SOME BULLIED ADOLESCENTS MORE SUSCEPTIBLE TO DEPRESSION

By

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To my Mother, Father, and Brother – I am who I am only because of you.

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ABSTRACT

ARE SOME BULLIED ADOLESCENTS MORE SUSCEPTIBLE TO DEPRESSION

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Prior research has documented long-term detrimental effects (e.g. emotional distress, anxiety, and depression; Kochenderfer-Ladd & Wardrop, 2001) experienced as a result of being bullied as an adolescent. Although being a recipient of chronic peer victimization is clearly related to a myriad of psychological problems, not all bullied children react to these experiences in the same way. That is, some children may be more predisposed to internalizing problems, specifically depression, when bullied than are other children. This study examined the influence of genetic polymorphism in the serotonin transport gene (5HTTLPR) on the victimization-depression link. A total of 125 adolescents (M_{age} = 12.35) took part in this study. For adolescents with the S-S or S-L variant, victimization was positively related to depression. No relationship between victimization and depression was found for children with the l-l variant. Additionally, there was a sex X 5HTTLPR X victimization interaction for overall daily cortisol, waking cortisol, and the cortisol awakening response (CAR). Peer victimization was not related to waking cortisol in boys with the s-s variant. For girls with the s-s variant peer victimization was related to lower levels of waking cortisol. Victimized boys with the s-s variant had a positive

CAR; conversely, victimized girls with the s-s variant had a flattened CAR. There was no relationship between victimization and CAR for the s-l or l-l variant adolescents. These findings suggest that the associations between victimization, neuroendocrine functioning, and depression is strongest for children who are at greater risk for developing internalizing problems (i.e., for children with at least one s-variant of 5HTTLPR).

TABLE OF CONTENTS

LIST OF ILLUSTRATIONS

LIST OF TABLES

xi

CHAPTER 1

INTRODUCTION

The month of April 2009, just like many months before, was filled with merciless bullying for Eric Mohat. The target of repeated bullying by his peers at school, Eric finally took the suggestion of one of his aggressors seriously, "Why don't you go home and shoot yourself, no one will miss you." For his parents, the month of April marked the end of their son's life (ABCnews, 2009). In the same month, while another mother was preparing dinner as usual, her son, Carl Walker-Hoover, decided to end his life because he could no longer endure the incessant daily taunts of being called gay by his peers at school. Mrs. Walker found her son hanging from the family's 3rd floor rafters with an extension cord around his neck. Tragedies such as these are not simply isolated events. Nearly 1000 US children commit suicide every year as a result of being bullied by their peers (CDC, 2009). Additionally, for approximately 30% of adolescents, peer victimization is frequent enough to cause detriments in later life (Eisenberg & Aalsma, 2005).

1.1 Definition of Peer Victimization

Being a peer victim involves being the repeated recipient of aggressive acts from one's peers. In other words, victimization involves being continually harmed by one's peers (Olweus, 2001). Peer victimization however does not pertain to (1) peers of similar status arguing or fighting with one another; (2) children or adolescents who tease or poke fun at one another in a good-natured manner; or (3) a child or adolescent being the occasional recipient of an aggressive act (Andreou, 2001). With the inclusion of the above criterion, peer victimization is best considered as a continuum lying on a spectrum of no abuse to frequent peer abuse. Based on the above definition, in the present thesis, the term peer victim was referenced in regards to those adolescent individuals who were consistently targets of aggression by their peers.

1

Peer victimization can be further divided into different types of victimization, such as relational and physical victimization (Crick & Grotpeter, 1995). Relational victimization often manifests itself as ostracism, harmful gossip, and deliberate manipulation of social relationships. In other words, this type of victimization involves harming another person's character, social relationships, or acceptance into a group, and is primarily employed to cause irreparable damage to social relationships (Putallaz et al., 2007). Subsequently, physical victimization evokes the use of verbal or physical threats, assaults, and insults that are meant to harm another's physical being, property, or status (Coie & Dodge, 1998; Putallaz et al., 2007).

Some researchers also describe a third type of victimization, namely verbal victimization which includes psychological abuse as the instrument of harm (Crick, 1997). Examples of verbal victimization include peers saying mean things to the victim or about the victim, name calling, and yelling (Olweus, 1991). All forms of victimization can also be either direct, openly confrontational acts (e.g., physical or verbal assaults) or indirect acts (e.g., spreading rumors anonymously, destroying property anonymously). Peer victimization can be experienced in any of these forms, or with a combination thereof (Crick & Bigbee, 1998; Mynard & Joseph, 2000) and research suggests that all types of victimization have psychological consequences (Paquette & Underwood, 1999).

<u>1.2 Consequences of Peer Victimization</u>

Research has indicated that being bullied is associated with emotional distress, anxiety, and depression (Kochenderfer-Ladd & Wardrop, 2001), as well as later psychological maladjustment and loneliness (Crick & Grotpeter, 1995; Crick & Bigbee, 1998; Crick, Casas, & Ku, 1999; Nansel et al., 2001; Prinstein, Boergers, & Vernberg, 2001; Storch & Masia, 2001). Children who are bullied by their peers are also likely to fear negative evaluations from peers and attempt to avoid social interactions (Crick & Grotpeter, 1996; Crick & Bigbee, 1998; Nansel et al., 2001; Storch & Masia, 2001; Grills & Ollendick, 2002). These tendencies to both fear

being evaluated by others and engage in social avoidance may put victimized children at a disadvantage for experiencing positive relationships, and as such, hinder the acquisition of both social skills, and self-esteem (Storch & Masia-Warner, 2004) as well as cause distorted views of social relationships (Storch & Masia-Warner, 2004; Crick & Grotpeter, 1996; Perry, Kusel, & Perry, 1998). Poor peer interactions and maladjustment outcomes can occur as a result of these negative coping skills that develop from being bullied.

<p style="text-align:center">1.3 Why Focus on Adolescents?</p>

Although people can be bullied at any point in the lifespan, early adolescence is a particularly important developmental period to study. First, early adolescence is associated with significant changes in biological, cognitive, and social functioning. The amount of changes occurring during the time period of adolescence is rivaled only by those changes seen in infancy. During early adolescence, peer relationships also gain increased importance, (Ellis, Rogoff, & Cromer, 1981) and tend to greatly influence a child's behavior (Elias & Zins, 2003). Ironically, as peer relationships become more important, bullying behaviors also peak sometime in early adolescence (Eisenberg & Aalsm, 2005).

In addition to bullying peaking in adolescence, other research has found that the onset of mental illnesses such as depression, anxiety disorders, and mood disorders increase substantially during adolescence as a result of a greater vulnerability to stress that occurs during this time period (e.g., Pause et al., 2008; Steinberg, 2004). Additionally, adolescent anxiety and depressive disorders show an approximate two to three fold increased risk for adulthood anxiety (Pine et al, 1998). For example, Stein et al. (2002) found that the experience of social anxiety during adolescents was an important predictor of later diagnosed depressive disorders.   Additionally, a longitudinal study conducted by Reinherz and colleagues (1993) found that both early adolescent boys and girls who were thought to be unpopular by their peers were at a greater risk for experiencing major depression in later adolescence than those

children who were considered popular by their peers. Goodyear, Herbert, Tamplin & Altham (2000) found that significant personal disappointments and losses altered cortisol production and exacerbated the expression of depression in adolescent boys and girls.

In summary, it is during this period, when adolescents are going through major physiological changes and are the most vulnerable to depression and anxiety problems that can persist into adulthood, that they are also most likely to become victims of peer abuse. Given the important link between peer relationships and psychological health, the current study examined how being bullied led to depression and anxiety. Perhaps more importantly, this study focused on an important boundary condition, namely individual differences in susceptibility to depression and anxiety. In particular, this study examined the influence of the 5HTT genetic polymorphism on the victimization-depression link. In addition, I examined whether victimization influenced diurnal patterns of cortisol (as a biological measure of stress). Finally, I examined whether the 5HTT genetic polymorphism influences the victimization-cortisol link.

In the next sections, I will begin by discussing previous links between victimization and internalizing problems. I will also examine how the endocrine system may be responsible for these associations. Finally, I will discuss why some adolescents may be particularly at risk for depression when bullied.

