanigawa and colleagues (2011), found that parental social support buffered depressive symptoms for male victims of bullying, but not for female victims (Tanigawa, Furlong, Felix, & Sharkey, 2011). Because of these conflicting findings we examined whether our hypotheses about the buffering effects of parental warmth on reactivity to bullying varied by sex of the adolescent.

Family Conflict

Social Learning Theory suggests that adolescents model their response to social conflict upon responses they see in the external environment (Bandura, 1973). It follows, then, that adolescents who witness violent responses to conflict at home may respond violently to conflict they experience themselves outside the home. In a study of externalizing behavior, Coohey and colleagues (2013) reported that, for white adolescents, both victimization and parental conflict

were associated with greater externalization for both girls and boys (Coohey, Renner, & Sabri, 2013). The converse was also shown to be true by Sapouna & Wolke (2013), who found that low family conflict was associated with both reduced externalizing and internalizing reactivity among victims of frequent bullying (Sapouna & Wolke, 2013). Additionally, violence at home may serve as implicit communication from parents about the acceptability of responding to conflict with violence (Bettencourt & Farrell, 2013). For example, Farrell and colleagues (2010) reported that African American adolescents whose parents responded to conflict with non-violence were also likely to respond to conflict with non-violence (Farrell et al., 2010). Taken together, these studies suggest that conflict experienced at home may serve to heighten, or exacerbate, externalizing reactivity among victims of bullying.

Based on Social Learning Theory and the extant literature, we hypothesized (Hypothesis 3) that adolescent victims of bullying who experienced family conflict as compared to those who did not would have a greater likelihood of membership in the profile characterized by high externalizing (Profile 3) compared to the other reactivity profiles. Furthermore, we hypothesized (Hypothesis 4) that family conflict would moderate the association between frequency of bullying victimization (an indicator of the severity of the bullying victimization) and reactivity profile such that more frequent victims of bullying would be disproportionately harmed from the exacerbating effects of family conflict. That is, more frequent victims of bullying will have greater likelihood of membership in the high externalizing profiles (Profile 3) compared to the other reactivity profiles when they are in environments characterized by higher, as opposed to lower, levels of family conflict .

Research on potential sex differences in the relationship between exposure to family conflict and reactivity to bullying victimization is scant; however, Yang & McLoyd (2015), in a

cross-lagged path analysis, found that greater family conflict significantly increased antisocial outcomes among girls but not boys who experienced peer victimization. We examined whether our hypotheses about the exacerbating effects of family conflict on reactivity to bullying varied by sex of the adolescent.

Method

Data were from a seven-wave longitudinal study of adolescent health risk behaviors (Ennett et al. 2008; Foshee et al. 2011b). Adolescents eligible for that study were those in grades 6 to 8 in the public school systems of three primarily rural counties in North Carolina, except for those who were unable to complete the survey in English (1-4 students per wave), in special education programs (.04% to .05% of students), or who were in long-term suspension or expulsion (1-4 students per wave). Response rates in this study were high, ranging from 89% at wave 1 to 73% at wave 7. Parents had the opportunity to refuse consent for their child's participation by returning a written form or by calling a telephone number. Trained data collectors administered the questionnaires in classrooms. Assent was obtained from adolescents immediately prior to the survey administration from students whose parents had consented. The Institutional Review Board for the University of North Carolina at Chapel Hill approved the study.

Analytic Sample

The current study used the fourth wave of data, which were collected from the adolescents in Fall 2003, when they were in grades 8 to 10. This wave of data was used for the current analyses because assessments of bullying were introduced at this wave and the greatest number of students reported being a victim of bullying during this wave, thereby maximizing sample size for the present study. A total of 5,017 adolescents, from 8 middle schools, 2 K-8

schools, 3 alternative schools, and 6 high schools (19 schools total), completed the wave 4 questionnaire (79.1% of those eligible). At the time of the questionnaire administration, data collectors provided each student with a Student Directory that alphabetically listed enrolled students along with a unique four-digit peer identification number for each student. Bullying victimization was assessed in the questionnaire by asking students to identify up to five peers *who had been mean to them or who had picked on them in the past 3 months* (i.e., bullied them). The analytic sample for the current study was limited to the 1,196 adolescents (23.8% of those who completed questionnaires) who indicated that any school peer had bullied them.

Measures

Independent Variable

Bullying victimization frequency. Each survey respondent was asked to indicate the frequency with which each nominated peer was mean to or picked on them. Response categories included: 5=6 or more times per week, 4=3 to 5 times per week, 3=1 to 2 times per week, 2=1 to 2 times per month, 1=2 times in the past 3 months. Frequency of bullying victimization was calculated by summing the frequency of victimization across all nominated peers (up to five). The bullying victimization frequency of the sample ranged from 1 to 25; M=8.72, SD=6.75.

Moderator Variables

Parental warmth. The survey included a total of six items assessing parental warmth that were asked first about the respondent's mother, and then about the respondent's father. Three items assessed how well the following statements described the adolescent's mother or father: He/she tells me when I do well on things; he/she makes me feel better when I am upset; and he/she wants to hear about my problems (Jackson, Henriksen, & Foshee, 1998). Responses ranged from 0 ("not at all like him/her") to 3 ("just like him/her"). A fourth item assessed how

often a parent kisses or hugs the respondent. Responses ranged from 0 ("never") to 3 ("a lot"). Two additional items asked how close the respondent feels to the indicated parent, and how close the respondent thinks the indicated parent feels to the respondent. Responses ranged from 0 ("not close at all") to 3 ("very close"). Scores for these six items were summed for the mother and father separately, and the highest **parental warmth score** in the household (either maternal or paternal) was used in analysis (maternal warmth Cronbach's alpha=0.88; paternal warmth Cronbach's alpha =0.90; Mean parental warmth score=14.38, SD=4.13, range 0-18).

Family conflict. Three items in the survey assessed the level of conflict in the adolescent's family: "Think about your family life in the past 3 months. How strongly do you agree or disagree with each statement? a. We fight a lot in our family, b. Family members sometimes get so angry they throw things, c. Family members sometimes hit each other" (Bloom, 1985). Responses ranged from 0 ("strongly disagree") to 4 ("strongly agree). Scores were summed across these three items to create a **family conflict score** (Cronbach's alpha=0.87; *M*=3.82, *SD*=3.76, range=0-12).

Sex of the adolescent. Adolescent sex was coded 0=female, 1=male.

Dependent Variable

Reactivity to Bullying Victimization Profile. As noted earlier, previous research using latent profile analysis (LPA) identified five profiles of internalizing and externalizing reactivity to bullying victimization (Eastman et al., unpublished). Eighteen items were used in those analyses to develop two indicators of internalizing (anxiety and depression) and two indicators of externalizing (delinquency and violence against peers). Items for the two internalizing indicators were four Likert-type scale items measuring symptoms of anxiety (Reynolds & Richmond, 1979; alpha =.86, *M*=8.34, *SD*=4.95, range=0-16) and four Likert-type scale items measuring

symptoms of depression (Angold, Costello, Messer, & Pickles, 1995; alpha = .86, *M*=6.47, *SD*=4.96, range=0-16). Four items for the two externalizing indicators measured the frequency with which the student perpetrated delinquent acts (Farrell, Kung, White, & Valois, 2000; alpha=.80, *M*=1.16, *SD*=2.77, range=0-16) and six items assessed the frequency with which the student perpetrated violent acts against peers (alpha=0.88, *M*=1.93, *SD*=4.18, range=0-24). The distributions of these two externalizing indicators were heavily right-skewed, violating the assumption of normality required for LPA. Thus, each of the externalizing indicators was trichotomized such that 0=none, 1=some, and 2=a lot of externalization. Cutoffs for these three categories were based on univariate statistics so that the "a lot" category captured individuals at approximately the 90th percentile for the outcome and above, the "none" category consisted of individuals reporting no externalization, and individuals with scores between 0 and the approximate 90th percentile cutoff fell into the "some" category. The five profiles resulting from the LPA were: Profile 1, non-reactors (i.e., low on internalizing and externalizing reactivity); Profile 2, high internalizers and moderate externalizers; Profile 3, high internalizers and high externalizers, Profile 4, moderately anxious, moderate externalizers; Profile 5, moderate internalizers and moderate externalizers. After the number and nature of the reactivity profiles were identified through LPA, individuals were assigned to their most likely profile based on their vector of posterior probabilities (that is, the set of values describing the likelihood of being assigned to that profile, given the data). A **nominal profile variable** was created and profiles were given values of 1-5 as identified above. 27% of the sample fell into Profile 1 (non-reactors), 14% fell into Profile 2 (high internalizers, moderate externalizers), 12% fell into Profile 3 (high internalizers, high externalizers), 25% were categorized as Profile 4 (moderately anxious,

moderate externalizers), and 22% were categorized as Profile 5 (moderate internalizers, moderate externalizers).

Control Variables

To control for potential confounding effects, analyses controlled for respondent race, grade in school, and parental education. Respondent race was coded as 0=White, 1=Non-white, where Non-white represented Black or African-American, Hispanic or Latino, American Indian or Native American, Asian or Pacific Islander, Multiracial (mixed race), and Other. The grade variable captured whether the student was in grade 8, 9, or 10 at the time of the assessment. Parental education was coded as 0=Did not graduate from high school, 1= Graduated from high school, 2=Some college or tech school, 3=Graduated from community college or tech school, 4= Graduated from college, 5=Graduate or professional school.

