PEER VICTIMIZATION AND THE COMT VAL^{158}MET POLYMORPHISM: A DIFFERENTIAL SUSCEPTIBILITY MODEL

by

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Abstract

PEER VICTIMIZATION AND THE COMT VAL^{158}MET POLYMORPHISM: A DIFFERENTIAL SUSCEPTIBILITY MODEL

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The link between peer victimization and various health related negative outcomes ranging from physical to psychological has been well established in the literature (Hodges & Perry, 1999; Rigby, 2001). While there are many individual differences in the observed relationship, few have explored the role of Catechol-O-methyl transferase (COMT) gene, val^{158}met, within this relationship. Prior research has shown that COMT is involved in altered stress reactivity, internalizing problems, and poor health outcomes (Armbruster et al., 2012; Hagen et al., 2006; Reuter & Hennig, 2005). This thesis explored the influence of the COMT gene on the relationship between peer victimization and adverse outcomes in a sample of 149 adolescents (M_{age} = 12.67). Regression analyses show that overall victimization as well as physical, verbal, and indirect subtypes, predicted poorer health outcomes, internalizing problems, and somatic complaints. COMT allelic variants, specifically individuals with at least one MET allele, were found to moderate the relationship between peer victimization and severity of health symptoms, internalizing problems, and somatic complaints. COMT, gender, and peer victimization significantly interacted to predict increased somatic complaints. Hierarchical linear model revealed that COMT and

victimization interacted to predict altered stress reactivity within the Trier Social Stress Test (TSST); victimized individuals consistently showed blunted cortisol response. Further, victimized girls showed lower cortisol responses compared to non-victims. Marginally, COMT interacted with victimization and the TSST to show that homozygous MET individuals with a low history of victimization show a blunted response. These results provide evidence that the COMT polymorphisms differentially influence victimization and negative health outcomes, explaining why some individuals are more susceptible to the adverse effect of bullying.

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Chapter 1

Introduction

In 2011, the Youth Risk Behavior Surveillance System that monitors health-risk behaviors among youth and young adults found in a nationally representative sample of high school students that 20.1% reported being bullied on school property; 16.2% nationwide reported that were victims of electronic bullying through social media websites, texting, online chat rooms and instant messaging services and 6% of students reported intentionally missing school because they felt unsafe at school or on their way to or from school (Eaton et al., 2012). Peer victimization, known colloquially as bullying, is a severe problem garnering increased media attention over the past decade. In the critically acclaimed documentary *Bully*, the filmmakers follow the daily lives of several bullied children: a quirky twelve year old named Alex who is persistently taunted and physically abused by his peers, a sixteen year old name Kelby who recently came out to her family and friends is forced to quit her sports teams and once was run down by a van driven by her peers, fourteen year old Ja'Meya who was verbally abused every day on the bus to and from school until she stole her mother's loaded handgun to threaten her tormentors, and the families of Tyler Long and Ty Smalley, two teenagers who tragically ended their lives as a result of persistent and severe bullying (Waitt & Hirsch, 2012). These adolescents' stories are part of a larger narrative within the bullying epidemic; those who bully their peers are unrelenting, creative, and persistent in their verbal and physical aggressions; their victims also have a wide range of responses from silently keeping their head down and taking it, to turning violent and aggressive like their tormentors, to becoming depressed and suicidal.

Not all adolescents respond to bullying the same way, exploring the various facets of peer victimization is a crucial task that seeks to illuminate these individual

differences in response to being peer victimized. Therefore, this thesis sought to examine how the genetic contributions of the Catechol-O-methyl transferase (COMT) influence the relationships of peer victimization on depression, health outcomes, and stress reactivity.

Defining Peer Victimization

Peer victimization is marked by repeatedly being the target of intentionally aggressive acts or behaviors of one's peers. However, peer victimization does not include peers of similar status arguing or fighting with one another, children or adolescents who tease one another in a good-natured manner, or when a child or adolescent is the recipient of an intermittent aggressive act (Andreou, 2001; Olweus, 1993). Several types of victimization exist: verbal, physical, and relational victimization, as well as cyberbullying (Crick & Grotpeter, 1995; Olweus, 2001). Verbal victimization includes shouting or yelling, name calling, and is focused on psychological abuse; Physical victimization includes kicking, punching, or shoving where children are harmed through physical damage of one's self or one's property, or threat of such actions (Underwood, Beron, & Rosen, 2009). Similar to verbal, relational or social victimization harms children through trying to control, damage, or attack their relationships by exclusion, rumors, gossiping, and other means to which one could damage a relationship (Crick, Casas, & Ku, 1999). Cyberbullying includes bullying behaviors that embarrass, threaten, or taunt the victim via communication in any form over mobile phones, video clips, internet, and photos and can be verbal, physical, or relational in nature (Smith, Mahdavi, Carvalho, Fisher, Russell, & Tippett, 2008).

Though there are different types of victimization, some researchers have suggested that frequency and severity of peer victimization are more important attributes, classifying individuals into three distinct categories, victimized, sometimes victimized, and non-victimized (Graham & Juvonen, 1998; Nylund, Bellmore, Nishina, & Grahman, 2007).

Indeed, peer victims often experience a combination of verbal, physical, and relational forms of abuse. Therefore, in this proposed thesis, peer victimization will be considered as a continuum ranging from no abuse to frequent peer abuse and mistreatment. It is expected that results will be the same for all types of bullying, but supplementary analyses will be conducted to examine possible differences among types of victimization, especially given current research has found that relational forms of abuse may be particularly harmful compared to more physical forms (Iyer-Eimerbrink, Scielzo, & Jensen-Campbell, under review; Guarneri-White & Jensen-Campbell, under review).

Importance of Studying Adolescents

Adolescence is a critical developmental period to study. Significant changes in biological, cognitive, and social functions are occurring and these changes can be greatly impacted by psychosocial stressors, such as peer victimization. During this developmental period, relationships with parents increase in bickering and decrease in closeness, self-esteem and self-identity are developing, and increased amounts of time are spent away from the home with friends. Friendships increase in intimacy, support, and trust during this developmental stage. Peers influence adolescents in both positive and negative ways, such as in areas of academic achievement, prosocial behaviors, drug and alcohol abuse, smoking, and delinquency (Steinberg & Morris, 2001). Also during this period, adolescents are more vulnerable to stress, which substantially influences the onset of mental illness such as depression, anxiety, and mood disorders (Hankin & Abramson, 2001; Lerner & Steinberg, 2009). This developmental period is critical, such that adolescent anxiety and depression increased the risk of as well as predicted adult anxiety and depression disorders (Pine, Cohen, Gurley, Brook, & Ma, 1998).

In regards to peer victimization in adolescents, it can lead to future adjustment problems such as academic struggles or failures, teen pregnancy, and mental health

issues like unipolar depression (Crick & Bigbee, 1998; Perrin, Ettekal, & Ladd, 2013; Schwartz, Lansford, Dodge, Pettit, & Bates, 2013 & 2014). Adolescents who are peer victimized have also been shown to have lower teacher engagement and less social satisfaction; such that they have fewer friends to talk to and play with or to get help from on assignments (Graham, Bellmore, & Mize, 2006; Kochenderfer-Ladd & Wardrop, 2001).

Furthermore, research has shown that adolescents who were recently exposed to peer victimization were at 2.4 time's greater risk of suicidal ideation than non-exposed adolescents (Rigby, 2001; Turner, Finkelhor, Shattuck, & Hamby, 2012). Nationwide in 2011, Youth Risk Behavior Surveillance System found that 15.8% of students had seriously considered attempting suicide within the past year, 12.8% of students had made a plan about how they would attempt suicide, and 7.8% of students had attempted suicide one or more times during the last year (Eaton et al., 2012). Further elucidating the link between peer victimization and depression could illuminate possible interventions for this vulnerable population.

Consequences of Peer Victimization

Psychosocial Health Outcomes

Regardless of the form of peer victimization, being the frequent recipient of aggressive acts and behaviors from one's peers can result in negative outcomes, both psychological and physical. Peer victimization has been shown to cause psychological maladjustments, in particular, victims may draw negative conclusions about themselves which lead to developing internalizing problems such that they become depressed, have difficulty asserting themselves in social situations, experience emotional distress, and report feeling lonely. Individuals who are recipients of relational bullying report more difficulty inhibiting anger, greater impulsivity, and are more submissive to their peers

(Crick & Bigbee, 1998). Also, the stability of peer victimization over time leads to difficulties with anxiety, loneliness, low self-esteem, and rejections by peers; these difficulties have also been shown to increase the levels of victimization experienced, which in turn increases the difficulties of the victimized individual resulting in a vicious downward cycle (Hawker & Boulton, 2000; Hodges & Perry, 1999). It has also been shown that frequent peer victimization is associated with low social support which can lead to poor mental health outcomes such as somatic symptoms like feeling run down, anxiety, social dysfunction, and depression (Rigby, 2000).

In a meta-analytic review of twenty years of peer victimization research, Hawker and Boulton (2000) showed that peer victimization led to more negative affect, negative thought of self and psychosocial maladjustment. Specifically, social anxiety, generalized anxiety, social self-concept, global self-esteem, and were all negatively impacted by peer victimization; overall, depression had the strongest relationship and largest effect sizes associated with peer victimization. Victims are typically characterized with having strong feelings of fearfulness, loneliness, and dysphoria; it has been suggested that these feelings are strongly associated with the development of depression (Hawker & Boulton, 2000). Furthermore, expounding on the work of Hawker and Boulton (2000), Iyer-Eimerbrink, Scielzo, and Jensen-Campbell (under review) conducted a meta-analytic review spanning thirty-five years of research and found similar results; peer victimization had a moderate relationship between the internalizing problems of depression, anxiety, and loneliness. Moreover, type of victimization was a moderator between peer victimization and internalizing problems; specifically, social victimization had a stronger association with depression and anxiety than physical victimization while loneliness had a stronger association with physical victimization. Gender differences were also observed, such as females reported more anxiety and depression when victimized than did boys.

Physical Health Outcomes

Peer victimization also causes long lasting physical health problems (Olweus, 1992; Rigby, 2001). Victimization has been shown to cause increased sleep problems, headaches, and respiratory problems (Biebl, DiLalla, Davis, Lynch, & Shinn, 2011). Bullied individuals also report higher levels of mouth sores, thumping in the chest, abdominal pain, tense feelings, bed-wetting, feeling tired, and poorer appetite compared to non-bullied individuals (Fekkes, Pijpers, Fredriks, Vogels, & Verloove-Vanhorick, 2006; Rigby, 2001). Additionally, victimized individuals have reported more sore throats, colds, and coughs than their fellow peers; these victims are also more likely to make up illnesses to stay home because they are more worried about attending school (Wolke, Woods, Bloomfield, & Karstadt, 2001). However, victimized individuals have a higher frequency of visits to health care professionals suggesting that victimization is associated with real illnesses (Knack, Jensen-Campbell, Baum, 2011). It has also been shown that frequency of victimization is associated with increased odds of having heart problems, bone and joint problems, chest pains, and high blood pressure (Knack, Iyer, & Jensen-Campbell, 2012).

Stress Reactivity

The relationship between peer victimization and poor psychological and physiological outcomes has been well established in the literature (Crick & Bigbee, 1998; Hawker & Boulton, 2000; Rigby, 2001). However, further research is needed to examine the possible biological mechanisms associated with peer victimization. Stressors have been shown to affect neuroendocrine functioning, specifically, altering the production of the glucocorticoid cortisol through the hypothalamic-pituitary-adrenocortical (HPA) system. During a stress response, corticotrophin releasing hormone (CRH) and arginine vasopressin (AVP) are released by the hypothalamus. Which in turn, CRH and AVP

travel to the anterior pituitary and stimulate the release of adrenocorticotropic hormone (ACTH); ACTH interacts with the adrenal gland to stimulate the release of glucocorticoids like cortisol into the circulation system. Thusly, during a psychological stressor cortisol levels are elevated to help prepare energy stores for a behavioral response to a threat (Gunnar & Quevedo, 2007). However, during the presence of a chronic stressor, like peer victimization, this cortisol response can be altered and be detrimental to the individual; specifically, frequent verbal victimization has been shown to be associated with hyposecretion of cortisol (Vaillancourt, Duku, Decatanzaro, Macmillam, Muir, & Schmidt, 2008).

Researchers mimic social stressors in the laboratory using the Trier Social Stress Test (TSST), which has been shown to activate the HPA axis (Kirschbaum, Pirke, & Helhammer, 1993). The TSST involves participants being asked to give a five minute speech (after ten minutes of preparation) in front of a committee of three individuals posing as experts who are allowed to probe and ask questions if the individual stops talking during their five minute speech. Throughout the various steps in the TSST, four salivary cortisol samples are collected in order to assess how an acute social stress affects stress reactivity. Furthermore, the TSST can be used to examine how a chronic stressor, like peer victimization, can alter an individual's cortisol stress reactivity in response to acute social stressors.