<u>1.4 Peer Victimization and Internalizing Problems</u>

Internalizing problems consists of factors related to personal distress as well as self-control issues, and behavioral inhibition (e.g. anxiety, depression, somatization, and social withdrawal) (Weiss, Jackson, & Susser, 1997). Chronic adolescent peer victimization has been found to be repeatedly associated with increases in internalizing problems over time (Crick & Bigbee, 1998; Crick, Casas, & Ku, 1999; Crick & Grotpeter, 1996; Janosz et al., 2008). Victimized children tend to cry easily, display anxiety in situations, tend to be socially withdrawn, have higher levels of depression, have more suicidal ideation, and tend to relinquish to their

4

attackers demands, all signs of internalizing problems (Hodges et al., 1997; Hodges & Perry, 1999; Patterson, Littman, & Bricker, 1967; Perry, Willard, & Perry, 1990).

One component of this psychological distress or internalizing problems that this thesis will focus on is depression (Kaltiala-Heino, Rimpela, Marttunen, Rimpela, Rantanen, 1999; Neary & Joseph, 1994). A meta-analysis conducted by Hawker & Boulton, (2002) found that peer victimization was most strongly related to depression, more so then any other component of psychological distress. Indeed, children who are victimized are more likely to exhibit decreased self-esteem, and increased feelings of loneliness, anxiety and depression than those children who were not victimized by their peers (Callaghan & Joseph, 1995; Hodges & Perry, 1996; Slee, 1994; Swearer, Song, Cary, Eagle, & Mickelson; 2001). The links between co-morbidity of depression and poor self-esteem in victimized children is not surprising, when one considers the fact that those children with lower self-esteem are more likely to be depressed (Harter, 1993; Lewinsohn, Roberts, Seeley, Rohde, Gotlib, & Hops, 1994; Swearer, Song, Cary, Eagle, & Mickelson; 2001).

Perhaps more ominous is the fact that the association between victimization and depression seems to hold steady over time. Moreover, this association appears to be bi-directional in nature. That is, victimized children are more likely to become depressed and have lower self-esteem, but depressed children and children with lower self-esteem are also more likely to be victimized since they exhibit distress that is often rewarding for the bullies, creating a viscous cycle of bullying and depression for these children (e.g., Egan & Perry, 1998). For example, Slee (1995) found that peer victimization in both adolescent boys and girls was highly related to depression and unhappiness in being at school. Further, Craig (1998), found that adolescent girls tended to report more depression as a result of peer victimization than males. Additionally, she found that older children tended to report more depression as a result of peer victimization than younger children. Conversely, Sourander et al. (2002) found that self-reports

of depression at age 8 were highly correlated with victimization at the age of 16 (Sweeting et al., 2006). Similar results were found in the research of Bond et al. (2001), where 13 and 14 year olds reported depression scores that were correlated with later reports of victimization.

<u>1.5 Depression and Neuorendocrine Functioning</u>

Cicchetti & Tucker (1994) proposed that the plasticity of biological systems may be responsible for the negative psychological outcomes in individuals as a result of environmental experiences. In other words, environmental factors can profoundly affect the normal trajectory of development, further increasing an individual's vulnerability to these said life experiences (Cicchetti & Walker, 2003; Dersh et al., 2002). The plasticity of biological systems when exposed to chronic peer victimization may be a contributing factor to the expression of depression and anxiety. With this idea in mind, the plasticity of the neuroendocrine system and its ability to adjust to reach homeostasis may be particularly important in understanding the long-term ramifications of being the recipient of chronic peer victimization.

A great deal of research has been conducted to show that the hypothalamic-pituitary-adrenal (HPA) system controls reactions to physical and social stress (Dickerson & Kemeny, 2004; Eisenberger et al., 2007; Purvis & Cross, 2006). Activation of the HPA first begins with the signal and release of corticotrophin releasing hormones (CRH) from the hypothalamus. This in turn activates the anterior pituitary gland which causes the adrenal cells to release adrenocortiocotripic hormones (ACTH) into the bloodstream. ACTH then begins its decent to the adrenal glands to release cortisol (McEwen et al., 1997).

Cortisol is a hormone which is an end product of hypothalamic-pituitary-adrenal (HPA) axis activation. Cortisol is associated with both regulatory roles (e.g., metabolism) as well as with stress reactions (Lovallo & Thomas, 2000). Its production is essential to deal with day to day stressors. However, increased production and exposure to high levels of cortisol can be detrimental and has been related to degeneration in hippocampal dendrites (Sapolsky, Uno,

6

Rebert, & Finch, 1990), disturbed learning and memory (McEwen, 2000), and diminished immune functioning (Miller, Cohen, & Ritchey, 2002). In other words, chronic stress can lead to frequent or persistent activation of the HPA axis or greater allostatic load, which in turn is responsible for repeated disruptions of the body's homeostatic system (McEwen, 1998). These repeated disruptions to the body's homeostatic system can then cause the body to reset itself to use a new level of homeostasis. This new level of homeostasis may actually predispose an individual to greater "wear-and-tear" on other biological systems and as a result, be more likely to experience increases in both psychological and physical health problems (Dougall & Baum, 2001; Gump & Matthews, 1999).

Increases in stress and disruptions to neuroendocrine functioning have been specifically linked to depression. Hammen (2006) found that bullied mice had altered mesolimbic dopamine systems, the brain pathway most often associated with the release of dopamine in response to addictive behaviors. The social defeat process of bullying produces brain derived neurotrophic factors responsible for neuronal changes that cause the symptom of social aversion, commonly associated with depression. Further, Rao, Hammen & Poland (2009) found that the increased prevalence of substance abuse seen in depressed individuals may be explained by high levels of stress and increased HPA activity.

Internalizing problems have also been linked to differences in neuroendocrine functioning in humans. Granger, Weisz, & Kauneckis (1994) suggested that cortisol reactivity to social challenges may be associated with a child's internalizing problem behaviors. Their research was among the first studies to find an actual link between children's behavioral response in socially challenging situations to neuroendocrine activation. Additionally, Cicchetti & Rogosch (2001) found that maltreated children with clinical reports of internalizing problems, displayed higher morning, afternoon, and average daily cortisol levels compared to those children who were not diagnosed with internalizing problems.

7

Very few studies to date have examined the impact of bullying on neuroendocrine functioning. A recent study of 154 12-year-olds found that both occasional and frequent verbal abuse by peers produced changes in HPA activity (Vaillancourt et al., 2008). In addition, being the recipient of workplace bullying was associated with lower awakening cortisol compared to those colleagues who did not experience any workplace bullying (Hansen et al., 2002). Knack, Jensen-Campbell, and Baum (in press) found that victimized children were likely to show differences in neuroendocrine functioning which, in turn, predicted poorer health outcomes. Girls, in particular, showed lower awakening cortisol levels and a flatter cortisol awakening response (CAR). Interestingly, previous research has provided links between lower awakening cortisol and posttraumatic stress disorder (PTSD) as well as with chronic fatigue (Hansen et al., 2006)

Although very few studies have examined the influence of peer victimization on neuroendocrine functioning, the impact of poor social relationships on neuroendocrine functioning is well-documented (Cicchetti & Rogosch, 2001; Gillespie & Nemeroff, 2007). Indeed, harmful social experiences have been found to negatively influence neuroendocrine systems functioning (Cicchetti & Rogosch, 2001; DeBellis, Baum, et al., 1999). As such, peer victimization should be associated with differences in neuroendocrine functioning as well as differences in internalizing problems.

<u>1.6 Genetic Contributions to Internalizing Problems</u>

A potentially important individual difference that may put some adolescents more at risk for developing internalizing problems when bullied is the genetic polymorphism 5HTT. 5HTT, also known as 5-hydoxy-tryptamine, is a serotonin transport protein responsible for the reuptake of extracellular serotonin back into the presynaptic neuron (Bah et al., 2007). In other words, the reuptake of serotonin into the synaptic cleft is mediated by a single protein, the 5HT transporter (5HTT). The gene that codes for 5HTT (SLC6A4) is found on the 17q12 chromosome (Bah et

al., 2007; Holmes et al., 2002; Holms & Hariri, 2003). Humans have a 44-base pair insertion/deletion polymorphism found in the promoter region of the 5HTT gene which is found further up from the coding region of SLC6A4 (the serotonin transport linked polymorphic region, 5HTTLPR) (Heils et al., 1996; Heinz et al., 2000; Lesch et al., 1996). This gene can be comprised of a long variant (l) and a short variant (s) (Heinz et al. 2000; Lesch et al 1996). Those individuals who carry the long variant (l) have 16 repeats within the gene while those who carry the short variant (s) have 14 repeats within the gene (Bah et al., 2007). Those persons having either one or two copies of the short (s) allele are known to have fewer serotonin transporters than those persons with an l/l genotype (Heinz et al., 2000; Holmes & Hariri, 2003; Lesch et al., 1996). Those individuals who contain the short (s) variant of the gene, have been shown to have a 50% decrease in transcriptional activity in vitro (Bah et al., 2007; Lesch et al., 1996). About 30% of the human population carries the two long variant (l) genes, 50% one short (s) and one long (l), and finally, 20% of the human population carries two short (s) variants of the gene (Caspi et al., 2003).