Analysis Strategy

To test the parental warmth hypotheses (Hypotheses 1 and 2) we created a dichotomous reactivity profile variable such that membership in Profile 1 (non- reactors) was coded as 1 and membership in Profiles 2-5 (all reactivity profiles) was coded as 0. Then, to test the family conflict hypotheses (Hypotheses 3 and 4) we created a different dichotomous reactivity variable where membership in Profile 3 (the only profile characterized by high externalizing) was set equal to 1 and membership in Profiles 1, 2, 4, and 5, was set equal to 0. We then used logistic regression to test study hypotheses, first testing the parental warmth hypotheses with the above described dichotomous outcome variable (non-reactors vs all reactivity profiles), and then the family conflict hypotheses with the other dichotomous variable (profile 3 vs all others). The first step in the logistic regression for testing each of the two sets of hypotheses was to test a full model that included all independent variables, including frequency of bullying victimization, the

targeted family characteristic variable, sex, race, grade in school, and parent education, the two-way interactions of frequency x the family characteristic, frequency x sex, and the family characteristic x sex, and the three-way interaction of frequency x the family characteristics x sex in a logistic regression model with the indicated dichotomous reactivity profile membership variable as the outcome. Family conflict was also included as a control variable in the model testing the parental warmth hypotheses and parental warmth was included as a control variable in the model testing the family conflict hypotheses with the intent of assessing the unique effects of each family characteristic on bullying victimization reactivity. We reduced the models by first eliminating the three-way interaction because it was not significant in either model, then by dropping non-significant two-way interactions using a backward elimination procedure (Hosmer, Lemenshow, and Sturdivant, 2013). This procedure involved removing the non-significant two-way interaction that contributed the least to the logistic regression model (i.e., the term with the highest non-significant *p*-value), then refitting the model, and repeating this procedure until the final reduced models included all of the independent variables and only statistically significant ($p < .05$) two-way interaction terms.

Interactive effects that were retained in the final model were probed and plotted according to procedures outlined by Aiken & West (1991) using an Excel worksheet developed by Jeremy Dawson (Dawson, n.d.). After conducting the binary logistic regression analysis, which was the primary test of the hypotheses, we conducted exploratory analysis with multinomial logistic regression, using the original 5-value profile reactivity membership variable as the dependent variable, to determine for which of the reactivity profiles (compared to Profile 1 for parental warmth; compared to Profile 3 for family conflict) family characteristics were influential.

Hypothesis 1 would be supported if the main effect of parental warmth is significant in the final logistic regression model such that it increases the odds of membership in the non-reactor profile (Profile 1) compared to all others. Hypothesis 2, that the relationship between frequency of bullying victimization and reactivity profile membership will be moderated by parental warmth, would be supported if the frequency x parental warmth interaction is retained in the final logistic regression model and the nature of the interaction is such that higher frequency of victimization in high parental warmth environments (as compared to low parental warmth environments) is associated with increased odds of membership in the non-reactor profile (Profile 1) compared to all others. Support for either Hypothesis 1 or Hypothesis 2 would be consistent with the stress-buffering conceptualization of parental warmth. Hypothesis 3 would be supported if the main effect of family conflict is significant in the final model such that it increases odds of membership in the high externalizing profile (Profile 3) compared to all others. Hypothesis 4, that the relationship between frequency of bullying victimization and reactivity profile membership will be moderated by family conflict, would be supported if the frequency x family conflict interaction is retained in the final logistic regression model and the nature of the interaction is such that higher frequency of victimization in high family conflict environments (as compared to low family conflict environments) is associated with increased odds of membership in the high externalizing profile (Profile 3), as compared to all others. Support for either Hypothesis 3 or Hypothesis 4 would be consistent with the idea that family conflict exacerbates reactivity to bullying victimization. Variation by sex in these hypotheses would be indicated if either the two-way interaction of family characteristic x sex or the three-way interaction of family characteristic x frequency x sex is found to be significant in the final logistic regression model.

Missing data for the variables used in the binary and multinomial logistic regression models were imputed using PROC MI, and PROC MIANALYZE was used to pool the results from the logistic regression models that were fit on the imputed datasets. 10.7% of observations were missing on parental warmth and 11.3% were missing on family conflict. 18.1% of observations were missing on parental education, 9.1% were missing on race, and 3.5% were missing on bullying frequency. There were no missing data on gender, grade, or latent profile. The multiple imputation missingness models included all variables (including interactions) that were included in the binomial logistic regression models as recommended by Allison (2002). All variables in the multiple imputation models were quantitative in nature (either binary, ordered categorical, or continuous). Minimum and maximum values were specified to ensure that plausible values were imputed for all categorical variables. Twenty imputations were run based on recommendations by Graham and colleagues (2007).

Results

Assessing the Buffering Effects of Parental Warmth

The results of the final reduced binary logistic regression model testing the parental warmth hypotheses (Hypotheses 1 and 2) are presented in Table 2.1. Counter to expectations (Hypothesis 2), the two-way interaction of frequency x parental warmth was not retained in the final model, indicating that parental warmth did not disproportionately benefit those who experienced greater severity of victimization. The frequency x sex interaction was also eliminated from the model, indicating that there was no variation by sex on the influence between frequency of victimization and reactivity profile membership. The significant interaction between parental warmth and victim's sex was retained in the final reduced logistic regression model (OR=.90; 95% CI: .83, .98). This interaction was probed by plotting regression

lines for boys and girls.

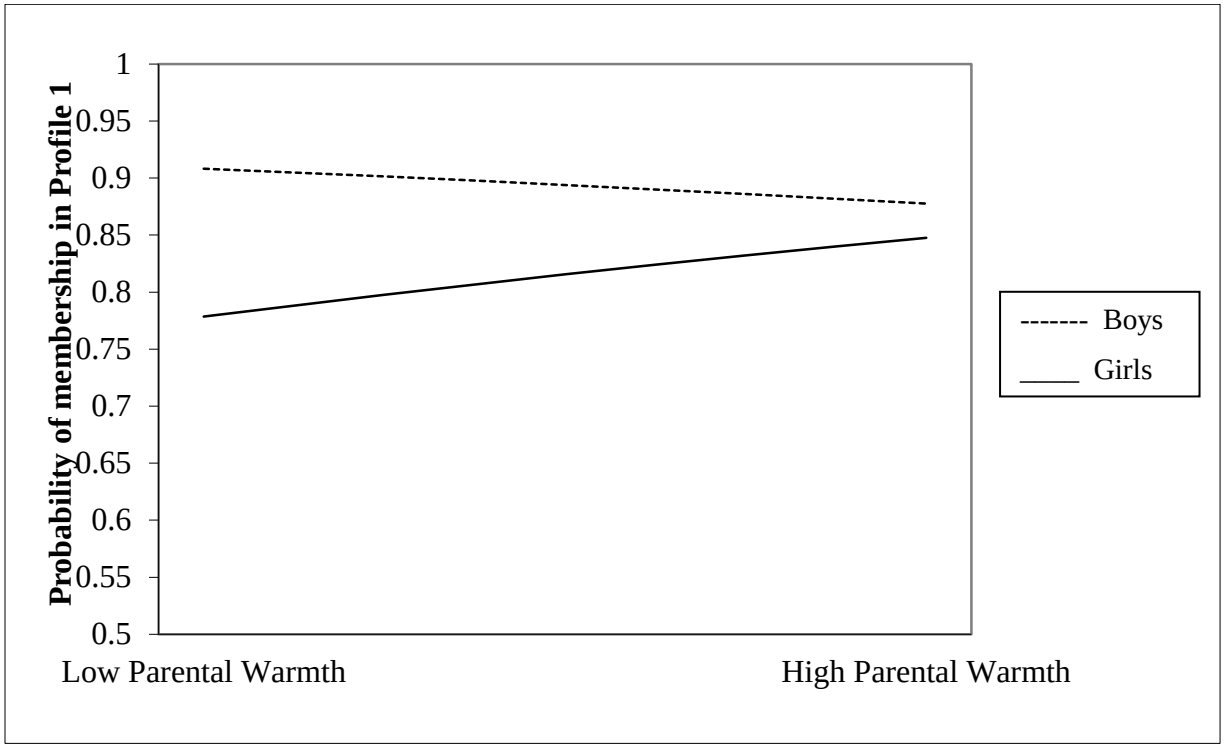
Table 2.1. Results of final binary logistic regression model testing the buffering effect of parental warmth hypotheses (Hypotheses 1 and 2)

Variable	OR	95% CI
Frequency	.98	(.96, 1.00)
Parental warmth	**1.06**	**(1.00, 1.13)***
Sex (M vs. F)	**8.61**	**(2.28, 32.50)*****
Parental warmth x sex	**.90**	**(.83, .98)****
Parental warmth, boys	.96	(.90, 1.01)
Parental warmth, girls	**1.06**	**(1.00, 1.13)***
Family conflict	**.84**	**(.79, .88)******
Race (white vs nonwhite)	1.16	(.87,1.56)
Parental education	.98	(.89,1.09)
Grade	**.78**	**(.66, .92)*****

Note: 0 (reference category)= Profile 2, High internalizers, moderate externalizers; Profile 3, High internalizers, high externalizers; Profile 4, Moderately anxious, moderate externalizers; and Profile 5, Moderate internalizers, moderate externalizers. 1=Profile 1, Non-reactors.
*p<.10
**p<.05
***p<.01
****p<.001

As shown in Figure 2.1, girls benefitted disproportionately from family warmth. Boys' likelihood of membership in the non-reactor profile was high compared to girls in both low- and high-parental warmth environments; however, the sex differences attenuated in environments with high parental warmth. Increased parental warmth increased girls' likelihood of being in the non-reactor profile as compared to the other reactivity profiles with marginal significance (p=07). This marginal buffering effect of parental warmth held for girls only.

Figure 2.1. Plot of interactive effect of parental warmth and sex



In the final binary logistic regression model we also found that family conflict had an independent harmful effect on reactivity profile membership, with each one-unit increase in conflict being associated with a 16% decreased likelihood of membership in the non-reactor profile as compared to Profiles 2-5 (OR=. 84; 95% CI: .79, .88). Grade was also significant, with each yearly increase in grade associated with a 22% decreased likelihood of membership in the non-reactor profile as compared to Profiles 2-5 (OR=.78; 95% CI: .66, .92).

To further examine the relationship of parental warmth and reactivity profile membership for girls, we conducted multinomial logistic regression on a girls-only restricted sample (n=715) with the non-reactor profile as the reference category. Results (shown in Table 2.3) indicated that parental warmth was significantly associated with decreased likelihood of membership in Profile 2 (high internalizers, moderate externalizers; OR=.89, 95% CI: .82, .96) and in Profile 3 (high internalizers, high externalizers; OR=.91, 95% CI: .84, .99) compared to the non-reactor profile.

Though not the focus of these analyses, family conflict exerted an independent effect on the likelihood of membership in each of the other reactivity profiles compared to the non-reactor profile.