Knack, Jensen-Campbell, and Baum (2011) found that chronically victimized adolescents reported feeling less accepted and had higher stress levels after the TSST. Moreover, victims displayed altered cortisol levels after the TSST such that victimized individuals had a significant decrease in levels of cortisol when compared to non-victims thirty minutes after the delivery of the TSST demonstrating that chronic stress alters HPA activity and social stress reactivity. After controlling for genetic background and family

environment, Ouellet-Morin and her colleagues (2011) found that in monozygotic twin pairs the bullied twin showed decreased or blunted cortisol reactivity levels in response to a psychosocial stress test compared to their non-bullied twin who showed an increase in cortisol due to the acute social stressor.

Prior research has shown that peer victimization and cortisol levels interacted to predict depressive symptoms over time. Highly victimized individuals with high levels of cortisol in wait of a social challenge were more than likely to display depressive symptoms a year later; although, high levels of anticipatory cortisol were thought to be beneficial to low-victimized individuals (Rudolph, Troop-Gordon, & Granger, 2011). Harkness, Stewart, and Wynne-Edwards (2011) found that adolescents with a history of maltreatment and mild to moderate levels of depression had higher levels of cortisol in response to a social challenge than adolescents with no history of maltreatment; however, adolescents with moderate to severe depression had a blunted cortisol response regardless of maltreatment history. It is critical that the link between peer victimization, cortisol, and depression be explored further.

Genetic Contribution of Catechol-O-methyl Transferase

Though those that suffer from chronic peer victimization have more negative physiological and psychosocial outcomes, not all victimized individuals display these poorer health outcomes. Accordingly, researchers need to better understand why some children may be more adversely affected by peer victimization than other children. One possible cause that could put some adolescents more at risk for developing symptoms of depression when victimized by peers is a genetic polymorphism within the Catechol-O-methyl transferase (COMT) gene. The COMT gene encodes an enzyme that is responsible for the degradation of catecholamine neurotransmitters such as dopamine, epinephrine, and norepinephrine. A specific functional single nucleotide polymorphism of

the COMT gene that codes the substitution of valine (VAL) by methionine (MET) at codon 158 (rs4680), val^{158}met, reduces the thermostability and activity of the enzyme and is of particular interest; three different genotypes are produced at this exact location: val/val, val^{158}met, and met/met. The val/val genotype has the highest level of COMT enzymatic activity, followed by the val^{158}met with intermediate activity and then the met/met genotype with the lowest activity; meaning that the val/val genotype degrades neurotransmitters at a higher rate than val^{158}met and met/met variants which have moderate and low degradation respectively (Zubieta et al., 2003; Nackley et al. 2006).

The nucleotide polymorphism of the COMT gene, val^{158}met, has been linked to several behavioral diseases and functional processes. When participating in the Trier Social Stress Test (TSST) for Children, which mimics a stressful life event, individuals with homozygous methionine genotype had a significantly higher cortisol response compared to heterozygous and val/val variants (Armbruster, Mueller, Strobel, Lesch, Brocke, & Kirschbaum, 2012). When participating in the Groningen Acute Stress test, a modified version of the TSST, individuals with the MET allele reported higher subjective stress responses and showed higher endocrine responses; sex differences are also observed: women showed higher levels of adrenocorticotropic hormone, a measure of the hypothalamic-pituitary-adrenal axis, in response to the stress test, and men showed higher mean plasma epinephrine responses (Jabbi, Kema, van der Pompe, te Meerman, Ormal, & den Boer, 2007). These results suggest that COMT is involved in the stress response and that certain allelic variations interact with the environment making those individuals more genetically vulnerable to stressors such as peer victimization.

The COMT polymorphism has also been associated with high neuroticism and introversion, which are both anxiety-related traits. Specifically, women with the met/met haplotype are more likely to exhibit high neuroticism and low extraversion (Stein, Fallin,

Schork, & Gelernter, 2005). Similarly, in women, the met/met genotype was associated with greater tendencies to avoid harm, fear of uncertainty, anticipatory worry, and higher levels of neuroticism as well (Enoch, Xu, Ferro, Harris, & Goldman, 2003). Tragically, it has been shown that met/met allelic variation was significantly over-represented among a group of violent suicide attempters, individuals who attempted suicide via hanging, shooting, stabbing, et cetera (Rujescu, Gieglin, Gietl, Hartmann, & Möller, 2003).

Individuals with the val^{158}met and met/met variant have been shown to have increased sensitivity to pain and increased likelihood of developing temporomandibular joint disorder (Diatchenko et al., 2005) when compared to the val/val genotype. Headaches were more prevalent among women with the MET variants than individuals with the val/val genotype (Hagen, Pettersen, Stovner, Skorpen, & Zwart, 2006). Also, the val/met haplotype was indicted in an increased rate of temporal summation of pain; other COMT single nucleotide polymorphisms were also associated with increased sensitivity to noxious stimuli such as thermal, ischemic, and pressure induced pain (Diathchenko et al., 2006).

In brain areas that are part of the corticolimbic circuit of emotional processing and arousal, such as the left hippocampus, right amygdala, and prefrontal cortex, met/met homozygotes have been shown to have greater activity when compared to their val/val counterparts when viewing faces displaying negative emotion; MET variants may be more sensitive to negative environmental cues which may exaggerate their arousal responses (Drabant et al., 2006). Increased limbic activity in methionine alleles has been shown to contribute to lower emotional resilience in negative mood states (Smolka et al., 2005). Furthermore, met/met genotypes are associated with greater expression of anger (Oppenheimer, Hankin, Jenness, Young, & Smolen, 2013). The homozygous MET allele has also been associated with heightened scores of negative emotionality, as well as

increased vulnerability to develop anxiety and depression (Reuter & Hennig, 2005; Olsson et al., 2005). However, individuals with the val/val variant are more likely to express their anger inwardly while individuals with the met/met variant are more likely to express their anger outwardly (Rujescu, Gieglin, Gietl, Hartmann, & Möller, 2003).

Individuals with the val/met and met/met haplotypes possessed better reading related skills such as phonological awareness, decoding, spelling, and overall, were better readers. MET variants had increased frontal lobe function, as well as increased activity in the occipitotemporal junction and left temporal region and prefrontal regions; MET variants have also shown higher executive functioning in the Wisconsin Card Sorting Test than the VAL homozygotes (Landi et al., 2013; Rosa et al., 2004). Conversely, the VAL allele has been associated with decreased cognitive control, working memory, and poorer performance in prefrontal tasks (Dumontheil et al., 2011; Barnett, Jones, Robbins, & Muller, 2007). Depending on the genetic polymorphism an individual possesses, they may be predisposed to emotional, psychological, and cognitive vulnerabilities that may be exacerbated by being victimized by peers. Simply, the genetic variants of COMT have a dyadic relationship. Mier, Kirsch, and Meyer-Lindenberg (2010) conducted a meta-analysis in which they found strong association between COMT genotypes and prefrontal cortex activation. Specifically, they found the MET allele had strong effects for executive cognition while the VAL allele had opposing effects; conversely, the VAL allele had strong effects for emotionally stability while the MET allele had opposing effects. This is consistent with the widely accepted Warrior/Worrier model proposed by Goldman, Oroszi, & Ducci (2005) which states that the warrior haplotype, the VAL allele, leads to better stress resiliency but also leads to lower cognitive performance while the worrier haplotype, the MET allele, responds to a stressful or painful event with lower pain thresholds and stronger affective pain response;

however, the MET allele has higher cognitive functions. COMT allelic variants counterbalance one another such that gains in cognition are juxtaposed to losses in stress and anxiety resilience or vice-versa (Stein, Fallin, Schork, & Gelernter, 2005).

In conjunction with the Warrior/Worrier model, to examine COMT Genotype X Environment (GXE) interactions it may be better to use a differential susceptibility model rather than the diathesis-stress model (Kochanska, Kim, Barry, & Philibert, 2011). Traditionally, the diathesis-stress model states that genetic variants make individuals more vulnerable or risk prone to environmental influences. Concerning peer victimization and COMT genotypes, the diathesis-stress model suggests that individuals with the MET allele may be more vulnerable to the effects of peer victimization and individuals with the VAL allele may be less, but still, vulnerable to this environmental cues. However, the differential susceptibility model states genetic variants cause individuals to be more malleable or susceptible to both positive and negative influences. In this regard, the differential susceptibility model suggests that individuals with the MET allele may be more susceptible to the adverse effects of peer victimization, but may also be have better outcomes when not victimized than individuals without the MET allele. Similarly, individuals with the VAL allele may be less susceptible or not affected by peer victimization; such that val/val variants may have a buffering effect on some of the negative psychosocial outcomes associated with peer victimization, but may also not benefit as much from more positive peer relationships (Iyer, Liegey Dougall, & Jensen-Campbell, 2013).

Current Study

Past research has established the link between peer victimization and deleterious physiological and psychosocial outcomes. However, few studies have examined genetic predispositions that may cause some individuals to be more vulnerable

or susceptible to the harmful effects of peer victimization. To further explore this idea, this proposed thesis seeks to assess the genetic contribution of the COMT gene on the influence of peer victimization on stress reactivity and health outcomes relationship. This biological marker has been linked to the development of depression and anxiety, as well as a variety of behavioral diseases and biological functions.

For the present study, it is hypothesized that individuals who have at least one MET allele are more vulnerable to develop health problems after being victimized by peers. More specifically, this study explored the relationships between peer victimization and frequency of health problems, severity of health problems, and internalizing problems. Furthermore, this study examined how COMT genetic variants moderated these relationships. Second, it is hypothesized that the peer victimization by COMT allelic variants will have an effect on an individual's stress reactivity, as measured by cortisol during the TSST. Again, I anticipated that individuals with at least one MET allele will shower higher baseline cortisol, but a more blunted cortisol response when victimized compared to individuals who are homozygous for VAL or who are not bullied. Supplementary analyses will examine differences amongst genders, victimization types, and depression subtypes.

Aim 1: to examine whether being victimized by one's peers predicts greater internalizing problems, such as depression, withdrawn behaviors, and anxiety, as well as greater frequency and severity of health outcomes (Figure 1).

Aim 1a: to examine whether the relationships between peer victimization and internalizing problems and poor health outcomes are moderated by allelic variations of the COMT gene (Figure 2). For ease of analyses and consistent with previous research, COMT was coded into two groups: (1) ≥ 1 MET; and (2) VAL/VAL for the focal

hypotheses. Probing of simple effects associated with each COMT allele was examined following significant interactions.

Aim 1b: to examine whether gender influences the relationship between victimization and COMT to my outcome measures.

Aim 1c: Supplementary analyses explored whether types of victimization influence the relations proposed in Aim 1.

Aim 2: to examine whether being victimized by one's peers predicts altered cortisol reactivity to the Trier Social Stress Test. Specifically, within-subject differences in cortisol reactivity in the TSST as a function of time and between subject differences as a function of peer victimization were assessed.

Aim 2a: to examine whether COMT moderates the relationship between victimization and cortisol reactivity during the Trier Social Stress Test. In other words, three-way interactions between peer victimization, COMT genotypes, and stress reactivity were examined.