The 5HTT is critical in determining the amount and duration of serotonin that is present in the synaptic cleft. Fewer serotonin transporters can have serious implications for developing internalizing problems given lack of serotonin has been implicated in depression and anxiety. Indeed, recent studies have found that the polymorphism of these gene (i.,e., at least one short (s) alleles) have been associated with depression, anxiety-related traits, and psychiatric disorders (Lesch et al., 1996; Melke et al., 2001; Bellivier et al., 2002; Hairiri et al., 2002; Anguelova et al., 2003; Caspi et al., 2003; Melke, 2003; Furmark et al., 2004; Kaufman et al., 2004; Schinka et al., 2004; Sen et al., 2004; Hairiri et al., 2005; Serretti et al., 2006).

Caspi et al. (2003) conducted a longitudinal study on stressful experiences and its relationship to depression. They found that the polymorphism of the 5HTT gene found on the promoter region of the serotonin transporter was found to moderate the link between stressful

9

life events and depression. Further, those individuals containing one or two variants of the short (s) allele were found to exhibit more depressive symptoms and were more often diagnosed with depression and found to have suicidal thoughts as a result of stressful events than those individuals containing the two long (l) alleles. As of 2008, fifteen independent studies have found a gene-environment interaction between the polymorphism of the promoter region of 5HTT and links between environmental adversities and its relationship to depression. Recent research has also found associations between the polymorphism of the serotonin transporter region, and internalizing disorders (Greenberg et al., 2000; Lesch et al., 1996; Melke et al., 2001; Seretti et al., 1999).

Similar results are found in animal models. Mice that have had their 5HTT gene disabled show behaviors that are analogous with anxious and depressive individuals (Uher & McGuffin, 2008). 5HTT "knockout" mice are often reluctant to explore spaces with bright lights or with elevated or exposed platforms. They also tend to give up exceedingly quickly in what is presumably an uncomfortable situation (Adamec, Burton, Blundell, Murphy, & Holmes, 2006; Holmes, Yang, Lesch, Grawley, & Murphy, 2003; Uher & McGuffin, 2008; Wellman et al., 2007). Although a growing wealth of research has indicated that 5HTT does indeed moderate the relationship between life stress and depression, little research has been conducted on the actual mechanism responsible for this relationship. Some research has suggested that stress reactivity may be one possible underlying mechanism. To the extent that peer victimization is stressful, it should lead to greater reactivity and higher incidence of depression and anxiety (Caspi et al., 2003). In other words, having at least one s variant of the 5HTTPR should strengthen the link between victimization and depression (Cicchetti & Blender, 2004).

Lowry (2002) found direct evidence that the serotonin transport system of 5HTT contributed to altered states of HPA functioning. Furthermore, research has suggested that two short alleles in 5HTTLPR leads to increased sensitivity to stress as can be measured by

10

endocrine function. Moreover, endocrine functioning has been found to be the mediator between stressful situations and depression (Barr et al., 2003; Cassano & D'mello, 2001; Gotlib et al., 2008). Based on this line of research, those with at least one 5HTT s variant should be biologically more sensitive to stress and as a result have exaggerated HPA axis responses, which in turn will lead to greater levels of depression. Similarly, Gotlib and colleagues found that those individuals possessing the s-s variant of the 5HTTLPR showed a greater increase in cortisol after a stressor. Similar findings were found in animal models (Barr et al, 2004). Based on these findings, I predict that individuals with at least one s-variant of 5HTT will show more exaggerated differences in their daily cortisol levels when bullied than will individuals with the l-l variant. Stress reactivity, however, was not examined in this study.

<div align="center">1.7 Current Study</div>

Recent research has highlighted some of the detriments caused to those who have experienced chronic peer victimization. However, research should also be conducted to determine possible reasons for why some children are more likely to be affected by those long-term negative outcomes associated with peer victimization, while other children are not. Based on this idea, it is possible that some children may actually have a genetic predisposition toward autonomic arousal, which over time, may in fact exacerbate the influence of chronic peer victimization on internalizing problems. For the purpose of this thesis, the genetic polymorphisms of 5HTT were assessed. This biological marker has been linked to stress sensitivity (as assessed by cortisol), changes in daily cortisol levels (as assessed by cortisol), and the increased likelihood of depression when exposed to greater levels of stress.

Based on the above research, it is hypothesized that the genetic polymorphism of 5HTT (at least one short allele) may act as a boundary condition between victimization, endocrine functioning, and internalizing problems. It is anticipated that having at least one s variant of the 5HTTLPR will lead to greater sensitivity to social stress associated with being bullied.

<div align="center">11</div>

The following aims were assessed as part of this master's thesis.

1.1.1 Aims

Aim 1: To examine whether adolescents who are recipients of chronic peer victimization have abnormal cortisol levels and greater levels of depression and anxiety compared to children who are not chronically victimized.

Aim 2: To examine whether variations in one functional polymorphism, namely 5HTT (serotonin transporter gene) lead to higher levels of depression and anxiety.

Aim 3.   To examine whether 5HTTLPR moderates the relationship between peer victimization with depression and biological functioning (i.e., cortisol levels). It was expected that children with at least one s- variant of the 5HTTLPR would be more vulnerable to depression, anxiety, and have abnormal cortisol levels.

Aim 4. To examine whether biological functioning mediates the link between victimization and depression. In addition, I examined whether 5HTTLPR moderates this predicted mediated relationship. First, it is possible that 5HTTLPR will have its influence on depression via the victimization-cortisol link only. That is, persons with at least one s variant of 5HTTLPR will be more reactive to victimization, which will lead to higher levels of daily cortisol, which in turn increases the incidence of internalizing problems (Model 1). Second, it is possible that 5HTTLPR will only influence the cortisol-depression link (Model 2).   Finally, it is possible that the 5HTTLPR will influence both pathways (i.e., the victim-cortisol association and the cortisol-depression association). Although all three models were tested, I anticipated that model 1 would most accurately represent these phenomena based on previous research. A visual description of Models 1-3 can be found in the appendix B.

CHAPTER 2

METHODS

2.1 Participants

A total of 125 participants took part in this study (boys; N = 57; 45.6%; girls; N = 68; 54.4%). The ethnic composition was diverse with 52.8% Caucasian, 12. 8% African American, 1.6% Asian, and 22.4% Hispanic or Latino, 0.8% American Indian or Native American, and 8.8% Other. Participants were 5th to 8th grade adolescents (M_{age} = 12.35, SD = 1.10; age range: 10-15 years) who were taking part in an ongoing study on peer social relationships and health. A total of 65 of the adolescents came back to give a sample of their DNA for this study. Additionally, another 60 adolescents were asked to provide a DNA sample when they first came into the lab. I needed a sample of 120 participants for a predicted effect size of r = .25, α = .05, two tailed, and a power of .80 (Cohen, 1988). Additionally, for a medium effect size (r = .30), a minimum sample size of 70 was needed to examine the mediation model with asymmetric confidence intervals and a power of .80 (MacKinnon, 2008).

Several methods were used to recruit participants. First, participants were randomly selected from a mailing list obtained from area schools and contacted for possible participation in a study on friendship, peer relationships, and health. In addition, researchers talked to several area schools in large groups to explain the scope of her project. Finally, researchers recruited participants through a local dance and cheer academy as well as several summer camp programs. Adolescents and their parents who were returning to the lab to provide their DNA were paid as part of a larger on-going longitudinal study. The newly enrolled participants were paid as part of their participation in the larger health study.