Table 2.2. Results of multinomial logistic regression testing the buffering effect of parental warmth for girls (n=715)

	Profile 2 vs. Profile 1		Profile 3 vs. Profile 1		Profile 4 vs. Profile 1		Profile 5 vs. Profile 1	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Frequency	1.04	(1.00, 1.09)	**1.07**	**(1.03, 1.12)****	1.03	(.99, 1.07)	1.00	(.96, 1.05)
Parental warmth	**.89**	**(.82, .96)****	**.91**	**(.84, .99)***	.95	(.88, 1.02)	.95	(.88, 1.03)
Family conflict	**1.27**	**(1.16, 1.40)*****	**1.36**	**(1.23, 1.51)*****	**1.14**	**(1.04,1.25)****	**1.25**	**(1.14, 1.36)*****
Race	.67	(.39, 1.15)	.60	(.32, 1.09)	.87	(.55, 1.39)	.90	(.55, 1.50)
Parental education	1.20	(1.00, 1.45)	1.15	(.93, 1.42)	1.11	(.95, 1.30)	1.13	(.95, 1.35)
Grade	1.33	(.98, 1.83)	1.27	(.89, 1.79)	1.05	(.80, 1.39)	1.26	(.94, 1.69)

Note: Profile 1= Non-reactors (reference category); Profile 2= High internalizers, moderate externalizers; Profile 3=High internalizers, high externalizers; Profile 4= Moderately anxious, moderate externalizers; Profile 5=Moderate internalizers, moderate externalizers.
*p<.05
**p<.01
***p<.001

Taken together, the results of the binary and multinomial logistic regressions support a marginal stress-buffering effect of parental warmth (Hypothesis 1) for girls; specifically, it was protective against membership in the two high internalizing profiles (Profile 2 and Profile 3). Counter to our hypothesis (Hypothesis 2), however, more frequent victims of bullying did not disproportionately benefit from the stress-buffering effects of parental warmth.

Assessing the Exacerbating Effects of Family Conflict

The results of the final reduced binary logistic regression model testing the family conflict Hypotheses (Hypotheses 3 and 4) are presented in Table 2.3. Counter to expectations (Hypothesis 4), the two-way interaction of frequency x family conflict was not retained in the final model, indicating that family conflict did not disproportionately harm those who experienced greater severity of victimization. The interactions containing sex were also eliminated from the model, indicating that the relationships between frequency of victimization and family conflict and reactivity profile membership did not vary by sex of the victim. Frequency of victimization, however, was associated with increased likelihood of membership in the high internalizers, high externalizers profile (Profile 3) as compared to all others (OR=1.06; 95% CI: 1.03, 1.09). Family conflict was even more strongly associated with reactivity profile membership, with each one-unit increase in family conflict equivalent to a 15% increase in odds of membership in Profile 3 (high internalizing, high externalizing) compared to all other profiles (OR=1.15, 95% CI: 1.09, 1.21).

Table 2.3. Results of final binary logistic regression model testing the exacerbating effect of family conflict hypotheses (Hypotheses 3 and 4)

Variable	OR	95% CI
Frequency	**1.06**	**(1.03, 1.09)***
Family conflict	**1.15**	**(1.09, 1.21)***
Sex (male vs female)	.74	(.50, 1.09)
Parental warmth	.98	(.94, 1.03)
Race	1.02	(.69, 1.52)
Parental education	1.07	(.93, 1.23)
Grade	1.15	(.93, 1.45)

Note: 0 (reference category)=Profile 1, Non-reactors; Profile 2, High internalizers, moderate externalizers; Profile 4, Moderately anxious, moderate externalizers; and Profile 5, Moderate internalizers, moderate externalizers. 1=Profile 3, High internalizers, high externalizers.
*p<.001

To further examine the relationship of frequency of victimization and family conflict and reactivity profile membership we conducted multinomial logistic regression with the high internalizing, high externalizing profile (Profile 3) as the reference category. Results presented in Table 2.4 show that frequency of victimization was independently associated with reactivity profile membership for all reactivity profiles compared to Profile 3 (high internalizers, high externalizers). Each one-unit increase in frequency of bullying victimization was associated with a 6% decrease in likelihood of membership in Profile 1 (non-reactors) compared to Profile 3 (OR=94; 95% CI: 91, 97), a 4% decreased likelihood of membership in Profile 2 (high internalizers, moderate externalizers) compared to Profile 3 (OR=96; 95% CI: 93, 99), a 6% decreased likelihood of membership in Profile 4 (moderately anxious, moderate externalizers) compared to Profile 3 (OR=.94, 95% CI=.92, .97), and a 6% decreased likelihood of membership in Profile 5 (moderate internalizers, moderate externalizers) compared to Profile 3 (OR=.94, 95% CI: .92, .97). Increased family conflict was associated with reduced likelihood of membership in the non-reactor profile (Profile 1) compared to the high internalizers, high externalizers profile (OR=.78, 95% CI: .73, .84). Family conflict was also associated with decreased likelihood of membership in the two profiles characterized by moderate levels of internalizing--Profile 4

(moderately anxious, moderate externalizing; OR=.84, 95% CI: .79, .89) and Profile 5 (moderate internalizers, moderate externalizers; OR=.92, 95% CI: .87, .97)—compared to Profile 3 (high internalizers, high externalizers).

Table 2.4. Results of multinomial logistic regression testing the exacerbating effect of family conflict

	Profile 1 vs. Profile 3		Profile 2 vs. Profile 3		Profile 4 vs. Profile 3		Profile 5 vs. Profile 3	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
Frequency	**.94**	**(.91, .97)*** **	**.96**	**(.93, .99)***	**.94**	**(.91, .97)*** **	**.94**	**(.92, .97)*** **
Family conflict	**.78**	**(.73, .84)*** **	.95	(.89, 1.02)	**.84**	**(.79, .89)*** **	**.92**	**(.87, .97)****
Sex (male vs female)	**2.17**	**(1.39, 3.39)*** **	.73	(.44, 1.20)	1.14	(.73, 1.77)	**1.58**	**(1.01, 2.45)***
Parental warmth	1.03	(.97, 1.09)	.99	(.94, 1.04)	1.02	(.97, 1.08)	1.03	(.98, 1.09)
Race (white vs nonwhite)	1.11	(.70, 1.76)	.73	(.44, 1.19)	.96	(.61, 1.51)	1.10	(.70, 1.72)
Parental education	.93	(.79, 1.09)	.93	(.79, 1.10)	.95	(.81, 1.11)	.93	(.79, 1.08)
Grade	**.71**	**(.55, .92)***	.96	(.73, 1.27)	.84	(.65, 1.09)	.96	(.74, 1.24)

Note: Profile 1= Non-reactors; Profile 2= High internalizers, moderate externalizers; Profile 3=High internalizers, high externalizers (reference category); Profile 4= Moderately anxious, moderate externalizers; Profile 5=Moderate internalizers, moderate externalizers

*p<.01

**p<.001

Taken together, the results of the binary and multinomial logistic regressions support an exacerbating effect of family conflict (Hypothesis 3); specifically, family conflict increased likelihood of membership in Profile 3 (high internalizers, high externalizers) compared to non-reactors (Profile 1) and the two profiles characterized by moderate levels of internalizing reactivity (Profiles 4 and 5). We found no evidence that these associations varied by sex. Counter to hypothesis 4, more frequent victims of bullying were not disproportionately harmed by the exacerbating effects of family conflict.

Discussion

This study sought to test the stress-buffering effects of family warmth and exacerbating effects of family conflict on reactivity to bullying victimization and whether these buffering and exacerbating effects moderated the relationship between degree of the stressor experienced (i.e., frequency of bullying victimization) and membership in profiles of internalizing and externalizing reactivity to bullying victimization. Several findings of note emerged from the binary and multinomial logistic regressions testing these hypotheses.

Our finding that parental warmth was marginally protective against reactivity to bullying victimization for girls, but not boys, is consistent with findings reported by Davidson and Demary (2007), who found that social support was protective against internalizing symptoms, specifically, for girls, but not boys. Not only did we find the buffering effect of parental warmth to be marginally significant for girls only in the binary logistic regression model, but we found that this buffering effect was limited to reducing membership in the two high internalizing profiles (Profile 2, high internalizers, moderate externalizers and Profile 3, high internalizers, high externalizers), in particular, using multinomial logistic regression. That the buffering effect did not extend to boys in this study is also consistent with findings of Davidson and Demaray

(2007). Eastman et al. (unpublished) found that boys were more likely than girls to be members of the non-reactor profile than each of the other reactivity profiles. It is, therefore, possible that boys do not feel the need to seek out nor do they elicit parental warmth in the wake of bullying victimization as do girls, as they are less likely to experience internalizing and externalizing sequelae to begin with.

We did not find support for Hypothesis 2, that parental warmth interacts with frequency of victimization to influence reactivity profile membership. Girls who were victims of bullying benefitted from the stress-buffering effects of parental warmth regardless of the extent of their victimization. This suggests that parental warmth has the potential to buffer the negative effects of both mild and more severe experiences of bullying for girls.

Our findings regarding family conflict are consistent with Bandura's Social Learning Theory of aggression. Using binary logistic regression, we found that increased family conflict was associated with membership in the profile characterized by high externalizing reactivity (Profile 3, high internalizers, high externalizers) compared to all others. This suggests that victims of bullying may model externalizing responses that they witness or experience directly in the home. Additionally, as noted by Bettencourt and Farrell (2013), violence at home may serve as implicit communication from parents about the acceptability of responding to conflict with violence, thus leading bullied adolescents to exhibit externalizing reactivity. In contrast to parental warmth, we did not find that the influence of family conflict on reactivity profile membership varied by victim's sex. This is in contrast to the findings of Yang & McLoyd (2015), who reported that family conflict increased antisocial outcomes among girls but not boys who experienced peer victimization. Our findings suggest that the social learning processes that confer risks for reactivity to bullying victimization may be the same for girls and boys.