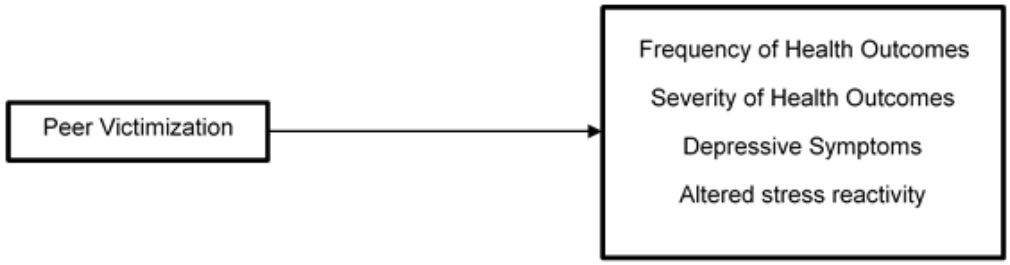

Figure 1. Victimization predicts negative health outcomes

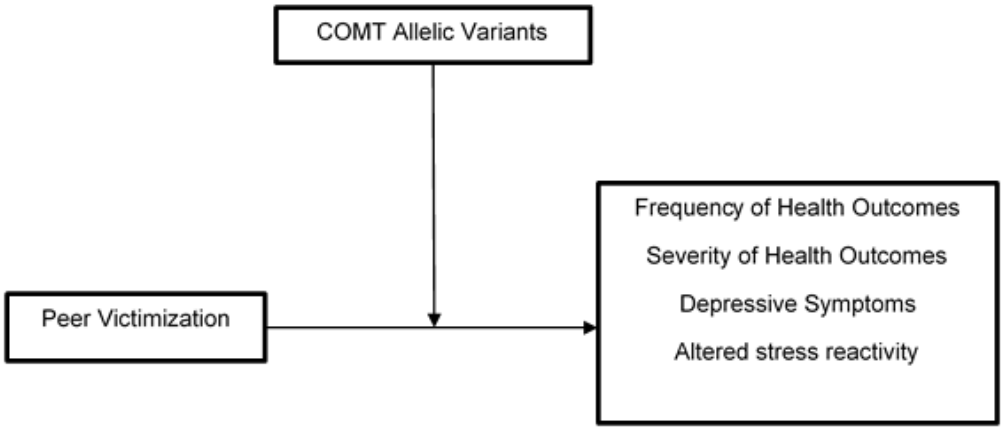

Figure 2. The proposed moderation model

Chapter 2

Method

Participants

Participants consisted of 203 adolescent boys and girls from the 5th through 8th grades in various area schools in the Dallas/Fort Worth metroplex. All participants were taking part in a larger ongoing study on peer relationships and health. Genetic variants, depression symptoms, victimization, and health outcomes were assessed concurrently. The sample consisted of 59.1% females with an average age of 12.17 years. The sample was ethnically diverse: 53% White or Anglo American, 22.1% Hispanic or Latino, 17.4% Black or African American, 3.4% Asian Indian, 1.3% American Indian or Alaskan Native, 1.3% Hawaiian or other Pacific Islander, 1.3% other.

Recruitment

To recruit participants, several methods were used. Adolescents were randomly selected from a mailing list obtained from the area schools and contacted for possible participation in a study on friendship, peer relationships, and health. Also, researchers visited area schools to explain the project in full, as well as various community organizations such as summer camps, dance and cheer academies, and mentoring programs in order to recruit participants. The Personality and Social Behavior lab utilized its website for recruitment through an interest form; those who expressed interest were contacted about participation in the study. Adolescents and parents were paid for their participation in the study.

Measures

Assessments of Victimization

Children's Self-Experiences Questionnaire—Self-Report (CSEQ-SR)

The CSEQ is a self-report measure that assessed the child's experience with peer relationships, specifically peer-related occurrences of victimization (Crick & Grotpeter, 1995). The questionnaire consists of three subscales which responses are recorded on a Likert scale, which ranges from 1 ("never") to 5 ("all the time"). Each subscale consists of five questions that measure the frequency of behaviors received. One scale measures overt victimization (such as "How often do you get pushed or shoved by another kid at school?"), one scale measures relational victimization (such as "How often do other kids leave you out on purpose when it is time to play or do an activity?"), and the frequency of receiving or being the target of prosocial behaviors (such as "How often do other kids let you know that they care about you?"). The CSEQ was also adapted to assess child's experiences with peer relationships from a parent-report.

Direct and Indirect Aggression Scale—Victim Version (DIAS-VS)

The victim version of the DIAS (Bjorkvist, Lagerspetz, & Osterman, 1992) assesses the frequency one experienced acts of aggression and/or victimization. Using a Likert scale with answers from 1 ("never") to 5 ("all the time"), the 24 item inventory assessed three different subscales: physical (such as "How often are you kicked by other classmates?"), verbal (such as "How often are you called names by other classmates?"), and indirect (such as "How often do classmates plan to secretly bother your child?"). Parents also completed the DIAS-VS in respect to their child's experiences.

Physical and Psychological Health Assessments

Child Behavior Checklist (CBCL) and Youth Self-Report (YSR)

The CBCL and YSR (Achenbach, 1991) assessed adolescents' social competency, emotional and behavior problems. Using a Likert scale with answers from 0 ("not true"), 1 ("somewhat or sometimes true"), and 2 ("very true or often true"), the 140 item inventory assessed several subscales: aggression, defiance, violence, attention problems, social problems, thought problems, somatic complaints, anxious/depressed, withdrawn, internalizing, externalizing, and total problems, and DSM-oriented scales. Parents also completed the CBCL and YSR in respect to their child's experiences. In this thesis, the subscales assessing anxious and withdrawn depression, internalizing problems, and somatic complaints were used.

Health Outcomes Survey

This survey assesses the frequency and severity of the child's health problems, with 14 questions on frequency and 14 questions on severity. Using a Likert scale, frequency items were measured from 0 ("not at all") to 4 ("all the time") and severity items were rated from 0 ("does not hurt at all") to 4 ("unbearable pain"). Health problems assessed included extreme fatigue, sleep problems, stomach aches, nausea, muscle aches and pains, headaches or migraines, weight gain or loss, low energy, trips to the nurse or doctors, and chest pain. Overall health is also included in the measurement and assessed from 0 ("extremely poor") to 5 ("excellent"). Parents completed the same measure as it pertains to the frequency and severity of their child's health.

Trier Social Stress Test (TSST)

The TSST is an experimental manipulation designed to assess stress reactivity. Adolescents were told that they would have ten minutes to prepare a speech concerning why they would make a good class president; adolescents were also told that a

committee would record the speech for further evaluation, take notes, and ask questions afterward. Four saliva samples were taken throughout the experiment to measure cortisol stress response (Knack, 2009). For this thesis, differences in cortisol stress reactivity were assessed across the four time points among levels of victimization.

Procedure

Participation in this study consisted of two phases; phase one had one session and took place at the child's school and phase two had two sessions that were conducted at the University of Texas at Arlington in the Personality and Social Behavior Lab. Due to the primary participant being an adolescent, a two-step consent process was utilized; parents gave consent for their child to participate and the minor gave assent to participate as well. During recruitment and when appointments were made, all participants were notified of the nature of the study, which focused on peer relationships and health outcomes in adolescence.

During phase one, adolescents completed a series of surveys that included assessments of victimization (CSEQ-SR; Crick & Grotepher, 1995; DIAS-VS; Bjorkqvist, Lagerspetz, & Osterman, 1992) and health outcomes at their school either in groups or online individually. As part of an overall continuing study measures of social support were also completed and weight, height, and waist circumference were also collected. However, these measures were not be analyzed in the present study.

During the first session of phase two, the participants, both parent and child came in to the Social and Personality Lab at the University of Texas at Arlington. During this session adolescents completed a second assessment of health outcomes and several questionnaires that assessed individual differences (CBCL; Achenbach, 1991; and Health Outcomes Survey). Measurements of personality and loneliness were also completed as part of an ongoing study, but will not be discussed in this thesis.

Simultaneously, parents completed a series of questionnaires that assess victimization, depression, and physical health as it pertained to their child online through Survey Monkey, including the DIAS-VS, CSEQ-SR, and HO; participants also completed other surveys that will not be part of this study. After the participants completed the assessments, salivary DNA was collected using the Oragene® DNA OG-500 (DNA Genotek Inc., Ontario, Canada) tubes as previously described; the parent and child were also instructed in salivary cortisol collection for the larger study; however, diurnal cortisol patterns will not be discussed in this current thesis.

For the second session of phase two, participants returned to laboratory at UTA approximately one week from the first session between 4:00pm and 7:00pm to control for diurnal cortisol patterns; the majority of participants began their sessions at either 4:00pm or 5:30pm. During this session, the parent completed several questionnaires about their child's personality, levels of depression, levels of loneliness, their child's social competency, and emotional and behavioral problems (CBCL & YSR; Achenbach, 1991). Again, measures of loneliness and personality will not be used in this thesis. Concurrently, adolescents were brought into an adjoining room where they rested for ten minutes in order to minimize potential HPA axis activation which could possibly increase cortisol levels. This potential increase in cortisol could confound later HPA axis activation during the TSST. After the ten minute period of rest, the adolescent was asked to provide the first saliva sample of the session.

Following, the adolescent was then led into another room where the TSST would occur. The room was set up such that when the adolescent walked into the room he/she was facing a table where two undergraduate research assistants sat in white lab coats. The research assistants were instructed to appear serious (i.e., did not smile or nod their heads, offered no reassurance, and kept a blank or neutral face). On the table, in front of

each research assistant was a clipboard and in between the research assistants was a video camera. At this point, the adolescent was told that he/she would have ten minutes to prepare a speech explaining to the committee why he/she would make a great class president. They were also told that the committee members would take notes concerning the content and delivery of the speech. The adolescent was also informed that each committee member was trained in behavioral observation and would be recording each behavior thusly. The adolescent was also informed that the speech would be videotaped so that it could be shown to their peers who would evaluate whether or not the adolescent would make an ideal class president. Furthermore, the task was summarized by notifying the adolescent that he/she should imagine they were invited to introduce themselves to a group of teachers who would also be evaluating their candidacy for class president. The speech was to last only five minutes. Adolescents were allowed to ask any questions before being led back to the preparation room.

In the preparation room, the adolescent was allowed to take whatever notes he/she wanted to for the speech, but the adolescent was informed that he/she would not be allowed to use the notes when giving the speech. Each participant was told to try very hard to make the speech believable since the committee members would ask additional questions in case there were disagreement about whether or not the adolescent would make a good class president or if they had any follow-up questions. The adolescent was reminded that the speech needed to be five minutes long and he/she was again given the chance to ask questions. The adolescents were left alone for ten minutes to prepare their speech, and were given a five minute warning halfway through. After ten minutes had passed, the second saliva sample was collected.

The adolescents were then led back into the experimental room. The video camera was turned on and the committee chair double checked that the participant was

in frame. The adolescent was told that he/she would be asked to state their identification number and then begin their speech. When the adolescent was speaking fluently, the committee members remained quiet. However, if the adolescent paused for more than twenty seconds, the committee chair said “You still have time, please continue.” If another ten seconds passed, the committee members began asking questions (e.g., what makes you a good leader?) until the five minutes ended. Once the adolescent finished his/her speech two follow-up questions (see Appendix B for list of questions) were asked regardless if the participant talked for the whole time or not. The committee chair then told the participant to wait while they completed an evaluation (see Appendix B for form). Then the adolescent was thanked for giving their speech and left the room.

The participant was then brought back to the resting room. A third saliva sample was collected at this time. The adolescent completed the post-TSST survey and the Youth Self Report (Achenbach, 1991). The fourth and final saliva sample was collected thirty minutes after the third sample. The parent and child participants were thoroughly debriefed and were paid for their participation.

DNA Analysis

DNA Collection: DNA was collected using Oragene® DNA OG-500 (DNA Genotek Inc., Ontario, Canada) tubes. The adolescent filled a tube marked with their specific identification number to the marked line with saliva via the passive drool method. Once completed, the researcher closed the funnel lid, which released a reagent into the saliva sample so that it can be stored at room temperature until extraction and analysis. The funnel lid was removed, the storage cap was screwed on the tube, and the sample was shaken for five minutes and stored in a biohazard marked container until analysis.

DNA Extraction: To extract the DNA, the sample was mixed by inversion and gentle shaken for a few seconds; this process ensured that the viscous samples was

properly mixed. Each sample was then incubated at 50°C for at least an hour which is a critical process that ensured that the DNA was adequately released and the nucleases were permanently inactivated. After incubation, 500µL of the sample was placed in a 1.5mL micro-centrifuge tube, to which 20µL of Prep-It L2P reagent was added and mixed via vortexing for a few seconds. The sample was incubated for ten minutes and then centrifuged at room temperature for five minutes at 15,000 x g. After this process, a pellet containing impurities formed; using a pipette tip, the clear supernatant was transferred to a new micro-centrifuge tube. In the new micro-centrifuge tube, 600µLof 95-100% ethanol at room temperature was added and then mixed gently by inversion ten times. To allow for full precipitation of DNA, the sample stood for ten minutes at room temperature undisturbed. The sample was then be placed in a known orientation into the microcentrifuge for two minutes at 15,000 x g. Upon formation of a DNA pellet, the remaining supernatant was removed with a pipette tip and discarded. The DNA pellet was washed with 250µLof 70% ethanol; the sample stood at room temperature for one minute then the ethanol was removed while taking care not to disturb the pellet. Then, 100µL solution of the DNA storage buffer, TE, was added to the micro-centrifuge tube to dissolve the pellet. The sample was then vortexed for at least five seconds.

DNA Genotyping: Samples were sent to DNA Genotek Inc. and processed using the 5’ exonuclease TaqMan Single Tube Assay technique for single nucleotide polymorphism (SNP) rs4680 on the COMT gene. The primer sequence for the val/met SNP was TCGAGATCAACCCCGACTGT (forward)/AACGGGTCAGGCATGCA (reverse) and the TaqMan probe sequence was 6FAM-CCTTGTCCTTCACGCCAGCGA/VIC-ACCTTGTCCTTCATGCCAGCGAAAT (Chen et al, 2004). The Taqman assay discriminated alleles using real-time polymerase chain reaction (RT-PCR) amplification and a pair of fluorescent dye detectors that targeted the single nucleotide polymorphism.