13

<u>2.2 Assessments of Victimization</u>

2.2.1. Children's Self Experiences Questionnaire – Self Reports (CSEQ-SR)

The CSEQ-SR assessed peer-related occurrences of victimization (Crick & Grotpeter,

1995). This questionnaire consisted of three subscales used to assess overt victimization (e.g.,

"I get hit and pushed by other kids"), relational victimization (e.g., "I am ignored by other

classmates when someone is mad at me"), and being the recipient of prosocial help (e.g., ""How

often does another kid give you help when you need it?"). Each subscale consisted of five items

used to measure the frequency children experienced a particular event 1 (never) to 5 (all the

time) on a Likert type scale. High reliability was found for self-reports of overt (α = 0.82),

relational (α = 0.86), and prosocial behavior/general support (α = 0.84).

2.2.2. Direct and Indirect Aggression Scales- Victim Version (DIAS)

The DIAS (Bjorkqvist, Lagerspetz, & Osterman, 1992) assessed how frequently one

experienced aggression/victimization on the three subscales of physical (e.g., "How often are

you hit by others?"), verbal (e.g., "How often are you insulted by others?"), and indirect

aggression/victimization (e.g., "How often are you ignored by others?"). The DIAS is a 24-item

inventory with questions being answered on a 1 (never) to 5 (very often) Likert type scale. High

reliability was found for both self and parental reports of physical (α = 0.84, 0.89), verbal (α =

0.86, 0.89), and indirect (α = 0.88, 0.93) victimization, respectively.

<u>2.3 Internalizing Problems</u>

2.3.1. Achenbach – CBCL and YSR

To assess depression and anxiety, I used the DSM subscales of affective problems and

anxiety problems of the CBCL and the YSR. The affective problems scale includes measures

of dysthymia and major depression (Achenbach, Dumenci, & Rescorla, 2000). , This subscale

includes 10 items from the YSR and CBCL, which were rated on a 0 = not true to 2 = very true, Likert-type scale. Participants were asked to rate how closely each item described them. Sample items included "Enjoys little," "Cries," "Worthless," "Guilty," "Tired," "Apathetic," "Talks Suicide," "Underactive," and "Sad." The anxiety problems scale is a 6-item subset from the YSR and CBCL. Items were again rated on a 0 = not true to 2 = very true, Likert-type scale. Participants rated how closely each item described them. Items included "Dependent," "Fears," "Fears School," "Nervous," "Fearful," and "Worries" (Achenbach & Rescorla, 2001).

2.3.2. Depression Inventory (D-inventory)

The D-Inventory was a second measure of affective problems that assessed the extent of the individual's depressive symptoms. Both parents and children completed the measure on the child. Knack (2009) modified previous standard measures to create an inventory composed of 20 questions. For each item, participants rated a statement from 0 (not at all) to 3 (very much). Higher scores indicate greater depression symptoms. These items had high reliability when previously used, $\alpha = 0.94$. In the present study, reliability was equally high for adolescents, $\alpha = 0.88$, and for parents, $\alpha = 0.72$.

<div align="center">2.4 Procedure</div>

As children were involved, a two step consent process took place. Parents gave consent and children gave their assent to participate in a study concerning the relationships, school performance, & health in adolescents. All participants completed the surveys in the present study at identical times in each phase described below. The only difference between the two subsamples is that one sample gave their DNA nearly a year after completing the procedures.   This is acceptable given that DNA does not change over time; as such, researchers typically collect DNA after major assessments have been completed (see Caspi et al., 2003 for an example). During the re-contacting period for the first sample of participants, parents were told about the DNA testing process and asked to arrange a time to have their child

give a DNA sample. Additional measures were collected from this subsample, which were not part of this study.

In phase one of the previous study and in the current study, adolescents completed victimization measures (DIAS – Victim Version; Bjorkqvist, Lagerspetz, & Osterman, 1992; CSEQ-SR; Crick & Grotepher, 1995) either at school or online individually. Adolescents and their parent then came into the laboratory at the University of Texas at Arlington for a two-part second phase of the research study. During session one of phase two, adolescents completed a series of questionnaires that included measures of depression and anxiety (Faulstich et al., 1986).

Parents completed parental-reports of their adolescent's experiences of victimization. Adolescents and their parent were then taught methods of proper collection and storage of the adolescent's saliva. Adolescents were instructed to collect four samples of their saliva over two non-sport school days. Samples were collected immediately when the adolescent awoke, 30 minutes after waking, immediately when the adolescent returned home from school, and 30 minutes before going to bed. The saliva samples were collected to measure salivary basal cortisol levels in order to determine each individual adolescent's diurnal cortisol pattern. All adolescents completed logs, either with paper-and-pencil, on-line, or with a Palm Pilot, to assess their accuracy of saliva collection; these logs also reminded the participants about the proper procedures for these collections.

All adolescents were given an instruction sheet to post on their refrigerator at home as well as the researcher's contact information in case they had problems or questions while collecting the saliva samples. If adolescents were part of the new study, during this point a sample of spit would be collected in an Oragene® kit for DNA. Participants were asked to spit

directly into the top of the Oragene® tube, filling it up with their saliva up until the marked line. After participants finished filling the tube, the top was closed releasing a reagent that can keep the sample good for up to five years. As stated previously, if participants had not provided a sample of DNA at this time, they were asked to come in for a session three where they gave a sample of DNA via the Oragene® kit.

Adolescents and their parent then returned to the laboratory for the second session of phase two. Typically, participants returned within three to nine days. Parents completed several questionnaires, which included levels their child's depression and the Achenbach-CBCL (which also measured depression and anxiety). Meanwhile, the adolescent completed the Trier Social Stress Test (TSST) that is not part of this study. After the TSST, adolescents completed the Achenbach-YSR. Parents and adolescents were then carefully debriefed together and paid for their participation. Additional measures were completed by the parent and child that were not part of this study (e.g., personality, externalizing problems, and health measures).

2.5 DNA Extraction and Analysis

The Oragene® kits were sent to the UT Southwestern Medical Center (at the McDermott Center for Human Growth and Development: Center for Human Genetics) for DNA extraction. DNA analysis was conducted at the UT Arlington Genomic Core. Only the gene that codes for the serotonin transport protein of 5htt, also known as 5-hydoxy-tryptamine, was assessed.

Following previous research (Caspi et al., 2003; Gelernter et al, 1997; Lesch et al., 1996), the primer for 5HTTLPR had the forward sequence (5' – ATGCCAGCACCTAACCCCTAATGT-3') and the reverse (CGACCGCAAGGTGGGCGGGA-3'). These primers were used to amplify the 419 base pair product for the 16 repeat (l) allele and the 375 base pair product for the 14 repeat (s) allele. PCR was carried out on a thermo cycler using the following cycling conditions: initial 15-minutes denaturing step at 95° C, followed by 35

cycles of 94° C for 30 sec, 66° C for 30 seconds and 72° C for 40 seconds and a final extension phase of 72° C for 15 minutes. Reactions were performed in 10X reaction Buffer, 10.0 μL GoTaq® Green Master Mix (Promega), 1.0 μL genomic DNA and 6 μL of 100bp DNA Ladder (Promega). PCR products were separated on a 1.5% agarose gel supplemented with Ethidium bromide (0.03%) and visualized by ultraviolet transillumination.

I anticipated that my sample would include 57% for the 16 repeat ('l') allele and 43% for the 14 repeat ('s') allele. I also followed the well-documented functional classification described by Lesch et al. (1996) to divide the sample into three groups on the basis of genotype, s/s (~20%), s/l (~50%) and l/l (~30%) (Caspi, et al., 2003; Nilsson et al., 2005; Taylor et al., 2006). Based on this information, I expected that approximately 20% of adolescents from my sample would have the s/s variant of the 5HTT and 50% of adolescents would have the s/l variant. In my sample, I found that 22.4% of adolescents had the s/s variant, 46.4% had the s/l variant, and 31.2% had the l/l variant (see Table 2.1).

Table 2.1 Distribution of each Allele type for boys and girls

DNA 5HTT Alleles	Boys	Girls	Total
S,S	15 (26%)	13 (19%)	28 (22%)
L,L	25 (44%)	33 (49%)	58 (46%)
L,L	17 (30%)	22 (30%)	39 (31%)

2.6 Cortisol Assays

Cortisol is reliably present in levels proportional to other bodily fluids (e.g., blood) in saliva and can be collected with little difficulty. That is, accessibility to cortisol levels appears to be as good as in other fluids and when collected properly, can provide good estimates of HPA activity. Compliance is less of a problem unless specific times for samples are derived. As such, we used Palm Pilot, on-line, and paper-and-pencil questionnaires to track compliance-rates. The Palm Pilots, on-line, and paper-and-pencil logs were used to ensure adolescents

18

were properly collecting saliva by prompting the adolescents with questions that asked if they completely soak the cotton swab, if they made sure the cap to the sample tube was on tightly, and whether or not they place the tube in the freezer. Adolescents were asked to log onto the Palm Pilots, SurveyMonkey, or fill in the paper-and-pencil logs during each time cortisol was taken. In addition, the participant was to log the exact time the sample was taken on the outside of each cortisol tube.