Multinomial logistic regression showed that family conflict distinguished membership in Profile 1 (non-reactors) from membership in Profile 3 (high internalizers, high externalizers), Profile 4 (moderately anxious, moderate externalizers) from Profile 3, and Profile 5 (moderate internalizers, moderate externalizers) from Profile 3. Family conflict did not distinguish membership in Profile 2 (high internalizers, moderate externalizers) from likelihood of membership in Profile 3 (high internalizers, high externalizers). This may suggest that family conflict has the potential to heighten both internalizing *and* externalizing reactivity.

Additionally, counter to Hypothesis 4 we did not find a moderating effect of family conflict on the relationship between frequency of victimization and membership in profiles of internalizing and externalizing reactivity. This indicates that victims who experience even a single incident of bullying (the criterion for inclusion in this study) will be harmed by the reactivity exacerbating effects of family conflict.

We note that there was no main effect of frequency in the binary logistic regression model testing the buffering effect of parental warmth, though it was found to increase likelihood of membership in Profile 3 (high internalizers, high externalizers) in comparison to Profile 1 (non-reactors) in the multinomial logistic regression model. In the multinomial family conflict model, frequency of victimization increased the likelihood of membership in Profile 3 (high internalizers, high externalizers) in comparison to all others. This suggests that frequency of victimization, independent of family characteristics, puts victims of bullying at greater risk for high internalizing and high externalizing reactivity, in comparison to more moderate or lower levels of reactivity.

This study has several strengths. Specifically, we used previously validated measures of parental warmth and family conflict with high internal consistency reliability. Additionally, our

large sample size (1,196 adolescent victims) increased our confidence of detecting associations between family characteristics and reactivity profile membership. Few studies have examined the role of parental warmth and family conflict in the same model (see Yang & McLoyd, 2015 as an exception). By controlling for parental warmth in our models testing associations between family conflict and reactivity profile membership and vice versa, we were able to examine the independent contribution of each of these family characteristics on internalizing and externalizing reactivity to bullying victimization.

Additionally, the use of profiles of reactivity in our analysis represents a person-centered approach, which allowed for examination of the role of family characteristics on complex typologies of reactivity to bullying victimization, rather than looking at the role of family characteristics on a single outcome of victimization sequelae. LPA by Eastman et al. (unpublished) and by Kretschmer et al. (2015) identified substantial comorbidity of internalizing and externalizing among victims of bullying. The analysis presented here preserves this reality of reactivity to bullying victimization.

Despite these strengths, a few limitations must be noted. Specifically, all of our measures were self-reported, so we cannot assess the level of method variance. For example, it is possible that an internalizing adolescent may perceive lower levels of family support than are actually available (Connors-Burrow et al., 2009). Additionally, we must acknowledge the cross-sectional nature of the study. We cannot, therefore, assert that exposure to bullying occurred before reports of internalizing and externalizing symptoms; our use of the term "reactivity" to describe these symptoms must be considered with this caveat. Limitations of the LPA used in our tests of statistical significance have been noted elsewhere (see Eastman et al., unpublished), and the present study must be considered in light of these limitations, as well.

Our results underscore the importance of the family environment in shaping adolescent responses to bullying victimization. Secondary interventions that aim to leverage warmth and reduce conflict within the family may reduce the negative sequelae of bullying victimization among adolescents. Inclusion of family members in secondary interventions may be more effective in reducing internalizing and externalizing reactivity than interventions that focus exclusively on the victim of bullying.

CHAPTER 5: ASSOCIATIONS BETWEEN 5-HTTLPR, BDNF, AND MAOA GENOTYPES AND LATENT PROFILES OF INTERNALIZING AND EXTERNALIZING AMONG ADOLESCENT VICTIMS OF BULLYING (MANUSCRIPT #3)

Introduction

In 2013, approximately 22% of students in grades 6-12 experienced bullying during the school year (National Center for Education Statistics, 2015) and it is estimated that between 20-40% of children experience bullying at least once during their school careers (Shetgiri, Lin & Flores, 2013). Bullying victimization in adolescence has been associated with a variety of negative internalizing (i.e., harmful to self) and externalizing (i.e., harmful to others) sequelae, and, furthermore, evidence suggests that victims of bullying are at heightened risk for psychological maladjustment in adulthood (Arseneault, Bowes, & Shakoor, 2010; Copeland, Wolke, Angold, & Costello, 2013; Hawker & Boulton, 2000; Menard, 2002). Studies suggest that biological factors, such as one's genotype, may influence reactivity to bullying (Beaver, Mancini, DeLisi, & Vaughn, 2011). Several genes, including *5-HTTLPR*, *BDNF*, and *MAOA*, have been found in animal and human studies to influence sensitivity to peer rejection and peer exclusion through their respective relationships with the serotonergic system, hippocampal function, and amygdala function. This study examines whether the genotypes for *5-HTTLPR*, *BDNF*, and *MAOA*, which have been found to be associated with sensitivity to social stress, are associated with reactivity to being bullied among adolescents.

Not all adolescent victims of bullying respond to the victimization in the same way. Some adolescent victims of bullying demonstrate internalizing sequelae such as heightened

social isolation, depression, and anxiety (Forero, McLellan, Rissel, & Bauman, 1999; Hawker & Boulton, 2000; Kaltiala-Heino, Rimpelä, Rantanen, & Rimpelä, 2000; Karatzias, Power, & Swanson, 2002; Nansel et al., 2001; Veenstra et al., 2005; Wolke, Woods, Bloomfield, & Karstadt, 2001). Others demonstrate externalizing sequelae, which may include violent behavior towards others, carrying a weapon (Arseneault, Walsh, Trzesniewski, Newcombe, Caspi, Moffit, 2006; Kim, Leventhal, Koh, Hubbard, & Boyce, 2006; Liang, Flisher, & Lombard, 2007; Nansel, Overpeck, Haynie, Ruan, & Scheidt, 2003), and becoming a perpetrator of bullying behaviors (Barker, Arseneault, Brendgan, Fontain, & Maughan, 2008). Still other victims may demonstrate both internalizing and externalizing reactivity (Arseneault, Bowes, & Shakoor, 2010; Hampel, Manhal, & Hayer, 2009; Hemphill et al, 2011). This variation in response to bullying victimization led researchers to develop profiles of bullying victimization along internalizing and externalizing dimensions (Eastman, et al., unpublished; Kretschmer, Barker, Dijkstra, Oldehinkel,& Veenstra, 2015). In one such study, Eastman (unpublished) used latent profile analysis to identify profiles of reactivity to bullying victimization in a sample of 1,196 North Carolina adolescents in grades 8-10 who had been a victim of bullying. The profiles identified were non-reactors (Profile 1); high internalizers, moderate externalizers (Profile 2); high internalizers, high externalizers (Profile 3); moderately anxious, moderate externalizers (Profile 4); and moderate internalizers, moderate externalizers (Profile 5).

The development of profiles of reactivity to bullying victimization represents a person-centered approach to understanding the sequelae of bullying victimization; that is, individuals have been grouped in categories based on their similarity with one another and differences from those in other categories (Muthén & Muthén, 2000; Laursen & Hoff, 2006). In contrast to single-outcome, variable-centered studies, this person-center approach enables the unmasking of any

underlying heterogeneity in reactivity to bullying victimization and allows for the possibility of complex reactions that incorporate *both* internalizing and externalizing elements. Indeed, the work of Eastman et al. (unpublished) and Kretschmer et al., 2015 has identified substantial co-morbidity of internalizing and externalizing among victims of bullying, which may be missed in studies that examine the effects of bullying victimization on a single outcome variable.

Examination of the influence of genotypes associated with reactivity to social stressors has not, to-date, been incorporated into person-centered approaches to understand the factors that may influence membership in high-risk profiles of internalizing and externalizing reactivity to bullying victimization. Rather than replicate previous variable-centered candidate gene analyses on the relationship between genotype and reactivity to social stressors based on a single outcome, the person-centered approach employed here enables for examination of the influence of genotype on more complex typologies of reactivity to bullying victimization. The purpose of present study is to quantify the association of genotype for 5-*HTTLPR*, *BDNF*, and *MAOA* on likelihood of membership in the five previously identified profiles of reactivity to bullying victimization among adolescents (Eastman et al., unpublished). We chose to examine the effects of *5-HTTLPR*, *BDNF*, and *MAOA* separately rather than attempt an aggregate risk scale because evidence suggests different biological mechanisms for sensitivity to social stressors for each and our hypotheses propose differing effects (suggesting different comparison groups) for each one, as described below.

5-HTTLPR

5-HTTLPR (rs25531) is an insertion-deletion polymorphism of the serotonin transporter gene (*SLC6A4*). Research has most commonly focused on two variations: a long allele (16 copies of a 20-23 bp repeat) and a short allele (14 copies of the bp repeat), with the short allele being

associated with reduced transcriptional efficiency (Heils et al., 1996). The function of the serotonin transporter is to move serotonin (5-HT) from the synapse so that it is returned to the presynaptic neuron for later re-release or degradation (Karg et al., 2011). This reuptake is the body's primary mechanism for clearing extracellular 5-HT and, therefore, the 5-HT transporter plays a critical role in the duration and intensity of 5-HT communication with its receptors (Hariri & Holmes, 2006). Areas of the brain that are implicated in emotional response, including the amygdala, are heavily innervated with 5-HT neurons and, therefore, are regulated in part by the serotonin transporter (Hariri & Holmes, 2006). In rodent models, carriers of the short (versus long) allele of the rodent version (i.e., orthologue) of *5-HTTLPR* called *sert* have demonstrated greater hypothalamic-pituitary-adrenal (HPA) reactivity in response to physical and psychological stress (Karg et al., 2011). Human carriers of the short allele of *5-HTTLPR* have also shown heightened cortisol levels following a psychosocial stress (Way & Taylor, 2010) and increased activity in the amygdala in response to a range of environmental stimuli (Munafò, Brown SM, and Hariri, 2008).

Karg et al. (2011) conducted a meta-analysis of candidate gene studies that examine interactions between stressful life events and *5-HTTLPR*. Stressful life events considered in the meta-analysis included child maltreatment and trauma, physical illness, and stressful life events and traumas in adulthood (Karg et al., 2011). Across studies considered, the short allele was associated with greater reactivity to stress (measured as anxiety and depression) (Karg et al., 2011). Of specific relevance to the experience of bullying, the short allele of *5-HTTLPR* has been found to decrease resilience (measured by the absence of emotional problems such as withdrawal and anxiety and depression symptoms) among bullying victims in childhood (Sugden et al., 2010) and increase depressive symptoms in adolescent girls who were victims of relational

bullying (i.e., bullying that intends to harm social relationships) (Benjet, Thompson, & Gotlib, 2010).