Specifically, one fluorescent dye was attached to the detector that matched the first allele, methionine, and a different dye was attached to the detector that matched the second allele, valine. Each sample was assessed for one of three genotypes: methioine, valine, and val^{158}met, such that those with homologous genotypes presented the color of the fluorescent dye specific to that variant and the heterologous variant presented a combination of the two fluorescent dyes specific to the two different variants. During the PCR, the polymerase released the fluorescent probe into the solution where it was detected using endpoint analysis in a Life Technologies, Inc. (Foster City, CA) 7900HT Real-Time instrument. Thermalcycling was performed with the first incubation step set at 95°C for 15 minutes; this was followed by 30 cycles of 95°C for 15 seconds and 58°C for one minute. In this sample, the genotype frequency for the COMT val^{158}met gene was 23.5% (*n* = 35) homozygous MET, 28.9% (*n* = 43), homozygous VAL, and 47.7% (*n* = 71) heterozygous val^{158}met (Table 1). The COMT genotypes were in Hardy-Weinberg equilibrium, meaning that the present sample's genetic distribution did not differ from what was expected in the population (*p* = .524).

Table 1. Descriptive statistics for COMT

COMT Alleles	Boys	Girls	Total
met/met	20(32.8%)	15(17.0%)	35(23.5%)
val^{158}met	24(39.3%)	47(53.4%)	71(47.7%)
val/val	17(27.9%)	26(29.5%)	43(28.9%)

Chapter 3

Results

Preliminary Analyses

Of the 202 participants, 53 (26.2%) saliva samples were not analyzed for COMT genotyping purposes. To determine whether these participants differed from the 149 individuals whose saliva samples were examined, a series of independent samples t-test were conducted. Results revealed that the two groups did not differ significantly in total victimization, depression, anxious/depressed, withdrawn/depressed, and internalizing problems. The two groups differed in the frequency of health outcomes; however, the participants whose saliva samples were not analyzed scored higher on this health outcome than those participants whose COMT genotype was known (Table 2). Therefore, since this thesis sought to examine the negative health outcomes associated with particular COMT allelic variants, the remainder of the statistical analyses were conducted using the 149 participants whose samples underwent COMT genotyping.

Table 2. *t*-Tests results for outcome measures comparing COMT and non-COMT

	COMT		No-COMT			
Measure	*Mean*	*SD*	*Mean*	*SD*	*t*	*p*
Total Victimization	-.05	.71	.17	.78	1.85	.067
Frequency of Symptoms	45.17	9.27	48.26	10.35	2.02	.045
Severity of Symptoms	37.98	7.65	40.07	8.72	1.65	.101
Depression	4.42	3.46	4.99	3.45	1.00	.318
Anxious/Depressed	5.46	4.84	6.47	4.96	1.26	.209
Withdrawn/Depressed	3.38	2.69	3.51	2.55	.299	.765
Internalizing Problems	12.61	9.27	14.22	8.87	1.06	.289
Somatic Complaints	3.80	3.26	4.33	2.67	1.01	.312

Descriptive statistics for self- and parent-reports for victimization measures can be observed in Tables 3. Descriptive statistics for health and internalizing measures can be found in Table 4.

Table 3. Descriptive statistics of self- and parent-report for measures of victimization

Measure	Range	Min.	Max.	*Mean*	*SD*	Skewness (Std. Error)	Kurtosis (Std. Error)
Total Victimization	3.49	-1.12	2.37	.004	.725	1.10(.20)	1.17(.40)
Self-Reports							
Overt	13.0	5.0	18.0	7.86	3.11	1.34(.20)	1.43(.40)
Relational	15.0	5.0	20.0	8.76	3.66	.872(.20)	.019(.40)
Physical	20.0	7.0	27.0	10.8	3.95	1.67(.20)	3.34(.40)
Verbal	17.3	5.0	22.3	10.4	4.34	.949(.20)	.340(.40)
Indirect	35.0	12.0	47.0	22.4	7.73	.798(.20)	-.090(.40)
Parent-Reports							
Physical	20.00	7.0	27.0	9.85	3.45	1.59(.20)	3.43(.40)
Verbal	18.00	5.0	23.0	9.66	3.55	1.10(.20)	1.84(.40)
Indirect	35.00	12.0	47.0	22.9	7.74	.699(.20)	.173(.40)

Table 4. Descriptive statistics of self- and parent-report for outcome measures

Measure	Range	Min.	Max.	*Mean*	*SD*	Skewness (Std. Error)	Kurtosis (Std. Error)
Self-Reports							
Frequency	45.00	28.0	73.0	45.17	9.27	.470(.20)	.001(.40)
Severity	36.30	28.0	64.3	37.98	7.65	.880(.20)	.324(.40)
Depression	16.0	.00	16.0	4.42	3.46	.895(.20)	.325(.40)
Anxious/ Depressed	20.0	.00	20.0	5.46	4.84	1.01(.20)	.311(.40)
Withdrawn /Depressed	12.0	.00	12.0	3.38	2.69	.786(.20)	.355(.40)
Internalizing Problems	40.0	.00	40.0	12.61	9.27	.858(.20)	.216(.40)
Somatic Complaints	16.0	.00	16.0	3.81	3.26	.963(.20)	1.05(.40)
Parent-Reports							
Frequency	33.00	28.0	61.0	41.95	7.43	.390(.20)	-.144(.40)
Severity	32.42	27.6	60.0	37.56	7.66	1.01(.20)	.707(.40)
Depression	11.5	.00	11.5	2.78	2.60	1.52(.20)	2.11(.40)
Anxious /Depressed	18.0	.00	18.0	3.31	3.51	1.79(.20)	3.84(.40)
Withdrawn /Depressed	12.0	.00	12.0	2.24	2.52	1.55(.20)	2.66(.40)
Internalizing Problems	31.0	.00	31.0	7.56	6.93	1.42(.20)	1.81(.40)
Somatic Complaints	10.0	.00	10.0	2.09	2.49	1.32(.20)	1.21(.41)

A series of bivariate correlations were conducted to examine the relationship between victimization subtypes; as expected, inter-correlations for all types of victimization were significant for both self- and parent-reports (Table 5). Significant correlations between self- and parent-reports were also found for physical, verbal, and indirect victimization subtypes (Table 6).

Table 5. Correlations of self- and parent-report of victimization

Measure	Overt	Relational	Physical	Verbal
Self-Report				
Overt	-			
Relational	.57**	-		
Physical	.69**	.36**	-	
Verbal	.71**	.55**	.70**	-
Indirect	.53**	.78**	.47**	.71**
Parent-Report				
Physical			-	
Verbal			.67**	-
Indirect			.53**	.81**

Note. * $p < .01$, ** $p < .001$

Table 6. Correlations of self- with parent- reports of victimization

	Parent-Report		
Measure	Physical	Verbal	Indirect
Self-Report			
Physical	.41**	.19**	.16**
Verbal	.36**	.33**	.30**
Indirect	.25**	.42**	.79**

Note. * $p < .01$, ** $p < .001$

A series of bivariate correlations were conducted to examine the relationships between internalizing problems. Inter-correlations for all measures (depression, anxious/depressed, withdrawn/depressed, and internalizing problems as well as somatic complaints) were significant for both self- and parent-report (Table 7). Significant correlations between self- and parent-reports of depression measures were also found (Table 8).

Table 7. Correlations of self- and parent- reports of depression

Measure	Depression	Anxious/ Depressed	Withdrawn/ Depression	Internalizing Problems
Self-Report				
Depression	-			
Anxious/Depressed	.96**	-		
Withdrawn/Depressed	.85**	.67**	-	
Internalizing Problems	.96**	.92**	.82**	-
Somatic Complaints	.61**	.57**	.53**	.80**
Parent-Report				
Depression	-			
Anxious/Depressed	.90**	-		
Withdrawn/Depressed	.81**	.47**	-	
Internalizing Problems	.96**	.88**	.75**	-
Somatic Complaints	.58**	.57**	.40**	.79**

Note. * $p < .01$, ** $p < .001$

Table 8. Correlations of self- with parent- reports of internalizing outcomes

Measure	Depression	Anxious/ Depressed	Withdrawn/ Depressed	Internalizing Problems	Somatic Complaints
Self-Report					
Depression	.24**	.17**	.26**	.23**	.11
Anxious/ Depressed	.20*	.19*	.16	.20*	.11
Withdrawn/ Depressed	.26**	.10	.39**	.23**	.10
Internalizing Problems	.25**	.18*	.27**	.25**	.16
Somatic Complaints	.19*	.14	.19*	.22**	.21*

Note. * $p < .01$, ** $p < .001$

As observed in Tables 5 and 6, victimization measures had significant inter-correlations between parent- and child reports ($r = 0.34 – 0.79$; $r_{mean} = 0.54$). As such, victimization measures were z-scored and self- and parent-reports were averaged together to create an overall measure of victimization. As seen in Tables 7 and 8,

although the average correlation between parent- and child- reports of internalizing problems was significant, the magnitude of that relationship was small, $r_{mean} = 0.275$ (7.6% of variance overlapped). Given the magnitude of the relationship between parent- and child- reports was small for internalizing problem measures and parent- reports showed a possible restriction of range (see Table 4), we examined parent- and child-outcome measures separately in all analyses. This is consistent with the literature that often finds parent and children may not agree on behaviors that are more covert in nature (e.g., internalizing problems) (Sourander, Helstela, & Helenius, 1999). Additionally, self- and parent-reports were averaged together to create an overall measure of depression as well as anxious and withdrawn subtypes, internalizing problems, and somatic complaints. Total victimization was significantly correlated with all self- and parent-report outcome measures (Table 9).

A series of bivariate correlations were conducted to examine the relationships between victimization and various health and depression outcomes by COMT allele type. Results showed that correlations were only significant for individuals with at least one MET allele for self-report measures (Table 10). To further explore this relationship bivariate correlations were conducted to examine gender; significant correlations were found for women with at least one MET allele (Table 11). The relationship between victimization, COMT, and gender will be assessed further using moderated multiple regression as part of Aim 1.

Table 9. Correlations of outcome measures with total victimization

Measure	Total Victimization
Self-Report	
Frequency	.45**
Severity	.40**
Depression	.34**
Anxious/Depressed	.30**
Withdrawn/Depressed	.35**
Internalizing Problems	.39**
Somatic Complaints	.37**
Parent-Report	
Frequency	.31**
Severity	.27**
Depression	.30**
Anxious/Depressed	.23**
Withdrawn/Depressed	.30**
Internalizing Problems	.32**
Somatic Complaints	.26**
Total	
Frequency	.48**
Severity	.42**
Depression	.41**
Anxious/Depressed	.35**
Withdrawn/Depressed	.39**
Internalizing Problems	.45**
Somatic Complaints	.40**

Note. * $p < .01$, ** $p < .001$

Table 10. Correlations of victimization with outcome measures for each allele type

Measure	met/met	val/met	val/val
Self-Report			
Frequency	.45**	.47**	.40*
Severity	.54**	.48**	.16
Depression	.52**	.34**	.20
Anxious/Depressed	.52**	.30*	.13
Withdrawn/Depressed	.45**	.33**	.28
Internalizing Problems	.54**	.44**	.20
Somatic Complaints	.42*	.54**	.13
Parent-Report			
Frequency	.08	.39**	.30
Severity	.08	.35**	.22
Depression	.17	.36**	.33*
Anxious/Depressed	.07	.27*	.34*
Withdrawn/Depressed	.27	.38**	.24
Internalizing Problems	.19	.37**	.35*
Somatic Complaints	.17	.23	.36*
Total			
Frequency	.36*	.57**	.41**
Severity	.41*	.55**	.21
Depression	.50**	.41**	.33*
Anxious/Depressed	.45**	.34**	.27
Withdrawn/Depressed	.44*	.42**	.31*
Internalizing Problems	.52**	.48**	.33*
Somatic Complaints	.41*	.48**	.24

Note. * $p < .01$, ** $p < .001$

Table 11. Correlations of victimization with self-report outcome measures by allele

	Male			Female		
Measure	met/met	val/met	val/val	met/met	val/met	val/val
N	20	24	17	15	47	26
Frequency	.60**	.55**	.50*	.35	.47**	.35
Severity	.65**	.47*	.40	.42	.48**	-.02
Depression	.36	.24	.30	.83**	.43**	.12
Anxious/Depressed	.41	.18	.29	.78**	.39**	.03
Withdrawn/Depressed	.25	.29	.27	.79**	.37*	.29
Internalizing Problems	.43	.37	.34	.77**	.52**	.09
Somatic Complaints	.52*	.53**	.35	.45	.62**	-.04

Note. * $p < .01$, ** $p < .001$

Aim 1: Peer Victimization Predicting Poor Health Outcomes Moderated by COMT Allelic Variants

First, I sought to examine whether being victimized by one's peers predicts higher levels of depressive symptoms, withdrawn and anxious depressive behaviors and internalizing problems as well as increased somatic complaints and frequency and severity of health outcomes. Concurrently, I also assessed whether these relationships were moderated by COMT allelic variants and the gender of the participant. For ease of analyses and consistent with previous research, COMT was coded into two groups: (1) ≥ 1 MET; and (2) VAL/VAL for the focal hypotheses. Probing of simple effects associated with each COMT allele was examined following significant interactions (Aiken & West, 1991). I also examined whether these relationships changed depending on the type of victimization. Self-, parent-, and combined-reports of the outcome measures were assessed in all analyses for completeness.