Samples were collected in salivettes (Starstedt), nested tubes that looked like small centrifuge tubes with a small cotton wedge (similar to the cotton rolls used by many dentists). Participants removed the cotton, placed it in their mouth between their cheek and gum, and gently moisten the cotton for about 60 sec. They were told not to bite down on the cotton as this would expel some of the cortisol that had already been absorbed. Rather, they were told to gum and lightly chew the cotton until it was saturated. When done, the cotton was placed in prelabeled tubes, the time and date of the donation was recorded, and the sample tube frozen. Once all samples for a session were collected, they were processed in Dr. Andy Baum's Cortisol Laboratory at the University of Texas at Arlington. First, they were spun and frozen at -20 C for future assay. The assay used to measure cortisol in saliva uses ELISA (Enzyme-Linked Immunoabsorbence Assay) techniques to measure the quantity of unknown (in this case cortisol) in each sample. I used the salivary cortisol kits from Salimetrics (State College, PA) for estimation of cortisol values as part of the larger on-going study. In order to get my overall values of cortisol, I log transformed and collapsed values across the two days.

CHAPTER 3

RESULTS

3.1 Descriptive Statistics

The reliabilities and descriptive statistics for the major measures can be found in Tables

3.1 and 3.2.

Table 3.1 Descriptive Statistics of Self and Parent-Reports for Victimization

Measure	Range	Min	Max	Mean	SD	Skewness (Std. Error)	Kurtosis (Std. Error)	A
Total Victimization	3.37	-1.11	2.26	0.002	0.766	1.059 (.22)	0.478 (.43)	0.82
Self-Reports								
Overt	14	5	19	8.17	3.39	1.21 (.22)	0.98 (.43)	0.82
Relational	15	5	20	8.97	3.85	0.94 (.22)	0.01 (.43)	0.86
Physical	21	7	28	11.15	4.27	1.67 (.22)	3.29 (.43)	0.84
Verbal	18	5	23	10.79	4.60	0.87 (.22)	0.06 (.43)	0.86
Indirect	35	12	47	22.59	8.07	1.02 (.22)	0.42 (.43)	0.88
Parent-Reports								
Physical	20	7	27	10.21	3.65	1.40 (.22)	3.29 (.43)	0.89
Verbal	16	5	21	9.91	3.40	.78 (.22)	0.06 (.43)	0.89
Indirect	33	12	45	22.9	7.63	.65 (.22)	0.42 (.43)	0.93

20

Table 3.2 Descriptive Statistics for Depression Measures

Measure	Range	Min	Max	Mean	SD	Skewness (Std. Error)	Kurtosis (Std. Error)	A
Self-Reports								
Depression	46	5	51	27.52	9.11	.09 (.22)	.25 (.43)	0.72
Anxiety Disorder	10	0	10	2.95	2.43	.75 (.22)	-.18(.43)	
Affective Disorder (AD)	18	0	18	3.91	3.44	1.20 (.22)	1.85 (.43)	
Parent-Reports								
Depression	42	17	59	30.35	7.31	.70 (.22)	1.48 (.43)	0.88
Anxiety Disorder	9	0	9	1.99	2.21	1.39 (.22)	1.42 (.43)	
Affective Disorder (AD)	18	0	18	2.86	3.06	1.78 (.22)	4.72 (.43)	
Collapsed Measures Affective Disorder (AD)	5.58	-1.04	4.54	0.08	0.92	.162 (.217)	4.44 (.430)	
Anxiety Disorder	3.9	-1.06	2.85	0.08	0.89	1.11 (.217)	.978 (.430)	
Depression (D-Inventory)	4.99	-2.09	2.9	0.07	0.82	.192 (.217)	1.37 (.430)	
Total Depression (AD +D-Inventory)	4.47	-1.56	2.9	0.07	0.76	.936 (.217)	1.65 (.430)	

Tables 3.3-3.7 show the inter-relationships among the victimization and depression measures.

Table 3.3 Correlations of Self and Parent Reports of Victimization

Measure	Overt	Relational	Physical	Verbal	Indirect
Self Reports					
Overt					
Relational	.68**				
Physical	.77**	.50**			
Verbal	.76**	.63**	.78**		
Indirect	.63**	.81**	.62**	.79**	
Parent Reports					
Physical					
Verbal			.65**		
Indirect			.53**	.81**	

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 3.4 Correlations of Self Reports with Parent Reports of Victimization

Measure	Physical_PR	Verbal_PR	Indirect_PR
Physical_SR	.46**	.37**	.28**
Verbal_SR	.30**	.45**	.46**
Indirect_SR	.25**	.40**	.52**

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

22

Table 3.5 Correlations of Self and Parent Reports of Depression

Measure	Depression_pr	Anxiety_pr	Affective_pr	Depression_sr	Anxiety_sr
Parent Reports					
Depression_pr					
Anxiety_pr	.276**				
Affective_pr	.327**	.525**			
Self Reports					
Depression_sr					
Anxiety_sr				.445**	
Affective_sr				.506**	.725**

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 3.6 Correlations of Self Reports of Depression with Parent Reports of Depression

Measure	Depression_PR	Anxiety_PR	Affective_PR
Depression_SR	.33**	.33**	.47**
Anxiety_SR	.32**	.59**	.44**
Affective_SR	.24**	.38**	.68**

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 3.7 Correlations of Victimization with Depression Measures for each Allele Type

DNA_5HTT Alleles	Achenbach-DSM Affective Disorder	Achenbach-DSM Anxiety Disorder	Total Depression - D-inventory	Depression (AD - D-inventory)
S,S	0.21 (0.45**)	0.22 (0.53**)	0.62**(0.60**)	0.52**(0.62**)
S,L	0.53**	0.15	0.51**	0.59**
L,L	0.05	0.15	0.34*	0.20

[1] Numbers in parenthesis are the correlations with the outliers removed

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

As can be seen in Tables 3.3-3.4 the inter-correlations among the victimization measures were high (rs = 0.50 – 0.81). As such, I z-scored all victimization measures and averaged together to create an overall measure of peer victimization. [1]

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[1] Another way to conceptualize victimization would be to factor analyze the eight victim composites and use the latent score to predict behavior and biological functioning. Using principal component analyses with VARIMAX rotation, one factor that accounted for 43.13% of the variance emerged. The factor score from this analysis was highly correlated with the current measure of victimization (r = 0.97) and produced virtually identical results to what is reported in this paper.

Table 3.8 Correlations of Self and Parent Reports of Depression with Total Victimization

Measure	Total Victimization
Self-Reports	
Depression	.42**
Anxiety	.25**
Affective	.33**
Parent Reports	
Depression	.39**
Anxiety	.07
Affective	.26**
Collapsed Measures	
Affective	.32**
Anxiety	.18*
Total Depression (D-Inventory)	.50*
Depression (AD + Total Depression)	.46**

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

In addition, the correlations among the depression measures (i.e., parent-rated depression, child-rated depression, YSR affective disorder, and CBCL affective disorder) were high so they were averaged to create an overall measure of depression (rs = 0.33-0.68; see Tables 3.6-3.7). Finally, the YSR and CBCL anxiety disorder measures were highly correlated (r = 0.58) and were averaged to create an overall measure of anxiety.

3.2 Internalizing Problems

Correlations revealed that the relationship between victimization and depression were significant only for adolescents who had at least one s-variant of 5HTTLPR (see Table 3.8). To further assess whether victimization, 5HTTLPR, and their interaction predict internalizing problems, a moderated multiple regression (MMR) was run. Victimization was centered and

treated as a continuous variable. 5HTTLPR was coded using unweighted effects codes (Aiken & West, 1991). Finally, the cross-product between victimization and each polymorphism code was created. Victimization, the unweighted effects codes, and their cross-products were then entered in the equation. Sex of participant was also entered into the model as a control variable using unweighted effects codes. [2] Dependent measures included overall depression (i.e., averaged affective problems (CBCL and YSR) and depression (both s- and p-reports)) and anxiety (averaged CBCL and YSR anxiety problems). Following significant interactions, simple effects analyses for victimization were conducted using the appropriate dummy codes for 5HTTLPR (see Aiken and West, 1991, pp. 130-133, for a review).