Based on the function of *5-HTTLPR* and candidate gene studies noted above, in the present study, we hypothesized that adolescents with more copies of the short allele of *5-HTTLPR* would have higher likelihood of membership in the profiles characterized by high internalizing (Profile 2, high internalizing, moderate externalizing and Profile 3, high internalizing, high externalizing) than the other reactivity profiles.

BDNF

Brain-derived neurotropic factor (BDNF) is a protein involved in hippocampal long-term potentiation (that is, the long-lasting strengthening of synaptic activity between neurons based on recent activity patterns) and, therefore, plays an important role in learning and memory (Egan et al., 2003). The Val66Met polymorphism (rs6265) of the brain-derived neurotropic factor (*BDNF*) gene causes a valine to methionine substitution at codon 66. This polymorphism has been shown to affect secretion and transport of the protein BDNF, with met allele carriers demonstrating poorer hippocampal function and impaired trafficking and secretion than Val allele carriers (Egan et al., 2003). Rodent models and human neuroimaging studies suggest that *BDNF* Val66Met influences reactions to stressful experiences.

In their study of a mouse model, Berton and colleagues (2006) showed that mice experiencing repeated aggression developed a long-lasting aversion to social contact. Using a local knockdown of *bdnf* in the ventral tegmental area, an area of high expression of BDNF (which, along with the nucleus accumbens comprises the mesolimbic dopamine pathway), they showed that BDNF was required for development of this social aversion. This suggests that BDNF plays an important role in neural and behavioral responses to social experience (Berton et

al., 2006). Patki and colleagues (2013) showed that rats experiencing social defeat showed decreased levels of BDNF in the hippocampus along with memory impairment and anxiety-and depression-like behaviors. Taken together, these studies suggest that individuals with the Met allele may have reduced baseline BDNF expression, which is further attenuated in the presence of a social stressor, leading to social withdrawal and anxiety-and depression-like behaviors.

Findings of the rodent models are supported by studies of human subjects and the interaction of stressors with *BDNF* genotype. In their systematic review and meta-analysis, Hosang and colleagues (2014) reported that the Val66Met polymorphism significantly moderated the relationship between life stress and depression (with Met carriers being more susceptible to depression following life stressors than Val carriers), but that this interactive effect held only when publications examining stressful life events (rather than childhood adversity measures) were considered. In contrast, Gottfredson and colleagues (2015) found that the Met allele was protective against internalizing reactivity in response to victimization. On the whole, however, the rodent and human studies suggest that individuals carrying the Met allele of *BDNF* are particularly susceptible to the effects of recent stressors, and that this vulnerability may manifest as depression.

Based on the function of BDNF and the candidate gene studies noted above, we hypothesized that *BDNF* genotype would be associated with reactivity profile membership such that adolescents with more copies of the Met allele of the Val66Met polymorphism would be more likely to be in profiles characterized by high internalizing (Profile 2, high internalizing, moderate externalizing and Profile 3, high internalizing, high externalizing) than the other reactivity profiles.

MAOA

Monoamine oxidase-A (MAO-A), is a mitochondrial enzyme located in the mitochondrial membrane in the presynaptic terminal of monoamine projection neurons and astrocytes that degrades serotonin, dopamine, and norepinephrine (Buckholtz & Meyer-Lindenberg, 2008). It helps to regulate release of serotonin and norepinephrine, specifically, by regulating the availability of monoamines for sequestration in vesicles and by breaking down the monoamines following release (Buckholtz & Meyer-Lindenberg, 2008). The "MAOA gene" is the gene that codes for this enzyme. A 30 bp variable number tandem repeat (VNTR) polymorphism in the promoter region of this gene has been found to be associated with enzyme expression, with 2, 3 or 5 repeats associated with lower MAO-A expression than 3.5 or 4 repeats (Sabol, Hu, and Hamer, 1998). Additionally, in behavioral studies, individuals with a low-expression (*MAOA*-L) allele (i.e, either 2, 3, or 5 repeats) have been found to be more prone to reactivity than individuals with a high-expression (*MAOA*-H) allele (i.e., 3.5 or 4 repeats) (Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993; Caspi et al., 2002). *MAOA* is an x-linked gene; therefore, men are hemizygous and are either *MAOA*-L or *MAOA*-H, whereas women may have no *MAOA*-L alleles, one *MAOA*-L alleles, or two *MAOA*-L alleles.

In a study of 97 healthy individuals, Meyer-Lindenberg and colleagues (2006) found structural and functional differences between the brains of *MAOA*-L individuals and *MAOA*-H individuals. Specifically, compared to those with the *MAOA*-H genotype, those with the *MAOA*-L genotype showed structural reductions in the cingulate gyrus (involved in emotion formation and processing, learning, and memory), the amygdala (involved in memory, decision-making, and emotional reactions), and the hippocampus (involved in memory), resulting in an 8% decrease in grey matter (Meyer-Lindenberg et al., 2006). Functionally, they noted among those

with *MAOA*-L genotype exaggerated activation of the amygdala and insula (involved in emotional processing) and diminished activation of the pre-frontal cortex (PFC) (associated with executive function) when confronted with fearful and angry faces; greater activation of the amygdala and hippocampus during recall of negative visual scenes and; reduced activation of the dorsal cingulate during a go/no go task[1] (Meyer-Lindenberg et al., 2006). This suggests exaggerated emotional response and reduced executive regulation of the emotional response. They also noted aberrant coupling between the ventromedial prefrontal cortex (vmPFC) and the amygdala among *MAOA*-L individuals, suggesting compensatory support from the vmPFC in attempt to regulate the hyperreactive amygdala (Meyer-Lindenberg et al., 2006). In other words, the vmPFC is recruited to assist with emotion regulation to compensate for reduced executive control. Among *MAOA*-L males, this aberrant coupling between the vmPFC and the amygdala was correlated with increased harm avoidance and decreased reward dependence as measured by the TPQ[2] and angry hostility as measured by the NEO-PI[3] (Meyer-Lindenberg et al., 2006). Taken together, these findings suggest a trait profile for those with the *MAOA*-L genotype characterized by enhanced reactivity to threat, reduced sensitivity to cues for prosocial behavior, and increased tendency towards anger, frustration, and bitterness (Meyer-Lindenberg et al., 2006).

To understand the psychological and neurological mechanisms underlying *MAOA*-mediated aggression, Eisenberger and colleagues (2007) conducted a study that simulated social

[1] Go/no go tasks are designed to test a participant's ability for impulse control and sustained attention. During the task, stimuli are presented in a continuous stream and participants make a binary decision (go or no-go) on each one. (http://www.cognitiveatlas.org/task/go/no-go_task)

[2] The Tridimensional Personality Test (Cloninger, 1987) is a personality test designed to discriminate between 3 personality dimensions which correspond to monoaminergic activity. The three dimensions are novelty-seeking (low dopaminergic activity, harm avoidance (high serotonergic activity), and reward dependence (low noradrenergic activity).

[3] The Neuroticism-Extraversion-Openness Personality Inventory (NEO-PI) (Costa & McCrae, 1985) is a personality test designed to measure facets of the "Big 5"personality dimensions: Neuroticism, Extraversion, Openess, Agreeableness, and Conscientiousness.

exclusion (via a game of "cyberball") while comparing brain activity to participants' self-reported social distress. fMRI data showed that areas of the amygdala and dorsal anterior cingulate cortex that corresponded to self-reported social exclusion-related distress were disproportionately active in test subjects who were *MAOA*-L (Eisenberger et al., 2007). This would suggest that *MAOA*-L carriers are particularly sensitive to social exclusion.

Noting that brain regions associated with social rejection, those influenced by MAOA activity, and those that are continuing to develop during adolescence overlap, Sebastian and colleagues (2010), sought to determine whether the neurological response to social rejection in adolescents is immature when compared to adults, and whether this response is influenced by *MAOA* genotype. Results of fMRI during an emotional Stroop test[4] showed that adolescents, when compared to adults, had lower activity in the ventrolateral prefrontal cortex (VLPFC), a region of the brain involved in affect regulation and inhibitory control (Sebastian et al., 2010). *MAOA*-L adults showed heightened amygdala response during the Stroop task, whereas *MAOA*-L adolescents showed a decreased amygdala response, but this response was less regulated by the VLPFC. Sebastian and colleagues concluded that the effect of *MAOA*-L on brain circuitry may be age-dependent and, specifically, may influence connections between the PFC and amygdala (Sebastian et al., 2010). Taken together, these studies indicate that adolescence is a sensitive period in responding to social exclusion, and further, that response to social exclusion may be influenced by *MAOA* genotype. Coupled with the aforementioned Meyer-Lindenberg et al. (2006) evidence suggests that *MAOA*-L individuals may be more likely than *MAOA*-H individuals to respond to such social exclusion with feelings of hostility.

[4] The Stroop test, named after the Stroop Effect, is designed to assess selective attention, cognitive processing and flexibility, and executive functioning.

Based on MAO-A function, neuroimaging studies, the candidate gene studies noted above, we hypothesized that *MAOA* genotype would be associated with reactivity profile membership such that adolescents with more copies of the low-activity allele will be more likely to be in profiles characterized by high externalizing (Profile 3, high internalizing, high externalizing) as compared to the other reactivity profiles.