To assess whether being victimized predicted internalizing problems and poor health outcome measures and whether gender and COMT moderated these relationships both separately and combined, a series of moderated multiple regressions were conducted (Aiken & West, 1991). Internalizing outcome measures included overall

internalizing problems, anxious-depression, withdrawn-depression, and general depression. Health outcomes included somatic complaints, frequency of health problems, and severity of health problems. As stated previously, I examined these outcomes in three ways due to the small overlap between parent and child reports: (1) collapsed across parent- and child- reports; (2) child reports; and (3) parent reports. None of the proposed three-way interactions were significant with parent- or overall- (parent and child) reports included in the model. As such, results focused only on child-reports of health outcomes. As a continuous variable, overall victimization was centered. Both gender and COMT were coded using unweighted effects codes and the interaction terms between victimization, sex of the participant, and COMT genotype were created. Victimization, gender, COMT, and their cross-products (all two- and three- way interaction terms) were then entered into the equation.

Even after controlling for gender, COMT and their respective interactions with victimization, chronic victimization by one's peers still significantly predicted child-reports of poor health outcomes (Table C1). Additionally, there were significant interactions between COMT and victimization on child-reported severity of health outcomes, internalizing problems, and somatic complaints (Table C2).

To examine in which way COMT moderates the relationship between peer victimization and severity of health outcomes dummy codes were created for the two groups (VAL/VAL and ≥ 1 MET) with each serving as the base group; interaction terms were computed respectively. The interaction between COMT and victimization was probed with two additional regression models. The first regression model was conducted using at least one MET at the base group; overall, the relationship between victimization and severity of health outcomes was significant, $b = 4.05$, $SE = .69$, $t(142) = 5.87$, $p < .001$, $sr^2 = .44$. The second regression model conducted with val/val as the base group

was not significant, b = 1.09, SE = 1.03, $t(142)$ = 1.05, p = .294, sr^2 = .08, suggesting that individuals with at least one MET allele experience more severe health outcomes when victimized than non-victims (Figure 3).

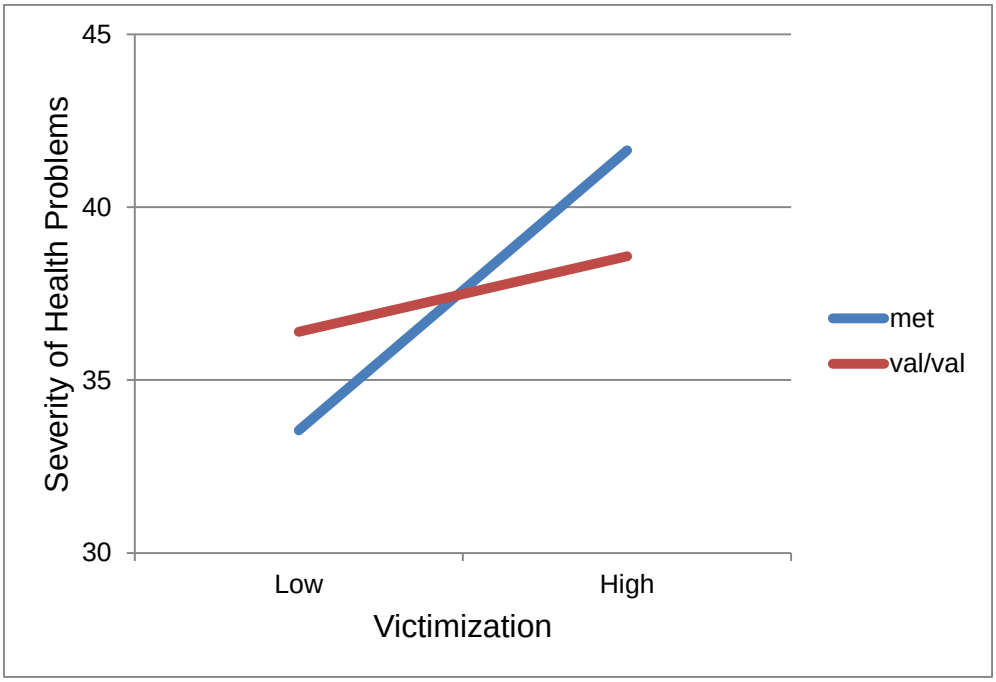

Figure 3. Participants' severity of health problems as a result of peer victimization based on allele variant

Similar results were observed for the relationship between peer victimization and internalizing problems. COMT allelic variants moderated this relationship such that individuals with at least one MET allele had a strong relationship between victimization and internalizing problems, b = 4.79, SE = .86, $t(138)$ = 5.57, p <.001, sr^2 = .42. The relationship was not significant for individuals with the val/val genotype, b = 1.71, SE = 1.25, $t(138)$ = 1.37, p = .174, sr^2 = .10 (Figure 4).

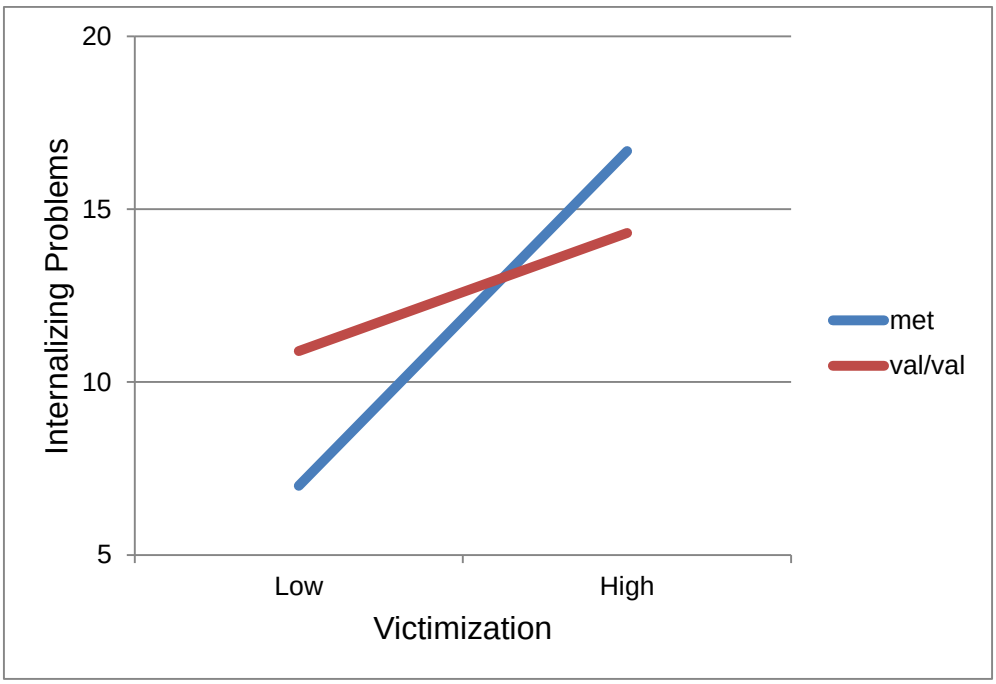

Figure 4. Participants' internalizing problems as a result of peer victimization based on allele variant

Additionally, COMT also moderated the relationship between peer victimization and somatic complaints. For individuals with at least one MET allele the relationship was significant, b = 1.72, SE = .29, $t(137) = 5.89$, p <.001, sr^2 = .43; However, for homozygous VAL individuals the relationship between victimization and somatic complaints was not significant, b = .358, SE = .43, $t(137) = .839$, p = .403, sr^2 = .06 (Figure 5).

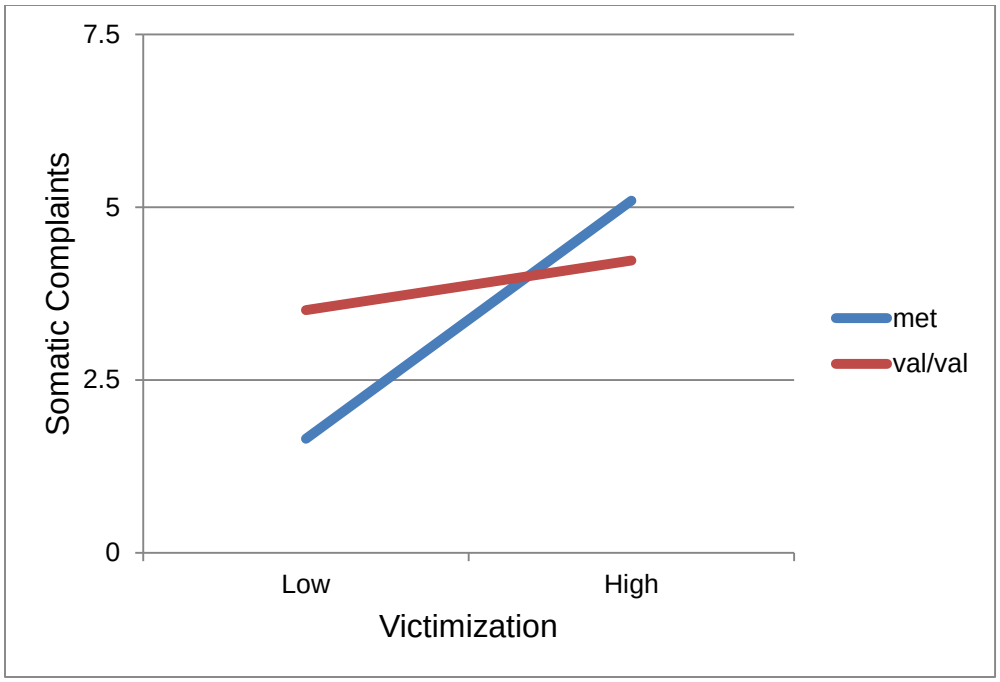

Figure 5. Participants somatic complaints as a result of peer victimization based on allele variant

Further, there was a significant three-way interaction between victimization, gender, and COMT on child-reported somatic complaints (Table C2). To examine this interaction, dummy codes were created both for COMT (VAL/VAL and ≥ 1 MET) and gender; interaction terms were computed respectively (e.g., at least on MET allele by boy by victimization). The interaction between COMT, gender, and victimization was probed with four additional regression models (Figure 6). The relationship between victimization and somatic complaints for boys with at least one MET allele was significant, b = 1.21, SE = .41, $t(134) = 2.97$, p = .004, sr^2 = .22; however, this relationship was stronger for girls with at least one MET allele, b = 2.21, SE = .41, $t(134) = 5.37$, p <.001, sr^2 = .39. The relationship between victimization and somatic complaints was not significant for individuals with the homozygous VAL allele: boys, b = .93, SE = .63, $t(134) = 1.46$, p = .146, sr^2 = .11, and girls, b = .15, SE = .57, $t(134) = -.25$, p = .800, sr^2 = .02. This finding

suggests that individuals with at least one MET allele are more susceptible to somatic complaints after being victimized by one's peers, especially if the target of victimization was a girl.