Victimization was related to depression ($B = 0.46$, $t(118) = 5.61$, $p = .01$, $sr = 0.43$) and anxiety ($B = 0.23$, $t(118) = 2.10$, $p = .04$). As anticipated, victimized children had greater levels of depression and anxiety. There was no evidence of a 5HTTLPR main effect for either depression or anxiety, $Fs (2, 118) = .20, .19$, $p = .823$, ns $p = .83$, ns, respectively. There was no evidence of a 5HTTLPR X victimization interaction for anxiety, $F(2, 118) = .121$, ns. As predicted, there was an interaction between 5HTTLPR and victimization for depression, $F(2, 118) = 5.51$, $p = .01$, $\Delta R^2 = 0.07$. For children with the S-S variant, victimization was positively related to depression, $B = 0.48$, $t(118) = 2.84$, $p = .005$, $sr = 0.22$. For children with the S-L variant, victimization was also positively related to depression, $B = 0.73$, $t(118) = 6.18$, $p = .001$, $sr = 0.47$. For children with the L-L variant, there was no evidence that victimization was related to depression, $B = 0.15$, $t(118) = 1.19$, $p = .24$, $sr = 0.09$. As predicted, children with at least one s variant of the 5HTTLPR were more susceptible to depression when victimized (see Figure 3.1).

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[2] A model with the interaction terms for sex of participant were originally entered in a model. There was no interaction between sex of participant with 5HTTLPR and victimization so these cross-products were dropped from the final model.

Susceptibility to Depression
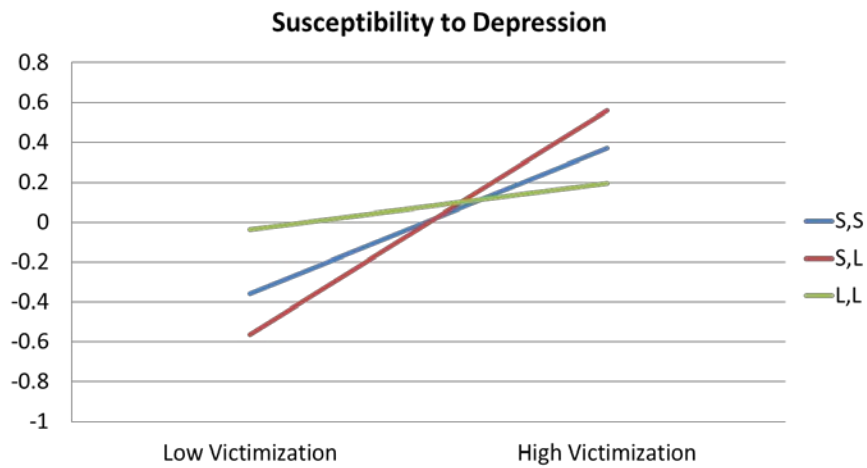
Figure 3.1. Participants' susceptibility to depression as a result of peer victimization based on allele variants.

Diagnostic analyses revealed that there were a cluster of 4 outliers in the s-s variant group. These four outliers involved two Hispanic and two Caucasian boys who reported extremely high levels of victimization, but moderate levels of depression. Supplementary analyses examined the model excluding these outliers. Without the 4 outliers, the magnitude of the relationship between victimization and depression was stronger, $B = 0.96$, $t(114) = 2.85$, $p = .01$) (see Figure 3.2 and Table 3.8).

Susceptibility to Depression Without Outliers
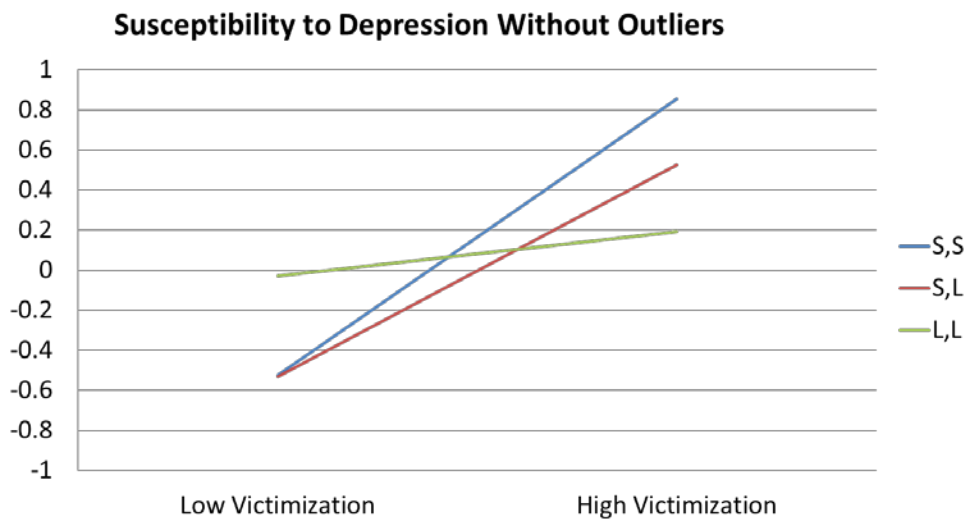
Figure 3.2. Participants' susceptibility to depression (outliers removed) as a result of peer victimization based on allele variant.

3.3 Cortisol Patterns

Cortisol was assessed as: (1) the average cortisol level; (2) waking cortisol (T2) (3) evening cortisol (T3 and T4); and (4) the cortisol awakening response (CAR) or the difference between wakening and wake (+ 30 minutes)[3]. The diurnal pattern is outside the scope of this paper and will not be examined for the thesis. To create the measure of cortisol awakening response or CAR (i.e., the change for T1-T2), change scores for cortisol was computed by regressing the T2 cortisol (i.e., 30 minutes after waking) onto the T1 cortisol[4] (i.e., the cortisol collected when they immediately woke up) (Applebaum & McCall, 1983). These residual scores from this analysis were then used as the change scores in my analyses. To assess overall cortisol throughout the day, area under the curve with reference to zero or ground (AUCg) standardized by time was computed (see Edwards, Evans, Hucklebridge, & Clow, 2001; Pruessner, Kirschbaum, Meinlschmidt, & Hellhammer, 2003). Given that previous research has

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[3] Several participants (N = 8) were dropped from the cortisol analyses because they had no useable cortisol.
[4] There was no evidence of any differences on T1 cortisol by victimization, 5HTTLPR, or sex of participant.

found gender differences in cortisol responses for victimized children, we first examined a model that included sex of participant as a moderator. As in the previous model, victimization was centered and treated as a continuous variable. 5HTTLPR and sex of participant were coded using unweighted effects codes (Aiken & West, 1991). Finally, the cross-product between sex of participant, victimization, and each polymorphism code was created. Victimization, the unweighted effects codes for 5HTTLPR and sex, and all of their cross-products were then entered in the equation.   Following significant interactions, the appropriate dummy codes were used to examine lower-order interactions and simple slopes (Aiken & West, 1991).

There was a gender X victimization interaction for overall daily cortisol levels, $F_{(1, 105)}$ = 5.94, p = .02, ΔR^2 = 0.05. For boys, there was no evidence that victimization was related to overall cortisol, B = 0.07, $t(105)$ = 1.15, p = 0.25, sr = 0.11. For girls, victimization was negatively related to overall daily cortisol levels, B = -0.23, $t(105)$ = -2.15, p = 0.03, sr = -0.20.

There was also a gender X victimization interaction for awake cortisol (T2), $F_{(1, 105)}$ = 5.55, p = 0.02, ΔR^2 = 0.05. For boys, there was no evidence that victimization was related to awake cortisol, B = 0.03, $t(105)$ = 0.28, p = 0.78, sr = 0.03. For girls, victimization was negatively related to awake cortisol, B = -0.40, $t(105)$ = -2.58, p =0.01, sr = -0.23.

Finally, there was a marginal gender X victimization interaction for the cortisol awakening response (CAR), $F_{(1, 105)}$ = 3.43, p = .07, ΔR^2 = 0.03. For boys, there was no evidence that victimization was related to CAR, B = -0.02, $t(105)$ = -0.30, p = 0.77, sr = -0.026. For girls, victimization was related to flattened cortisol level, B = -0.30, $t(105)$ = -2.34, p = 0.02, sr = -0.21.