Method

Data for the present study came from the *Genes in Context Study* (NICHD R01-HD057222; PI Vangie A. Foshee). For the *Genes in Context Study,* biospecimens were collected for genotyping from young adults (ages 19–25) who had participated as adolescents in a multi-wave longitudinal study. For the longitudinal study, seven waves of survey data were collected from adolescents on intrapersonal and contextual factors (family, peers, school, and neighborhood) that influence adolescent risk behaviors. The survey data were collected from adolescents, during school, from two county-wide school systems in North Carolina beginning when participants were in the spring semester of the 6^{th}, 7^{th}, and 8^{th} grades and ending when participants were in the fall semester of the 10^{th}, 11^{th}, and 12^{th} grades. All students in grades 6, 7, and 8 who were able to complete the survey in English, who were not in special education programs, and who were not out of school due to a long-term suspension or expulsion were eligible for the longitudinal study at wave one. Parents had the opportunity to refuse consent for their child's participation by returning a written form or by calling a telephone number. Trained data collectors administered the questionnaires in classrooms. Assent was obtained from adolescents immediately prior to the survey administration from students whose parents had consented. The Institutional Review Board for the University of North Carolina at Chapel Hill approved the study. Response rates were high, ranging from 89% at wave 1 to 73% at wave 7.

In 2010, young adults who participated in at least one wave of data collection for the longitudinal study (n=3,835) were contacted by telephone to provide either a saliva sample (via Oragene collection kit) or a blood spot (using a lancet) and return the sample to the study office by mail. Respondents were given $35 for a saliva sample and $50 for a blood spot. A total of 1,064 wave 4 participants (40.4%) provided biospecimens for genotyping.

Analytic Sample

Questions about bullying victimization were added to the survey at wave 4 (Fall 2003), when students were in the 8th, 9th, and 10th grades. At the time of the questionnaire administration, data collectors provided each student with a Student Directory that alphabetically listed enrolled students along with a unique four-digit peer identification number for each student. Students were asked to identify up to five peers *who had been mean to them or who had picked on them in the past 3 months* (i.e., bullied them).

Although waves 4-7 of the longitudinal study included these questions about bullying victimization, wave 4 (Fall 2003, when students were in the 8th, 9th, and 10th grades) was selected as the basis for the analytic sample because the greatest number of students reported being a victim of bullying during this wave, thereby maximizing sample size for the present study. A total of 2,636 adolescents completed the questionnaire (76.9% of those eligible) at wave 4, of which 1064 (40.4%) later provided a biospecimen. 281 of these of these individuals had reported at wave 4 that they had been a victim of bullying. To eliminate potential confounding by population stratification, analyses were conducted on the subset of these individuals who reported their race as being White (n=132).

Genotyping

DNA was extracted from samples at the University of North Carolina Biospecimen Processing Facility and subsequently genotyped at the Institute of Behavioral Genetics at the University of Colorado, Boulder. Genotyping of *5-HTTLPR* was performed as described in Whisman, Richardson, and Smolen (2011). As recommended by Hu et al., (2006) the S and L_G alleles were coded as "short" and the L_A was coded as "long" to denote activity levels. The SNP in *BDNF* was assayed using the Applied Biosystems (Foster City, CA) Open Array1 System (a low volume Taqman1 method) as described in Surtees et al., 2007 using the allelic discrimination mode (Livak, 1999). The 30bp polymorphism of *MAOA* was assessed as described in Haberstick, Schmitz, Young, & Hewitt (2005).

Measures

Independent Variables

5-HTTLPR. *5-HTTLPR* was coded according to the strategy of Caspi et al. (2003). Individuals homozygous for short alleles were assigned a value of 2, those with one short and one long allele were assigned a 1, and those homozygous for long alleles were assigned a value of 0. Twenty percent of individuals had two short alleles, 46% had one short and one long allele, and 33% had two long alleles. This distribution was consistent with Hardy-Weinberg equilibrium (p=.49).

***BDNF*.** Individuals homozygous for the Met allele were assigned a value of 2, those with one Val and one Met allele were assigned a 1, and those homozygous for the Val allele were assigned a value of 0. Four percent of individuals were homozygous for the Met allele, 38% had one Val and one Met allele, and 58% were homozygous for the Val allele. This distribution was consistent with Hardy-Weinberg equilibrium (p=.45).

MAOA. *MAOA* was coded such that girls homozygous for low-expression alleles (i.e., either 2,3, or 5 repeats) were assigned a value of 2, those with one low-expression allele and one high-expression allele (i.e, either 3.5 or 4 repeats) were assigned a value of 1, and those homozygous for high-expression alleles were assigned a 0. Since *MAOA* is an x-linked gene, boys are hemizygous and were coded such that those with low-expression alleles were assigned a value of 0 and those with high-expression alleles were assigned a value of 2 (the equivalent of homozygosity for the low-expression allele among girls). Of the girls in the sample, 19% were homozygous for the low-expression alleles, 55% had one low- and one high-expression allele, and 26% were homozygous for the high-expression alleles. Sixty one percent of boys had a low-expression allele and 39% had a high-expression allele.

Dependent Variable

Reactivity to Bullying Victimization Profile. As noted earlier, previous research using latent profile analysis (LPA) identified five profiles of internalizing and externalizing reactivity to bullying victimization (Eastman et al., unpublished). Eighteen items were used in those analyses to develop two indicators of internalizing (anxiety and depression) and two indicators of externalizing (delinquency and violence against peers). Items for the two internalizing indicators were four Likert-type scale items measuring symptoms of anxiety (Reynolds & Richmond, 1979; alpha =.86, *M*=8.34, *SD*=4.95, range=0-16) and four Likert-type scale items measuring symptoms of depression (Angold, Costello, Messer, & Pickles, 1995; alpha = .86, *M*=6.47, *SD*=4.96, range=0-16). Four items for the two externalizing indicators measured the frequency with which the student perpetrated delinquent acts (Farrell, Kung, White, & Valois, 2000; alpha=.80, *M*=1.16, *SD*=2.77, range=0-16) and six items assessed the frequency with which the student perpetrated violent acts against peers (alpha=0.88, *M*=1.93, *SD*=4.18, range=0-24). The

distributions of these two externalizing items were heavily right-skewed, violating the assumption of normality required for LPA. Thus, each of the externalizing outcomes was trichotomized such that 0=none, 1=some, and 2=a lot of externalization. Cutoffs for these three categories were based on univariate statistics so that the "a lot" category captured individuals at approximately the 90th percentile for the outcome and above, the "none" category consisted of individuals reporting no externalization, and individuals with scores between 0 and the approximate 90th percentile cutoff fell into the "some" category. The five profiles resulting from the LPA were Profile 1, non-reactors (low on internalizing and externalizing reactivity); Profile 2, high internalizers and moderate externalizers; Profile 3, high internalizers and high externalizers, Profile 4, moderately anxious, moderate externalizers; Profile 5, moderate internalizers and moderate externalizers. After the number and nature of the reactivity profiles were identified through LPA, individuals were assigned to their most likely profile based on their vector of posterior probabilities (that is, the set of values describing the likelihood of being assigned to that profile, given the data). A **nominal profile variable** was created and profiles were given values of 1-5 as identified above. Eighteen percent of the 132 individuals comprising the sample for the present study fell into Profile 1 (non-reactors), 19% fell into Profile 2 (high internalizers, moderate externalizers), 14% fell into Profile 3 (high internalizers, high externalizers), 27% fell into Profile 4 (moderately anxious, moderate externalizers), and 22% fell into Profile 5 (moderate internalizers, moderate externalizers).

Control Variables

To control for potential confounding effects, demographic control variables used in analyses assessing associations between family characteristics and reactivity profiles were respondent race, grade in school, and parental education. The grade variable captured whether the student

was in grade 8, 9, or 10 at the time of the assessment. Parental education was coded as 0=Did not graduate from high school, 1= Graduated from high school, 2=Some college or tech school, 3=Graduated from community college or tech school, 4= Graduated from college, 5=Graduate or professional school. Sex was coded 0=female, 1=male.

Analysis Strategy

SAS v.9.4 was used for all statistical analyses. To test the hypothesis that adolescents with more copies of the short allele of *5-HTTLPR* would have higher likelihood of membership in the profiles characterized by high internalizing (Profile 2, high internalizing, moderate externalizing and Profile 3, high internalizing, high externalizing) than the other reactivity profiles, we created a dichotomous reactivity profile membership variable, where membership in Profiles 2 and 3 was set equal to 1 and membership in Profiles 1, 4, and 5 was set equal to 0. Binary logistic regression was then conducted with *5-HTTLPR* genotype as the independent variable and this dichotomous reactivity profile membership variable as the dependent variable, with the value of 0 (i.e. membership in Profiles 1, 4,or 5) as the reference category. Demographic controls were also included in the model.

To test the hypothesis that adolescents with more copies of the Met allele of the *BDNF* Val66Met polymorphism would be more likely to be in profiles characterized by high internalizing (Profile 2, high internalizing, moderate externalizing and Profile 3, high internalizing, high externalizing) than the other reactivity profiles, we conducted binary logistic regression with *BDNF* genotype as the independent variable and the same dichotomous reactivity profile membership as described above as the dependent variable, again with a value of 0 (i.e. membership in Profiles 1, 4, or 5) as the reference category. Demographic controls were included in the model.

To test the hypothesis that adolescents with more copies of the low-expression allele of *MAOA* will be more likely to be in profiles characterized by high externalizing (Profile 3, high internalizing, high externalizing) as compared to the other reactivity profiles, we created a different dichotomous reactivity profile membership variable, this time with membership in Profile 3 assigned a value of 1 and membership in Profiles 1,2,4, and 5 assigned a value of 0. Demographic controls were included in the model.

Missing data for the variables used in the binary logistic regression models were imputed using PROC MI, and PROC MIANALYZE was used to pool the results from the logistic regression models that were fit on the imputed datasets. Eighteen percent of observations were missing on parental education and 5% were missing on *BDNF* genotype. There were no missing data on *5-HTTLPR* or *MAOA* genotype sex, grade, or latent profile. The multiple imputation missingness models included all variables that were included in the binomial logistic regression models as recommended by Allison (2002). All variables in the multiple imputation models were quantitative in nature (either binary, ordered categorical, or continuous). Minimum and maximum values were specified to ensure that plausible values were imputed for all categorical variables. Twenty imputations were run based on recommendations by Graham and colleagues (2007).

Results

Associations between *5-HTTLPR* Genotype and Reactivity Profile Membership

We hypothesized that adolescents with more copies of the short-allele of *5-HTTLPR* would have greater likelihood of membership in the high internalizing profiles (Profile 2, high internalizers, moderate externalizers and Profile 3, high internalizers, high externalizers) as compared to the other reactivity profiles. The results from testing this hypothesis are presented in

Table 3.1. There was no statistically significant association between *5-HTTLPR* and reactivity profile membership. In this analysis, however, we found a significant association between sex and reactivity profile membership, with boys being 66% less likely than girls to be in the high internalizing profiles as compared to all others (OR=.34; 95% CI: .14, .83).