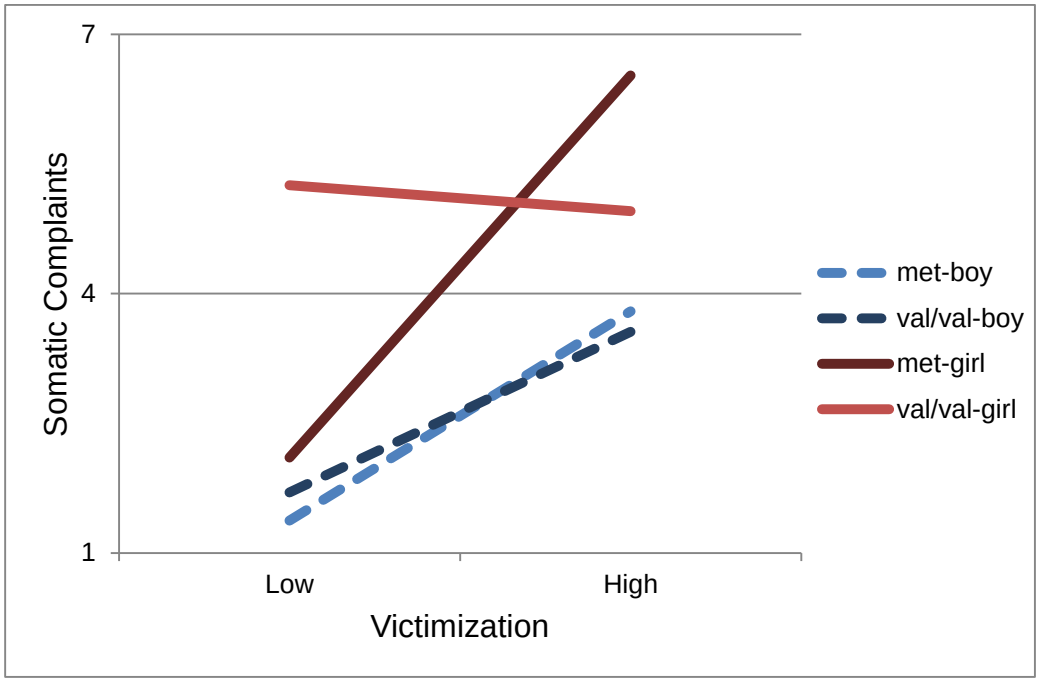

Figure 6. Participants' somatic complaints as a result of peer victimization based on gender and allele variant

To assess differences in victimization type (e.g. physical, verbal, and indirect) and whether each subtype predicts negative health outcomes, a series of moderated multiple regressions were conducted. Specifically, victimization type, COMT, and the interaction term were entered into the regression equation; for these analyses gender was used as a control. Significant interactions between COMT alleles and victimization subtype were probed further to examine differences. Results showed that while controlling for gender, physical, verbal, and indirect victimization significantly predicted negative health outcomes (Table C3).

Additionally, there were significant interactions amongst the victimization subtypes and COMT alleles on various health outcomes (Table C4). Specifically, there were significant interactions between physical victimization and COMT genotypes on severity of health outcomes and somatic complaints. Such that the relationship between physical victimization and severity of health outcomes was significant for individuals with at least on MET, $b = 4.44$, $SE = .82$, $t(144) = 5.43$, $p < .001$, $sr^2 = .41$, but not significant for individuals with the val/val genotype, $b = .22$, $SE = 1.48$, $t(144) = .073$, $p = .882$, $sr^2 = .01$. COMT also moderated the relationship between physical victimization and somatic complaints: the relationship was significant for individuals with at least one MET, $b = 1.63$, $SE = .36$, $t(139) = 4.58$, $p < .001$, $sr^2 = .35$, but not significant for homozygous VAL individuals, $b = .12$, $SE = .62$, $t(139) = .189$, $p = .851$, $sr^2 = .01$.

COMT allelic variants also moderated the relationship between indirect victimization and somatic complaints. Individuals with at least one MET allele had a stronger relationship, $b = 1.76$, $SE = .35$, $t(137) = 5.08$, $p < .001$, $sr^2 = .38$. The relationship was not significant for individuals with the val/val genotype, $b = .48$, $SE = .52$, $t(137) = .921$, $p = .333$, $sr^2 = .07$.

Of the three victimization subtypes (e.g. physical, verbal, and indirect), verbal victimization appears to have the most salient and consistent relationships between victimization and poor health outcomes as moderated by COMT allelic variants. Table 15 shows that COMT does moderate the relationships; individuals with at least one MET allele had strong relationships between verbal victimization and severity of health outcomes, anxious depression, internalizing problems, and somatic complaints. Homozygous VAL individuals did not have any significant interactions.

Table 12. Regression results for verbal victimization by COMT alleles

Predictor	*b*	SE_b	β	*t*	*p*	sr^2
Outcome						
Verbal X MET_COMT						
Severity	4.65	.85	.50	5.44	<.001	.41
Anxious/Depressed	2.87	.54	.49	5.31	<.001	.40
Internalizing Problems	6.20	1.00	.55	6.19	<.001	.45
Somatic Complaints	2.07	.35	.52	5.93	<.001	.43
Verbal X VAL_COMT						
Severity	1.07	1.32	.11	.82	.417	.06
Anxious/Depressed	.60	.83	.10	.73	.467	.06
Internalizing Problems	2.11	.153	.19	1.38	.170	.10
Somatic Complaints	.44	.53	.11	.83	.406	.06

Aim 2: Peer Victimization Predicting Stress Reactivity Moderated by COMT Allelic Variants

Secondly, I to examine whether COMT polymorphisms influenced the relationship between victimization and cortisol stress reactivity as assessed in the Trier Social Stress Test (TSST), salivary cortisol was assessed at: (1) 10 minutes after arrival/rest period; (2) 10 minutes after speech preparation; (3) immediately after delivering the speech; and (4) 30 minutes after giving the speech. As part of the analysis, I examined whether the link between victimization and cortisol reactivity differed for boys versus girls.

For this analysis, I conducted multi-level modeling (Snijders & Bosker, 2011; Raudenbush & Bryk, 2002) using a two level model, with variations in change parameters in cortisol levels within a child at level 1 and variation among children on levels of victimization at level 2. That is, nested within the adolescent are cortisol assessments associated with the TSST. The within-subject predictor was the time in which cortisol was taken during the TSST in minutes at intervals at 10, 30, 45, and 75 minutes. At the level of the child, I examined peer victimization, COMT (≥ 1 MET or VAL/VAL), and gender of

the child and their interactions. Peer victimization was treated as a continuous variable and centered. Both the gender of the participant and COMT were coded using unweighted effects codes as was previously done in the moderated multiple regression. There was an overall effect for peer victimization, b = -0.07, SE = 0.036, t(137) = -2.03, p = .04. Victimized children showed a blunted cortisol reaction compared to non-victimized children. That is, they exhibited less overall cortisol over the TSST lab task. There was also an overall gender X victimization interaction, b = -0.11, SE = 0.04, t(137) = -3.13, p = .002. Victimized girls exhibited lower levels of cortisol than non-victimized girls did during the lab task, b = -0.19, SE = 0.05, t(137) = -3.74, p < .001. There was no evidence that victimization was related to overall cortisol levels for boys, b = 0.04, SE = 0.05, t(137) = 0.76, p = .45.

There was an overall TSST time main effect, b = 0.052, SE = 0.02, t(427) = 2.67, p = .008. That is, cortisol levels increased over the lab task after controlling for victimization, COMT, Gender, and their respective product-terms; suggesting a successful implication of the TSST. As anticipated, there was a victimization X TSST Time interaction, b = -0.05, SE = 0.01, t(427) = -3.31, p = .001. Victimized children showed a blunted cortisol response during the stress reactivity task compared to non-victimized children. For illustration purposes, I created victim groups via cluster analysis and graphed their reactivity response during the TSST (Figure 7). More importantly perhaps, there was a COMT X victimization X TSST interaction, b = -0.03, SE = .01, t(427) = -2.01, p = .045. As seen in Figure 8, adolescents with low levels of victimization and val/val showed the typical cortisol reactivity response, namely an increase in cortisol during the stressful task, b = 0.008, SE = 0.002, t(427) = 3.35, p < .001. However, children with low levels of victimization and at least one MET showed a blunted response, b = 0.002, SE = 0.002, t(427) = 1.49, p = .136. There was no evidence that victimization

was related to changes in cortisol during the stressor for adolescents with high levels of victimization (b_s = 0.00, -0.00, SE_S = 0.00, 0.00, t_s = 0.21, -.30, p_s = 0.831, 0.768, for ≥ 1 MET and VAL/VAL respectively). There were no four-way interactions involving gender.

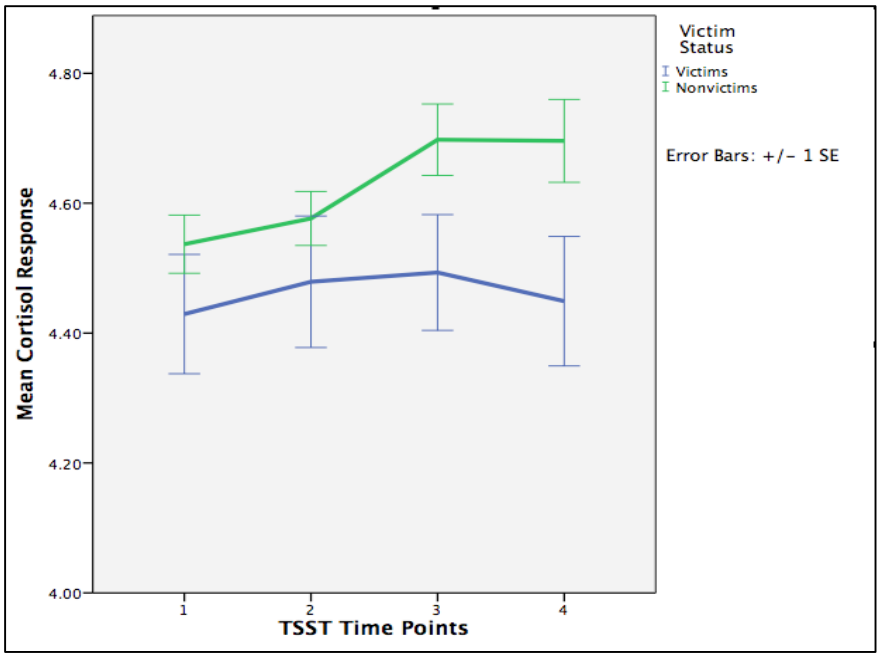

Figure 7. Participants' Stress Reactivity Response by Peer Victimization Status

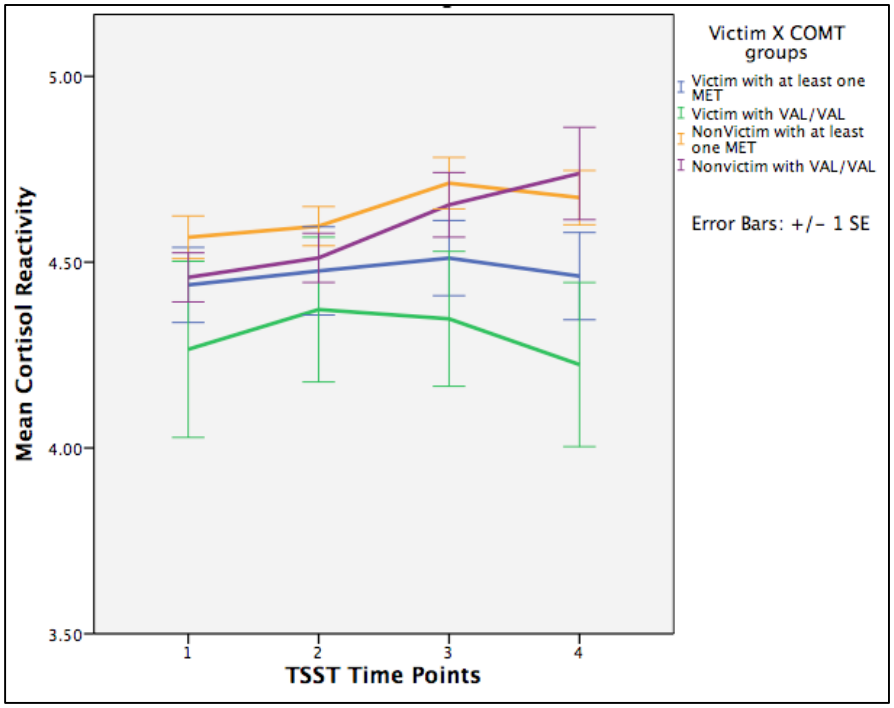

Figure 8. Participants' Stress Reactivity Response by Peer Victimization and COMT Status

Chapter 4

Discussion

This current thesis explored relationships between peer victimization, negative health outcomes, and altered stress reactivity. Specifically, the focus of this research was to explore how allelic variants of the COMT gene differentially influence an individual's susceptibility to internalizing problems, somatic complaints, and health symptoms. Concurrently, I examined whether victims of bullying report greater physiological and psychological health problems and whether these relationships were influenced by the COMT val^{158}met polymorphism. Specifically, I explored whether having at least one MET allele would lead to poorer outcomes; the influence of gender and subtypes of victimization were also examined. Secondly, I considered whether chronic peer victimization leads to altered stress reactivity to an acute social stressor using the Trier Social Stress Test. Further, I investigated whether the COMT val^{158}met polymorphisms moderate this relationship. This thesis analyzed the COMT val^{158}met genotype in a novel way, placing it in the context of peer victimization and poor health outcomes.