However, I predicted a three-way interaction. That is, 5HTTLPR would moderate the association between victimization and biological functioning; however, the pattern would be different for boys and girls. There was no evidence of a sex X 5HTTLPR X victimization interaction for evening cortisol (i.e. ,T3 and T4), $Fs_{(2, 105)}$ = 1.16, 0.52, ns, ΔRs^2 = 0.02, 0.01.

29

As predicted, there was a sex X 5HTTLPR X victimization interaction for waking cortisol (T2) $(F_{(2, 107)} = 5.08$, $p = .01$, $\Delta R^2 = 0.08)$ and CAR $(F_{(2, 105)} = 6.90$, $p = .002$, $\Delta R^2 = 0.11$. There was also a marginal sex X 5HTTLPR X victimization interaction for AUCg, $F_{(2, 105)} = 2.64$, $p = 0.08$, $\Delta R^2 = 0.045$.

3.3.1. Wake Cortisol (T2)

There was a victim X 5HTTLPR interaction for boys, $F_{(2, 105)} = 3.98$, $p = .02$, $\Delta R^2 = 0.06$. There was no evidence that victimization was related to waking cortisol for boys with the s-s variant $(B = 0.27$, $t(105) = 1.59$, $p = 0.12$, $sr = 0.14)$ or the s-l variant, $B = 0.13$, $t(105) = 0.76$, $p = 0.45$, $sr = 0.07$. For the L-L variant, victimization was related to lower levels of cortisol, $B = -0.32$, $t(105) = -3.60$, $p = .03$, $sr = -0.20$. There was a marginal victim X 5HTTLPR interaction for girls, $F_{(2, 105)} = 2.80$, $p = .07$, $\Delta R^2 = 0.06$. For girls with the s-s variant, victimization was related to lower levels of waking cortisol (T2), $B = -1.07$, $t(105) = -2.63$, $p = 0.01$, $sr = -0.24$. There was no evidence that victimization was related to lower levels of cortisol for girls with the S-L variant $(B = -0.11$, $t(105) = -0.79$, $p = 0.43$, $sr = -0.07)$ or the L-L variant $(B = -0.03$, $t(105) = -0.18$, $p = 0.86$, $sr = -0.02)$.
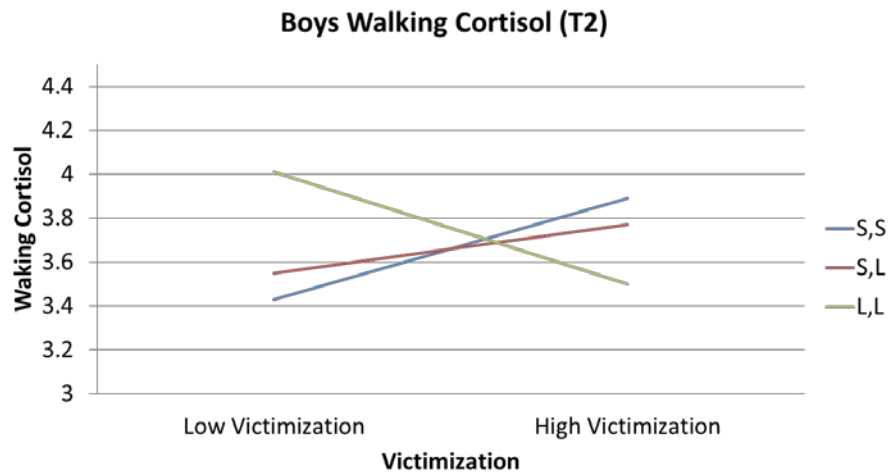
Figure 3.3. Boys waking cortisol (T2) as a result of peer victimization based on allele variants

Girls Walking Cortisol (T2)
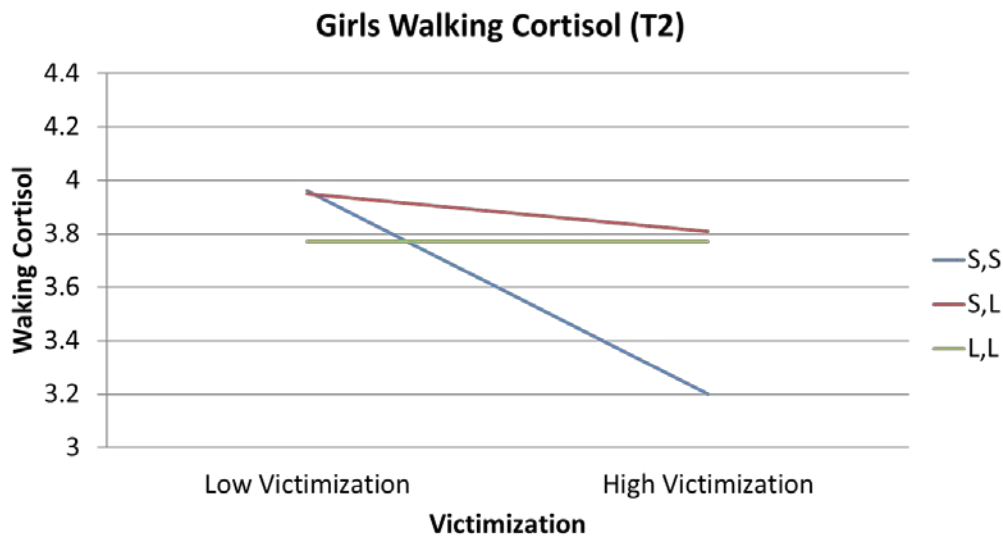
Figure 3.4. Girls waking cortisol (T2) as a result of peer victimization based on allele variants.

3.3.2. CAR

There was a victim X 5HTTLPR interaction for boys, $F_{(2, 105)} = 5.63$, $p = .01$, $\Delta R^2 = 0.09$. For boys with the s-s variant, victimization was related to a positive CAR, $B = 0.24$, $t(105) = 1.70$, $p = 0.09$, sr $= 0.15$. Conversely, victimization was negatively related to CAR for boys with the L-L variant ($B = -.36$, $t(105) = -2.97$, $p = .004$, sr $= -0.27$). There was no evidence of a victimization-CAR link for boys with the S-L variant ($B = 0.05$, $t(105) = 0.35$, $p = .73$ sr $= 0.03$).

For girls, there was also a victim X 5HTTLPR interaction for CAR, $F_{(2, 105)} = 3.38$, $p = .04$, $\Delta R^2 = 0.05$. For girls with the s-s variant, a flatter CAR was associated with victimization, $B = -0.90$, $t(105) = -2.71$, $p = 0.01$, sr $= -0.24$. There was no evidence of a victimization-CAR link for girls with either the S-L variant ($B = -0.02$, $t(105) = -0.19$, $p = 0.85$, sr $= -0.02$) or the L-L variant ($B = 0.02$, $t(105) = 0.14$, $p = 0.89$, sr $= -0.01$).
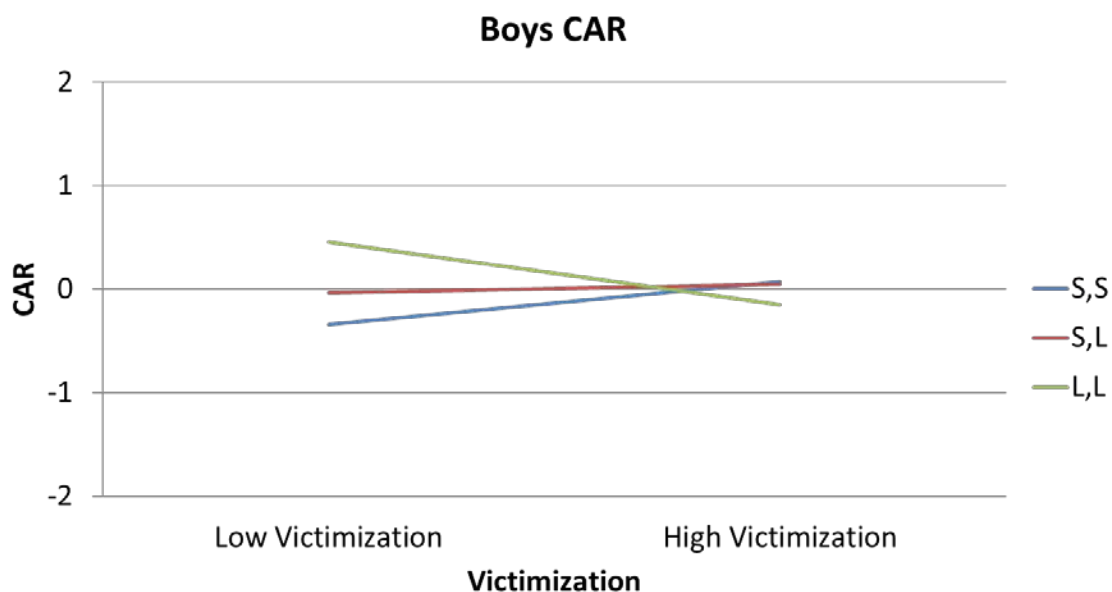
Boys CAR