Table 3.1. Binary logistic regression (OR and 95% CI) of reactivity profile membership on *5-HTTLPR* genotype

Variable	OR	95% CI
5-HTTLPR	.98	(.57, 1.67)
Sex (M vs. F)	**.34**	**(.14, .83)***
Parent education	.93	(.71, 1.22)
Grade	1.50	(.92, 2.46)

Note: For the dependent variable, 1=membership in Profile 2 (high internalizers, moderate externalizers) or Profile 3 (high internalizers, high externalizers); 0 (reference category) = membership in Profile 1 (non-reactors), Profile 4 (moderately anxious, moderate externalizers), or Profile 5 (moderate internalizers, moderate externalizers).
*p<.05

Associations between *BDNF* Genotype and Reactivity Profile Membership

We hypothesized that adolescents with more copies of the Met allele of the Val66Met polymorphism of *BDNF* would have greater likelihood of membership in the high internalizing profiles (Profile 2, high internalizers, moderate externalizers and Profile 3, high internalizers, high externalizers) as compared to the other reactivity profiles. The results from testing this hypothesis are presented in Table 3.2. Binary logistic regression testing the association between *BDNF* genotype and reactivity profile membership revealed no significant association. However, we found a significant association between sex and reactivity profile membership with boys being significantly less likely than girls to be in the high internalizing profiles as compared to the other reactivity profiles (OR=.34; 95% CI: .14, .82).

Table 3.2. Binary logistic regression (OR and 95% CI) of reactivity profile membership on *BDNF* genotype

Variable	OR	95% CI
BDNF	.70	(.34, 1.41)
Sex (M vs. F)	**.34**	**(.14, .82)***
Parent education	.91	(.70, 1.20)
Grade	1.52	(.94, 2.46)

Note: For the dependent variable, 1=membership in Profile 2 (high internalizers, moderate externalizers) or Profile 3 (high internalizers, high externalizers); 0 (reference category) = membership in Profile 1 (non-reactors), Profile 4 (moderately anxious, moderate externalizers), or Profile 5 (moderate internalizers, moderate externalizers).
*p<.05

Associations between MAOA Genotype and Reactivity Profile Membership

We hypothesized that adolescents with more copies of the low-expression alleles of *MAOA* would have greater likelihood of membership in the profile characterized by high externalizing (Profile 3, high internalizers, high externalizers), as compared to all other profiles. The results from testing this hypothesis are presented in Table 3.3. Binary logistic regression revealed no significant association between *MAOA* genotype and reactivity profile membership. We note, however, that boys had significantly lower likelihood than girls of being in the high externalizing reactivity profiles compared to all other profiles (OR=.08; 95% CI: .01, .66).

Table 3.3. Binary logistic regression (OR and 95% CI) of reactivity profile membership on *MAOA* genotype

Variable	OR	95% CI
MAOA	1.23	(.54, 2.79)
Sex (male vs female)	**.08**	**(.01, .66)***
Parent education	1.04	(.73, 1.47)
Grade	1.16	(.61, 2.19)

Note: For the dependent variable, 1=membership in Profile 3 (high internalizers, high externalizers); 0 (reference category) = membership in Profile 1 (non-reactors), Profile 2 (high

internalizers, moderate externalizers), Profile 4 (moderately anxious, moderate externalizers), or Profile 5 (moderate internalizers, moderate externalizers).

Other Findings (These are reported for the purposes of the dissertation but will be deleted for manuscript submission).

Chi-square tests revealed there were no significant differences in the distribution of putative alleles for *5-HTTLPR, BDNF,* or *MAOA* across the five reactivity profiles. These results are presented in Tables 3.4-3.6.

Table 3.4. Results of Chi-Square Test for distribution of short alleles of *5-HTTLPR* by bullying victimization reactivity profile

# of short alleles	Profile 1 N (column %)	Profile 2 N (column %)	Profile 3 N (column %)	Profile 4 N (column %)	Profile 5 N (column %)	Total
0	7 (29.2)	9 (36.0)	7 (36.8)	10 (28.6)	11 (37.9)	44
1	9 (37.5)	12 (48.0)	8 (42.1)	19 (54.3)	13 (44.8)	61
2	8 (33.3)	4 (16.0)	4 (21.1)	6 (17.1)	5 (17.2)	27
Total	24	25	19	35	29	132

Note: X^2=4.18, df=8, p=.84. Profile 1=non-reactors; Profile 2=high internalizers, moderate externalizers; Profile 3=high internalizers, high externalizers; Profile 4=moderately anxious, moderate externalizers; Profile 5=moderate internalizers, moderate externalizers.

Table 3.5. Results of Chi-Square Test for distribution of Met alleles of *BDNF* by bullying victimization reactivity profile

# of Met alleles	Profile 1 N (column %)	Profile 2 N (column %)	Profile 3 N (column %)	Profile 4 N (column %)	Profile 5 N (column %)	Total
0	14 (60.9)	14 (58.3)	12 (63.2)	18 (56.3)	15 (55.6)	73
1	7(30.4)	10 (41.7)	7 (36.8)	13 (40.6)	10 (37.0)	47
2	2 (8.7)	0 (0.0)	0 (0.0)	1 (3.1)	4 (7.4)	5
Total	23	24	19	32	27	125

Note: X^2=4.50, df=8, p=.81. Profile 1=non-reactors; Profile 2=high internalizers, moderate externalizers; Profile 3=high internalizers, high externalizers; Profile 4=moderately anxious, moderate externalizers; Profile 5=moderate internalizers, moderate externalizers.

Table 3.6. Results of Chi-Square Test for distribution low-activity *MAOA* alleles by bullying victimization reactivity profile

# of low-activity alleles[§]	Profile 1 N (column %)	Profile 2 N (column %)	Profile 3 N (column %)	Profile 4 N (column %)	Profile 5 N (column %)	Total
0	14 (58.3)	12 (48.0)	7 (36.8)	18 (51.4)	10 (34.5)	61
1	5 (20.8)	10 (40.0)	10 (52.6)	13 (37.1)	11 (37.9)	49
2	5 (20.8)	3 (12.0)	2 (10.5)	4 (11.4)	8 (27.6)	22
Total	24	25	19	35	29	132

Note: X^2=8.88, df=8, p=.35. Profile 1=non-reactors; Profile 2=high internalizers, moderate externalizers; Profile 3=high internalizers, high externalizers; Profile 4=moderately anxious, moderate externalizers; Profile 5=moderate internalizers, moderate externalizers.
§ Boys hemizygous for the high-activity allele coded as 0; Boys hemizygous for the low-activity allele coded as 2.

A second set of chi-square tests was conducted to test distributions of putative alleles by reactivity profiles when grouped according to the hypothesis for each candidate gene. The distribution of short alleles of *5-HTTLPR* was not significantly different when compared between the high internalizing (Profiles 2 and 3) and the other reactivity profiles (Profiles 1, 4, and 5) grouped together (Table 3.7).

Table 3.7. Results of Chi-Square Test for distribution of short alleles of *5-HTTLPR* by dichotomized bullying victimization reactivity profile

# of short alleles	Non-high internalizing profiles (Profiles 1, 4, 5) N (column %)	High internalizing profiles (Profiles 2 and 3) N (column %)	Total
0	28 (31.8)	16 (36.4)	44
1	41 (46.6)	20 (45.5)	61
2	19 (21.6)	8 (18.2)	27
Total	88	44	132

Note: X^2=.36, df=2, p=.84. Profile 1=non-reactors; Profile 2=high internalizers, moderate externalizers; Profile 3=high internalizers, high externalizers; Profile 4=moderately anxious, moderate externalizers; Profile 5=moderate internalizers, moderate externalizers.

Likewise, the distribution of Met alleles of *BDNF* was not significantly different when compared between these two groups of profiles (Profiles 2 and 3 vs. Profiles 1, 4, and 5; Table 3.8).

Table 3.8. Results of Chi-Square Test for distribution of Met alleles of *BDNF* by dichotomized bullying victimization reactivity profile

# of Met alleles	Non-high internalizing profiles (Profiles 1, 4, 5) N (column %)	High internalizing profiles (Profiles 2 and 3) N (column %)	Total
0	47 (57.3)	26 (60.5)	73
1	30 (36.6)	17 (39.5)	47
2	5 (6.10)	0 (0.00)	5
Total	82	43	125

Note: X^2=2.74, df=2, p=.25. Profile 1=non-reactors; Profile 2=high internalizers, moderate externalizers; Profile 3=high internalizers, high externalizers; Profile 4=moderately anxious, moderate externalizers; Profile 5=moderate internalizers, moderate externalizers.

The distribution of low-activity alleles of *MAOA* was not significantly different when compared across the high externalizing profile (Profile 3) and the other reactivity profiles (Table 3.9).

Table 3.9 Results of Chi-Square Test for distribution of low-activity alleles of *MAOA* by dichotomized bullying victimization reactivity profile

# of low-activity alleles§	High externalizing profile N (column %)	Non-high externalizing profiles N (column %)	Total
0	54 (47.8)	7 (36.8)	61
1	39 (34.5)	10 (52.6)	49
2	20 (17.7)	2 (10.5)	22
Total	113	19	132

Note: X^2=2.36, df=2, p=.31. Profile 1=non-reactors; Profile 2=high internalizers, moderate externalizers; Profile 3=high internalizers, high externalizers; Profile 4=moderately anxious, moderate externalizers; Profile 5=moderate internalizers, moderate externalizers.
§ Boys hemizygous for the high-activity allele coded as 0; Boys hemizygous for the low-activity allele coded as 2.