Preliminary analyses revealed that self- and parent- reports of overall victimization as well as physical, verbal, and indirect subtypes were strongly correlated with one another. Additionally, overall victimization was significantly correlated with all outcome measures examined in this thesis: frequency of health symptoms, severity of health symptoms, overall depressive symptoms, anxious/depressed symptoms, withdrawn/depressed symptoms, internalizing problems, and somatic complaints. Also, parent- and child-reports of these outcome measures were significantly yet moderately correlated with one another, therefore this thesis examined parent- and child-reports both separately and combined (Table 7, 8). Though parent- and combined-reports yielded some significant results, the relationships between peer victimization and outcome

measures were more prominent for child-reports. This is consistent with previous literature that suggests discrepancies between Achenbach's Child Behavior Checklist and Youth Self-Report measures exists, especially for internalizing problems that may be overlooked by adults (Sourander, Helstela, & Helenius, 1999). Further, in reference to themselves, children generally report higher levels of internalizing problems compared to their parents and may in fact be more accurate than their parents in assessing their own health related quality of life (van der Ende, Verhulst, & Tiemeier, 2012; Dey, Landolt, & Mohler-Kuo, 2013). Thusly, analyses were conducted and results were reported using child-reports of outcome measures.

As previous research suggested, peer victimization significantly predicted poorer health outcomes and increased internalizing problems in this study (Fekkes, Pijpers, Fredriks, Vogels, & Verloove-Vanhorick, 2006; Iyer-Eimerbrink, Scielzo, and Jensen-Campbell, under review). Additionally, physical, verbal, and indirect victimization subtypes also significantly predicted poorer health outcomes with the exception that those who experience physical victimization did not report higher levels of anxious/depressed symptoms. This finding is consistent with previous literature that suggests social rather than physical forms of victimization are more strongly associated with depression and anxiety (Calhoun, Helms, Heilbron, Rudolph, Hastings, & Prinstein, 2014).

No study has been published examining the genetic influences of the COMT gene on the relationship between victimization and negative health outcomes. Therefore it is notable that this thesis found that COMT moderated the relationships between peer victimization and severity of health symptoms and somatic complaints; such that these relationships were stronger for individuals with at least one MET allele. Past research by Diathchenko and colleagues (2005, 2006) found that homozygous MET and

heterozygous val/met individuals show increased susceptibility to both perceived and quantifiable sensitivity to pain and noxious stimuli. Past research has also showed though physical and social pain are unique constructs, each may rely on the same neurobiological substrates such as the dorsal anterior cingulate cortex and anterior insula, areas involved in the affective processing of pain. Simply, physical and social pain may be processed similarly within the brain (see Eisenberger, 2012 for a review; Eisenberger, Lieberman, & Williams, 2014).

Therefore viewing peer victimization as a form of social pain and accepting that COMT MET carriers are overall more sensitive to painful stimuli, it is understandable that these individuals would rate their health symptoms more severe and report higher somatic complaints. Further, given that MET carriers have been shown to be more emotionally sensitive and vulnerable to develop anxiety and depression, it was not surprising that this thesis found COMT allelic variants to moderate the relationship between peer victimization and internalizing problems (Drabant et al., 2006; Olsson et al., 2005). Specifically, MET carriers reported more internalizing problems after being victimized.

Moreover, there was a significant three-way interaction between peer victimization, COMT val^{158}met polymorphisms, and gender on somatic complaints. The saliency of this relationship was expected considering that peer victimization has been shown to predict anxious and withdrawn depression symptoms as well as internalizing problems in this thesis and in previous research; in turn, research has shown that these variables lead to increased and more severe somatic complaints (Ruchkin & Schwab-Stone, 2014). Furthermore, for individuals with at least one MET allele the relationship was significant, although stronger for girls than boys; which may be driven by the notion that girls are more affected by being bullied than boys (Iyer-Eimerbrink, Scielzo, &

Jensen-Campbell, under review). The relationship was not significant for homozygous VAL boys nor girls. As observed in Figure 6, it appears that both boys with homozygous VAL and with at least one MET allele report somatic complaints at similar rates regardless of the intensity of bullying. Comparatively, overall girls reported higher levels of somatic complaints, a trend seen throughout the literature (Barsky, Jones, Robbins, & Muller, 2007; Delisle, Beck, Dobson, Dozois, & Thombs, 2012). Though future research should assess regions of significance to determine at what point differences are observed; particularly for boys, it may be that genetic influences of the COMT gene are more striking at the extreme ends of the victimization continuum, +/- 2 *SD*s. Nevertheless, girls with at least one MET allele appear to be more susceptible to the effects of peer victimization compared to girls with the val/val variant. The observed gender differences may be due to the sexually dimorphic nature of the COMT gene. In a review by Harrison and Tunbridge (2008), COMT was found to differentially impact the brain function as well as psychiatric dysfunction of men and women. Specifically for individuals with at least one MET allele, harm avoidance, episodic anxiety, and panic disorders were more highly associated with women while obsessive-compulsive disorder and suicidality were associated with men.

No previous studies examined the differential susceptibility of the COMT gene on victimization subtypes, as such this thesis sought to examine how COMT val^{158}met polymorphisms moderate the relationships between victimization subtypes (e.g., physical, verbal, and indirect) and various poor health outcomes. Results showed that physical victimization interacted with COMT to predict more severe health symptoms and higher levels of somatic complaints for individuals with at least one MET allele. Indirect victimization also predicted increased somatic complaints for the MET group. Given that val/met and met/met alleles are associated with increased sensitivity to pain, physical

victimization may have a greater effect on those individuals (Diatchenko et al., 2005). Interestingly, verbal victimization predicted a variety of poor outcomes for individuals with at least one MET such as severity of health symptoms, anxious depressed, internalizing problems, and somatic complaints (see Table 15). Suggesting again that the MET allele of the COMT gene may be linked to the affective experience of social pain (Masten et al., 2009). Research has shown that social pain is more easily recalled and re-experienced than physical pain (Chen & Williams, 2012). Therefore, the effects of peer victimization may be compounded for individuals with at least one MET. For future research, it would be valuable to explore the role gender plays in these relationships and assess victimization subtypes using cluster analyses to form unique victimization groups in addition to treating victimization as a continuous variable.

Similar to the findings of Ouellet-Morin and colleagues (2011), the thesis found children who are chronically victimized by their peers have a blunted cortisol response during the Trier Social Stress Test. Further, results revealed a gender by victimization interaction effect on cortisol with in the laboratory experiment; victimized girls had lower overall cortisol compared to non-victimized girls, there were no differences for boys, regardless of victimization. Gender differences in HPA-axis dysregulation and stress reactivity, specifically for girls, may be due to the fact that women tend to be more vulnerable to and subjectively experience stress to a greater extent than men (Kudielka, Buske-Kirchbaum, Hellhammer, & Kirschbaum, 2004). Also, in animal studies, it has consistently been shown that women produce higher levels of glucocorticoids after activation of the HPA axis; though in human studies the relationship is not as clear (Jabbi et al., 2007; for review, Kudielka & Kirschbaum, 2005; Yoshimura et al., 2003). Therefore, considering that girls are more sensitive to stress and produce more hormones in

response to a stressor, the presence of a chronic stressor like peer victimization is likely to affect them to a greater extent.

After controlling for victimization, gender, and COMT as well as their interaction terms, there was an overall effect throughout the four time points of the TSST. There was also a significant interaction between victimization and time; overall victims displayed a blunted cortisol response compared to non-victims (see Figure 7). This is consistent with previous findings that showed individuals with a history of maltreatment or victimization had altered stress reactivity (Harkness, Stewart, and Wynne-Edwards, 2011; Knack, Jensen-Campbell, and Baum, 2011). As of now, no published study has examined the moderating role of COMT in the relationship between HPA dysregulation observed in victimized individuals. For that reason, it is interesting that this thesis found a significant interaction between peer victimization, COMT, and cortisol reactivity in the TSST. Individuals with a history of low victimization who were homozygous VAL showed a typical stress response; individuals with at least one MET, showed a blunted cortisol reactivity response. Though this is contrary to previous research which suggests that homozygous MET individuals have higher cortisol response compared to other COMT genotypes, this suggest that at low levels of victimization COMT polymorphisms play a role in the relationship between peer victimization and stress responses (Armbuster et al., 2012; Jabbi et al., 2007). I also found no evidence for a four-way interaction including gender in the relationship. Since these relationships are novel, future research should examine these the roles COMT, gender, and peer victimization as it pertains to acute social stressors thoroughly. Also, poor health outcomes, internalizing problems, and somatic complaints should be included in future analyses.

Although this thesis established a novel gene by environment interaction, there are limitations worth noting. For ease of analyses though consistent with prior literature,

this study examined the COMT val^{158}met polymorphism using two groups: homozygous VAL and at least one MET. This allowed me to have enough power to run the more complex interactions and linear modeling analyses. However in the future, a larger sample would be needed to explore the COMT gene using three groups: met/met, val/met, and val/val; doing such would allow for a better overall understanding of the COMT gene and would capture more subtle and nuanced effects it has on peer victimization outcomes. Also, previous research has shown that the COMT gene is dyadic in nature and that though one allelic variant made lead to poor outcomes in one domain, it may serve as a buffer or even lead to better outcomes in another. Hence, I recommend that future research considers the positive effect the val/val allelic variant may have on peer victimization outcomes. It is also important to consider that the analyses in this thesis were conducted using cross-sectional data; therefore, assumptions about causality should be tempered. In the future, it would be beneficial to assess these relationships longitudinally to establish causation and determine the persistence of the negative outcomes observed. Additionally, the lack of research focusing on COMT and peer victimization allows for other avenues to be explored. Future researcher should focus on other potential candidate genes, their interaction with COMT, and the development of possible genetic profiles that would make individuals more susceptible to poor health outcomes, internalizing problems, and somatic complaints.

Regardless of these limitations, this thesis was in line with the breadth of prior research establishing the link between peer victimization and negative psychological and physiological outcomes. This thesis also added to the field of peer victimization research by exploring a possible genetic influence that makes individual more susceptible to negative health outcomes. As bullying continues to garner national and social media attention (see Bezos, 2014), it important to emphasize that peer victimization is not a

normative adolescent experience but rather one with serious mental and physical consequence. This present study provided preliminary evidence of genetic influences on the expression of internalizing problems, the development of somatic complaints and altered stress reactivity, helping to explain why some children are more negatively affected by peer victimization than others.

Appendix A

Self- and Parent-Report Survey Measure

"Things that Happen to Me at School"
Children's Self-Experiences Questionnaire, Self-Report
(CSEQ-SR; Crick & Grotpeter, 1995)

Directions: Here is a list of things that sometimes happen to kids at school. How often did they happen to you while you were at school? Bubble in the circle that best describes your experiences at school.

Scale

1 = never	3 = sometimes	5 = all the time
2 = almost never	4 = almost all the time	

At school other kids make fun of me.
At school I get hit and pushed by other kids.
I get picked on by other kids at school.
I get beat up by other classmates.
I am ignored by other classmates when someone is mad at me.
I do not get invited to things (e.g., parties) because my friends sometimes don't like to include me.
I get left out of things when someone is mad at me or wants to get back at me for something.
Other kids tell rumors about me behind my back.
I am very strong.
If I were in an arm wrestling contest, I would win.
I make fun of people.
I hit and push others around.
I tell lies.
I sometimes take things that belong to someone else.
I make noise or bother others in class.
I do not follow the rules.
I act like a baby.
I get upset when called on to answer questions in class.
I complain a lot and nothing makes me happy.
I try to get other kids to play with me even when they don't want to.
On the playground, I just stand around.
I don't talk much.
I am afraid to do things.
I seem unhappy and look sad often.
When other kids are playing, I watch them but don't join in.
In a group, I share things and give other people a turn.
I am always friendly.
I am always willing to help my classmates.
I try to cooperate with my classmates.

Direct and Indirect Aggression Scale – Victim Version
(DIAS-VS; Bjorkvist, Lagerspetz, & Osterman, 1992)

Directions: Answer each question by bubbling in the answer which seems to most closely tell you about how your classmates behave toward you.

Scale

1 = never	3 = sometimes	5 = very often
2 = seldom	4 = quite often	

How often are you hit by other classmates?
How often are you shut out of the group by other classmates?
How often do other classmates yell at you or argue with you?
How often do classmates become friends with another classmate as a kind of revenge?
How often are you kicked by other classmates?
How often are you ignored by other classmates?
How often are you insulted by other classmates?
How often do classmates who are angry with you gossip about you?
How often are you tripped by other classmates?
How often do classmates tell bad or false stories about you?
How often do classmates say they are going to hurt you?
How often do classmates plan to secretly bother you?
How often are you shoved by other classmates?
How often do classmates say bad things about you behind your back?
How often are you called names by other classmates?
How often do classmates tell others “Let’s not be friends with him/her!”?
How often do other classmates take things from you?
How often do classmates tell your secrets to a third person?
How often are you teased by other classmates?
How often do classmates write small notes where you are criticized?
How often are you pushed down to the ground by other classmates?
How often do other classmates criticize your hair or clothing?
How often do other classmates pull at you?
How often do classmates who are angry with you try to get others to dislike you?

Assessing Health Outcomes – Parent Report

Directions: Rate the frequency and severity of the following health symptoms.
Scale:

Frequency:	not at all	sometimes	often	all the time
Severity:	does not hurt at all	hurts a little	hurts a lot	unbearable pain

Extreme fatigue (feeling extremely tired)
Allergic reaction
Sleep problems
Stomach ache
Nausea/vomiting (sick to your stomach/throwing up)
Diarrhea
Muscle aches and pains
Headaches or migraine
Weight gain of 5 or more pounds
Weight loss of 5 or more pounds
Respiratory congestion (cold in your chest)
Runny nose
Coughing
Sore throat
Sneezing
Blocked nose
Fever or chills
Dizziness
Double or blurred vision
Trouble catching breath
Having a cold
Chest pains
Numbness or tingling
Low energy
Ear infections
Getting sick
Heart beating too fast
Visits to the doctor
Visits to the school nurse
When your child was an infant, how frequently did he/she get sick?
Does your child smoke? (yes/no)
When did your daughter begin menarche (i.e., her first menstrual cycle)?
Is your child currently taking any medication? Yes/no

If so, please note what medication (or what the medication is for, e.g., ADHD, seizures)
_____

Assessing Health Outcomes – Child Report

Directions: Rate the frequency and severity of the following health symptoms.
Scale:

Frequency:	not at all	sometimes	often	all the time
Severity:	does not hurt at all	hurts a little	hurts a lot	unbearable pain

Extreme fatigue (feeling extremely tired)
Allergic reaction
Sleep problems
Stomach ache
Nausea/vomiting (sick to your stomach/throwing up)
Diarrhea
Muscle aches and pains
Headaches or migraine
Weight gain of 5 or more pounds
Weight loss of 5 or more pounds
Respiratory congestion (cold in your chest)
Runny nose
Coughing
Sore throat
Sneezing
Blocked nose
Fever or chills
Dizziness
Double or blurred vision
Trouble catching breath
Having a cold
Chest pains
Numbness or tingling
Low energy
Ear infections
Getting sick
Heart beating too fast
Visits to the doctor
Visits to the school nurse

Appendix B

Trier Social Stress Test Questionnaires

Questions to Ask During the Trier Social Stress Test

Do you participant in extracurricular activities? Do you have enough time to devote to being class president? How will you juggle extracurricular activities (e.g., sports, drama club, band, etc.) and being class president?
Would you feel comfortable meeting with teachers or the principal to discuss issues that are important to your class? How might you communicate with teachers and/or the principal?
If you were elected class president, what would your first order of business be (i.e., what would you do first)? Why?
Do you think you are a good leader? Have you had any experience being a leader? What makes you a good leader? How will past leadership experience help you as class president?
What qualities about yourself would help you be a good class president? (e.g., good leader, get along w/ classmates well, etc)
How important do you think it is for the class president to help promote school spirit? Do you have any ideas about how to increase or maintain school spirit?
Do you have any ideas for fundraisers for your class? As class president, how would you like to use class funds/money?
Students often complain about cafeteria food. Do you think cafeteria food is a problem at your school? Is there anything you could do as class president to help improve the food?
Do you think there needs to be more variety in the food choices available in your cafeteria? Would you as class president be able to do anything to increase the choices students have?
As class president, would you do anything to help students who have difficulty doing well in their classes? What might you do?
Are there things in your school you'd like to change?

Form to Mark on While Participant Gives Speech

Note: This form should be filled in as the participant gives his/her 5-minute speech. I am not so concerned with what you are actually writing; rather I am interested in keeping with the cover story in which the participant thinks you are coding both the content of their speech and their nonverbal behavior. Below are a series of questions; following the questions is the time during the speech when I want you to record an answer for it.

Committee Chair: "Please state your ID."
Participant ID: _____
(when stated)

Is the participant: MALE FEMALE (30seconds)

Is the participant using a lot of "umms" (or the like)? YES NO (60seconds)

What has the participant told you his/her reason for being ideal president is?(90seconds)

_____

Does the participant seem nervous? YES NO (180 seconds)

Ask two follow-up questions from list (if ran full time) when finished;
if short, ask questions until time is up, then ask two follow-up questions

Committee Chair: "Please give us just a minute to finish our evaluations."

Quickly complete (to further support cover story that you're evaluating him/her):

Did participant take the entire 5 minutes? YES NO

Rate the overall quality of the speech

1	2	3	4	5
Very poor		Average		Excellent

How convincing was this participant that he/she would make an ideal class president?

1	2	3	4	5
Not at all		Average		Extremely

Did the participant convince you that he/she would make an ideal class president?
YES NO

Committee Chair: "Okay, I think we have enough information to make our decision. Thank you for speaking with us today."

Appendix C

Regression Results for Aim 1

Table C1. Regression results for victimization, gender, and COMT predicting poor health outcomes

Predictor	*b*	SE_b	β	*t*	*p*	sr^2
Outcome						
Victimization						
Frequency	3.95	.74	.429	5.35	<.001	.39
Severity	2.55	.62	.335	4.11	<.001	.30
Depression	1.11	.30	.318	3.75	<.001	.29
Anxious/Depressed	1.30	.42	.266	3.12	.002	.24
Withdrawn/Depressed	.93	.24	.342	3.94	<.001	.31
Internalizing Problems	3.31	.76	.354	4.36	<.001	.32
Somatic Complaints	1.05	.26	.322	4.08	<.001	.30
Gender						
Frequency	2.53	.77	.272	3.30	.001	.24
Severity	1.63	.65	.212	2.53	.013	.19
Depression	.55	.31	.155	1.77	.078	.14
Anxious/Depressed	1.05	.43	.212	2.42	.017	.19
Withdrawn/Depressed	.044	.24	.016	.18	.856	.01
Internalizing Problems	2.10	.79	.222	2.66	.009	.20
Somatic Complaints	1.05	.27	.319	3.92	<.001	.28
COMT						
Frequency	-.47	.77	-.046	-.61	.546	.04
Severity	-.24	.65	-.029	-.38	.708	.03
Depression	.10	.31	.025	.31	.755	.02
Anxious/Depressed	.15	.43	.027	.34	.737	.03
Withdrawn/Depressed	.05	.24	.016	.19	.848	.02
Internalizing Problems	.36	.79	.035	.45	.653	.03
Somatic Complaints	.21	.27	.059	.78	.438	.06

Table C2. Regression results for interaction terms predicting poor health outcomes

Predictor	*b*	SE_b	β	*t*	*p*	sr^2
Outcome						
Victimization X Gender						
Frequency	.17	.74	.018	.23	.819	.02
Severity	-.55	.62	-.072	-.88	.378	.07
Depression	.20	.30	.058	.69	.494	.05
Anxious/Depressed	.23	.42	.046	.54	.590	.04
Withdrawn/Depressed	.18	.24	.067	.78	.439	.06
Internalizing Problems	.43	.46	.045	.56	.577	.04
Somatic Complaints	-.02	.26	-.005	-.06	.950	.01
Victimization X COMT						
Frequency	-.67	.74	-.072	-.91	.365	.07
Severity	-1.43	.62	-.186	-2.30	.023	.17
Depression	-.43	.30	-.123	-1.46	.146	.11
Anxious/Depressed	-.71	.42	-.143	-1.69	.093	.13
Withdrawn/Depressed	-.16	.24	-.060	-.69	.490	.06
Internalizing Problems	-1.50	.76	-.159	-1.97	.051	.15
Somatic Complaints	-.66	.26	-.201	-2.56	.011	.19
Gender X COMT						
Frequency	.63	.77	.068	.82	.414	.06
Severity	1.08	.65	.143	1.68	.095	.12
Depression	-.07	.31	-.020	-.23	.822	.02
Anxious/Depressed	.05	.43	.009	.11	.916	.01
Withdrawn/Depressed	-.19	.24	-.069	-.76	.450	.06
Internalizing Problems	.001	.79	.000	.001	.999	.00
Somatic Complaints	.19	.27	.058	.70	.487	.05
Victimization X COMT X Gender						
Frequency	-.31	.74	-.033	-.42	.678	.03
Severity	-.73	.62	-.095	-1.17	.243	.09
Depression	-.44	.30	-.124	-1.47	.144	.11
Anxious/Depressed	-.64	.42	-.130	-1.53	.128	.12
Withdrawn/Depressed	-.24	.24	-.086	-1.00	.319	.08
Internalizing Problems	-1.36	.76	-.159	-1.97	.076	.13
Somatic Complaints	-.52	.26	-.159	-2.01	.046	.15

Table C3. Regression results for victimization types predicting poor health outcomes

Predictor	*b*	SE_b	β	*t*	*p*	sr^2
Outcome						
Physical Victimization						
Frequency	3.85	1.01	.348	3.88	<.001	.32
Severity	2.33	.85	.255	2.74	.007	.21
Depression	.92	.41	.219	2.23	.027	.18
Anxious/Depressed	.92	.57	.158	1.61	.110	.13
Withdrawn/Depressed	.91	.32	.280	2.85	.005	.23
Internalizing Problems	2.67	1.07	.238	2.50	.014	.20
Somatic Complaints	.87	.36	.222	2.42	.017	.18
Verbal Victimization						
Frequency	4.32	.93	.380	4.64	<.001	.18
Severity	2.87	.79	.305	3.65	<.001	.36
Depression	1.43	.36	.339	4.04	<.001	.38
Anxious/Depressed	1.74	.49	.294	3.52	.001	.29
Withdrawn/Depressed	1.13	.29	.344	3.95	<.001	.34
Internalizing Problems	4.16	.91	.367	4.55	<.001	.40
Somatic Complaints	1.26	.32	.316	3.94	<.001	.33
Indirect Victimization						
Frequency	3.90	.92	.366	4.25	<.001	.32
Severity	2.28	.78	.259	2.92	.004	.23
Depression	1.09	.36	.270	3.03	.003	.24
Anxious/Depressed	1.38	.50	.246	2.77	.006	.22
Withdrawn/Depressed	.79	.29	.253	2.78	.006	.23
Internalizing Problems	3.34	.92	.311	3.62	<.001	.28
Somatic Complaints	1.12	.32	.299	3.52	.001	.26

Table C4. Regression results for victimization type by COMT

Predictor	*b*	SE_b	β	*t*	*p*	sr^2
Outcome						
Physical X COMT						
Frequency	-1.33	1.00	-.120	-1.33	.185	.10
Severity	-2.11	.84	-.230	-2.51	.013	.19
Depression	-.26	.41	-.062	-.64	.524	.01
Anxious/Depressed	-.50	.57	-.085	-.88	.379	.07
Withdrawn/Depressed	-.02	.32	-.005	-.06	.956	.05
Internalizing Problems	-1.30	1.06	-.115	-1.23	.221	.10
Somatic Complaints	-.76	.36	-.192	-2.12	.036	.16
Verbal X COMT						
Frequency	-.96	.93	-.084	-1.04	.302	.08
Severity	-1.79	.79	-.189	-2.28	.024	.17
Depression	-.64	.36	-.150	-1.80	.074	.14
Anxious/Depressed	-1.14	.49	-.191	-2.30	.023	.17
Withdrawn/Depressed	-.14	.29	-.043	-.50	.622	.04
Internalizing Problems	-2.05	.91	-.180	-2.24	.027	.16
Somatic Complaints	-.81	.32	-.203	-2.55	.012	.18
Indirect X COMT						
Frequency	-.01	.90	-.001	-.01	.993	.00
Severity	-1.21	.76	-.136	-1.58	.117	.12
Depression	-.57	.35	-.140	-1.62	.107	.13
Anxious/Depressed	-.82	.49	-.145	-1.68	.094	.13
Withdrawn/Depressed	-.31	.28	-.100	-1.13	.262	.09
Internalizing Problems	-1.73	.90	-.159	-1.91	.058	.15
Somatic Complaints	-.64	.31	-.169	-2.06	.042	.15

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Biographical Information

Erin Boyd is a native of Fort Worth, Texas who graduated from Abilene Christian University in Abilene, Texas with a Bachelor of Science in Biology and Psychology in 2012. She began her graduate career at the University of Texas at Arlington under the mentorship of Dr. Lauri Jensen-Campbell and received her Masters of Science in Experimental Psychology from UTA in 2014.

Erin's research interests include psychological and physiological health outcomes of adolescent interpersonal relationships as well as genetic influences, biological functioning, and peer victimization. She is also interested in peer victimization intervention programs. Erin plans to pursue a career in research, in which she wishes to continue exploring the effect of gene and environment interactions on various health outcomes.