Although they are hypothesized to affect reactivity to social stressors through different mechanisms *5-HTTLPR* and *BDNF* were both hypothesized to be associated with high internalizing profiles (Profiles 2 and 3); therefore, a potential additive effect of the putative alleles for *5-HTTLPR* and *BDNF* was tested through binary logistic regression. The model tested whether the combined number of putative alleles of *5-HTTLPR* and *BDNF* would be associated

with membership in the high internalizing profiles as compared to the other reactivity profiles. Therefore, the dependent variable was a dichotomous reactivity profile membership variable where membership in the high internalizing profiles (Profiles 2 and 3) was set equal to 1 and membership in all other reactivity profiles (Profiles 1, 4, and 5) was set equal to 0. The results of this binary logistic regression are shown in Table 3.10. There was no significant association between putative allele risk scale and reactivity profile membership. Sex was significantly associated with profile membership, however, with boys 65% less likely than girls to be in the high internalizing profiles as compared to the other profiles (OR=.35; 95% CI: .14, .84).

Table 3.10. Binary logistic regression (OR and 95% CI) of membership in the high internalizing profiles on additive *5-HTTLPR* and *BDNF* putative allele scale

	OR	95% CI
Risk scale	.91	(.58, 1.42)
Sex (M vs. F)	**.35**	**(.14, .84)***
Parent education	.92	(.70, 1.21)
Grade	1.48	(.91, 2.41)*

Note: For the dependent variable, 1=membership in Profile 2 (high internalizers, moderate externalizers) or Profile 3 (high internalizers, high externalizers); 0 (reference category) = membership in Profile 1 (non-reactors), Profile 4 (moderately anxious, moderate externalizers), or Profile 5 (moderate internalizers, moderate externalizers).
*p<.05

A second binary logistic regression model tested whether the combined number of putative alleles of *5-HTTLPR*, *BDNF*, and *MAOA* would be associated with membership in any of the reactivity profiles as compared to the non-reactor profile. In this model, the dependent variable was a dichotomous reactivity profile variable where membership in Profiles 2, 3, 4, or 5 was set equal to 1 and membership in the non-reactor profile was set equal to 0. The results of this binary logistic regression are shown in Table 3.11. There was no significant association between putative allele risk scale and reactivity profile membership. Boys were significantly less likely

than girls to be in any of the reactivity profiles compared to the non-reactor profile (OR=.31, 95% CI: .12, .83). Additionally, increased grade level was associated with an increased likelihood of being in the more problematic reactivity profiles compared to the non-reactor profile (OR=2.11, 95% CI: 1.09, 4.06).

Table 3.11. Binary logistic regression (OR and 95% CI) of membership in any reactivity profile on additive *5-HTTLPR, BDNF,* and *MAOA* putative allele scale

	OR	95% CI
Risk scale	1.13	(.74, 1.71)
Sex (M vs. F)	**.31**	**(.12, .83)***
Parent education	.92	(.66, 1.28)
Grade	**2.11**	**(1.09, 4.06)***

Note: For the dependent variable, 1=membership in Profile 2 (high internalizers, moderate externalizers) or Profile 3 (high internalizers, high externalizers), Profile 4 (moderately anxious, moderate externalizers), or Profile 5 (moderate internalizers, moderate externalizers); 0 (reference category) = membership in Profile 1 (non-reactors).
*p<.05

Discussion

We have confidence in the rationale for testing the influence of *5-HTTLPR, BDNF* and *MAOA* on bullying victimization based on previous candidate gene studies and also rodent and human studies that provide biological plausibility for the role of genotype in reactivity to social stressors. However, we did not find significant associations between genotype for *5-HTTLPR, BDNF,* and *MAOA* and reactivity profile membership, even when not controlling for demographic variables. There are several potential explanations for our null results. One is that the sample size was too small (n=132) to detect significant associations. Post-hoc power analysis was conducted in SAS 9.4. The analysis indicated that the power to detect the obtained effect for 5-HTTLPR genotype at the .05 level was .03; the power to detect the obtained effect for *BDNF* genotype at the .05 level was .17; and the power to detect the obtained effect for *MAOA* genotype at the .05 level was .07.

Another explanation for our null findings is that *5-HTTLPR*, *BDNF*, and *MAOA* may influence reactivity, but other markers of the genes are needed to capture those associations. For example, the addition of other SNPs of the *SLC6A4* gene to our analysis (in addition to *5-HTTLPR*) may have uncovered associations between that gene and the high reactivity profiles. Additionally, it is also possible that these genes or a subset thereof operate additively or epistatically to influence reactivity to bullying victimization. As previously noted, we chose to examine the effects of *5-HTTLPR*, *BDNF*, and *MAOA* separately rather than create an aggregate risk scale because we hypothesized different mechanisms and effects for each one. In the exploratory analyses where a sum of putative alleles was attempted, no significant effects were found, but it is possible that alternate combinations or interaction terms are needed. Analysis of gene x gene interactions was precluded by sample size considerations. Also, we tested only three genes that have been implicated in response to peer rejection and social exclusion. Other genes that regulate the serotonergic and dopaminergic systems, such as *DAT1*, *DRD2*, and *DRD 4*, for example, may be considered in future research. Additionally, there may be other genes that influence these systems that have yet to be identified. Lastly, although the use of profiles of reactivity to victimization is a strength of this study that uses a person-centered methodology to capture sequelae of bullying victimization, it is possible that these profiles obscured associations if the genes examined here are associated with only one or a subset of the dimensions of internalizing and externalizing reactivity that are reflected in Eastman et al.'s (unpublished) profiles of reactivity.

Despite our null findings regarding genotype, we found associations between demographic variables and reactivity profile membership that are of some interest. We found that boys were less likely than girls to be in the high internalizing profiles (when testing associations

between *5-HTTLPR* and *BDNF* and profile membership) and the high externalizing profile (when testing associations between *MAOA* and profile membership). This is consistent with findings of Eastman et al (unpublished), who found that boys were less likely than girls to be in any of the reactivity profiles compared to the non-reactor profile. While studies show that the prevalence of bullying victimization is the same for girls and boys (Kochenderfer-Ladd, Ladd, & Kochel, 2009), our results suggest that the experience of victimization may be more damaging for girls. One potential explanation is that girls—more so than boys--are socialized to maintain and protect social relationships (Carbone-Lopez, Esbensen, & Brick, 2010). The social failure represented by bullying victimization may, therefore, be felt more acutely by girls than boys.

Some limitations of this study must be noted. As acknowledged previously, our ability to detect significant associations between genotypes for the candidate genes of interest and reactivity profile membership may be limited by the size of our sample (n=132). Additionally, we note the cross-sectional nature of the study. We cannot assert that exposure to bullying occurred before reports of internalizing and externalizing symptoms; our use of the term "reactivity" to describe these symptoms must be considered with this caveat. Limitations of the LPA used in our tests of statistical significance have been noted elsewhere (see Eastman et al., unpublished), and the present study must be considered in light of these limitations, as well.

Although we failed to find significant associations between genotype for *5-HTTLPR*, *BDNF*, and *MAOA* and reactivity profile membership, we remain supportive of research that explores the potential relationship between genotype and reactivity to bullying victimization. Such research holds promise to elucidate the biological processes and mechanisms that underlie internalizing and externalizing reactivity to bullying victimization among adolescents.

CHAPTER 6: SUMMARY

This dissertation aimed to identify heterogeneity in reactivity to bullying victimization among adolescents and to examine whether characteristics of the bullying (type and frequency), family characteristics, and selected biologically plausible genetic polymorphisms explain this heterogeneity in reactivity to bullying victimization.

In the study addressing Aim 1, we identified five profiles of internalizing (anxiety and depression) and externalizing (delinquency and violence against peers) reactivity using Latent Profile Analysis (LPA) in a sample of adolescents who had been victims of bullying. These profiles demonstrated distinct characteristics: Profile 1: low internalizing, low externalizing (non-reactors); Profile 2: high internalizers, moderate externalizers; Profile 3: high internalizers, high externalizers; Profile 4, moderately anxious, moderate externalizers; and Profile 5, moderate internalizers, moderate externalizers. The person-centered approached taken to address Aim 1 allowed for identification of more complex responses to bullying victimization than could be identified in a single-outcome, variable-centered study.

In the study addressing Aim 2, we examined the influence of bullying characteristics (type and frequency) on membership in the five reactivity profiles. We found direct victimization (i.e., physical violence, name calling) increased odds of membership in the high internalizers, high externalizers profile compared to all other profiles. Indirect victimization (i.e., damage to social relationships) increased odds of membership in the high internalizing profiles compared to the lower internalizing profiles. Dual (i.e., direct and indirect) victimization increased odds of membership in the high internalizers, high externalizers profile compared to each other profile.

More frequent victimization was associated with increased odds of membership in the two high internalizing reactivity profiles compared to the non-reactor profile. Although there were no gender differences in these associations, girls were significantly more likely than boys to be in any of the non-reactor profiles. The results of Aim 2 underscore the importance of taking into account characteristics of the victimization in understanding reactivity to bullying victimization.

Aim 3 sought to examine the stress-buffering effects of parental warmth and the exacerbating effects of family conflict on membership in the reactivity profiles. We found that parental warmth had a buffering effect on reactivity profile membership for girls only, and that buffering effect protected girls from membership in the two high internalizing profiles, specifically. Family conflict increased likelihood of membership in the high internalizing, high externalizing profile compared to all others, and its effect did not vary by sex of the victim. We hypothesized that family characteristics would moderate the relationship between frequency of victimization (a measure of the severity of the bullying stressor) and reactivity profile membership. This was not the case; however, our results did indicate a stress-buffering effect of parental warmth (for girls) and an exacerbating effect of family conflict. These results underscore the importance of the family environment in shaping reactivity to bullying victimization among adolescents. Secondary interventions that aim to leverage warmth and reduce conflict within the family may reduce the negative sequelae of bullying victimization among adolescents.

In Aim 4, we tested the influence of genotype for *5-HTTLPR, BDNF,* and *MAOA.* Despite biological plausibility of these genes to influence reactivity to bullying victimization, the results of our binary logistic regression models for each gene showed no association between

genotype and reactivity profile membership. These null results reflect the need for further research on the biological underpinnings of reactivity to bullying victimization

This dissertation used a person-centered approach, LPA, to identify complex typologies of reactivity to bullying victimization and examined factors at multiple levels of influence. In doing so, this dissertation adds to the body of knowledge on reactivity to bullying victimization and its predictors.

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