Figure 3.5. Boys CAR as a result of peer victimization based on allele variants.
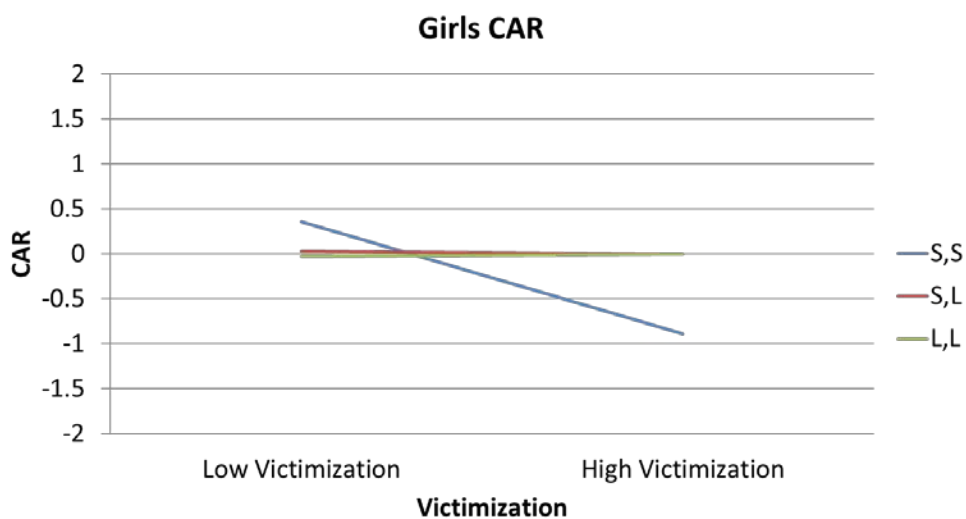
Girls CAR

Figure 3.6. Girls CAR as a result of peer victimization based on allele variants.

3.3.3. AUCg

For boys, there was no evidence of a victimization X 5HTTLPR interaction, $F_{(2, 105)}$ = 0.39, p = 0.68, ΔR^2 = 0.01. For girls, there was a marginal victim X 5HTTLPR interaction for girls, $F_{(2, 105)}$ = 2.42, p =.09, ΔR^2 = 0.04. For girls with the s-s variant, victimization was

negatively associated AUCg, B = -0.63, t(105) = -2.29, p = .02, sr = -0.21. Victimized girls with the s-s- variant had lower overall daily cortisol levels than girls who were not bullied with the s-s variant. There was no evidence that victimization was related to AUCg for girls with the S-L and L-L variant of 5HTTLPR, Bs = 0.01,-0.06, ts(105) = 0.08, -0.47 ps = 0.93, 0.64, srs = 0.01, -0.04.
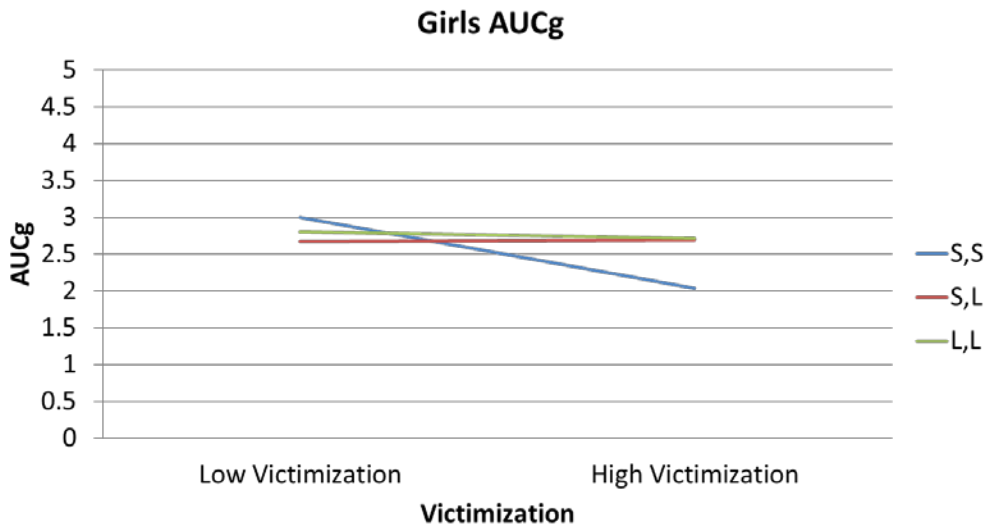
Girls AUCg

Figure 3.7. Girls AUCg as a result of peer victimization based on allele variants.
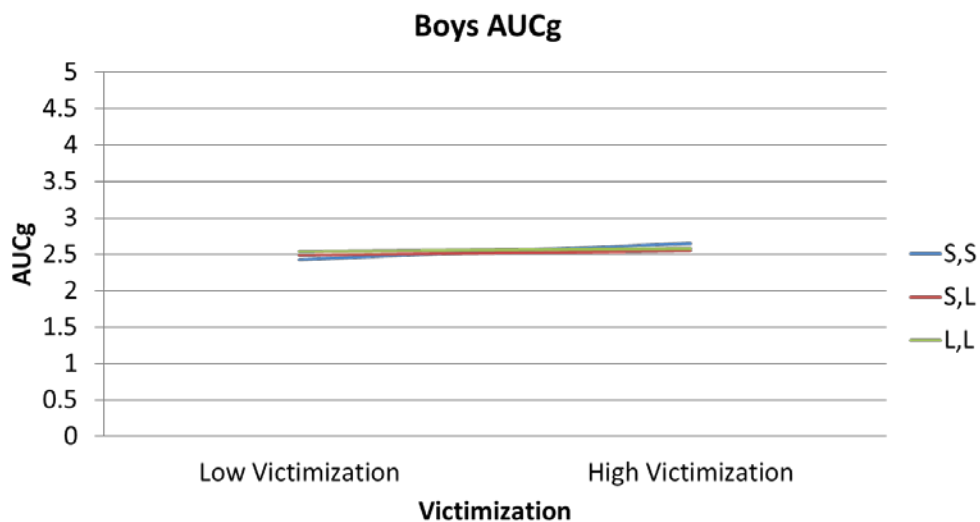
Boys AUCg

Figure 3.8. Boys AUCg as a result of peer victimization based on allele variants.

33

3.4 Mediation Models

I had predicted that victimization would be related to depression via endocrine

functioning (as assessed by cortisol levels). There was no evidence that cortisol was related to

depression for boys and girls (see Table 3.9 and 3.10).

Table 3.9 Girls Correlations of Depression and Anxiety at each Cortisol Measure

Measures	Cortisol T1	Cortisol T2	Cortisol T3	Cortisol T4	Overall Cortisol	CAR
Affective	-0.12	-0.06	0.17	0.14	-0.03	-0.01
Anxiety	0.00	0.12	0.33**	0.15	0.14	0.15
Total Depression (D-Inventory)	-0.14	-0.11	0.1	0.18	-0.03	-0.05
Depression (AD + Total Depression)	-0.14	-0.09	0.15	0.18	-0.04	-0.03

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 3.10 Boys Correlations of Depression and Anxiety at each Cortisol Measure.

Measures	Cortisol T1	Cortisol T2	Cortisol T3	Cortisol T4	Overall Cortisol	CAR
Affective	0.03	0.11	-0.12	0.14	0.07	0.09
Anxiety	0.14	0	-0.14	0.15	0.08	-0.11
Total Depression (D-Inventory)	-0.14	-0.7	-0.06	0.18	0.01	0.04
Depression (AD + Total Depression)	-0.14 -0.08	0.01	-0.09	0.18	0.04	0.07

However, it was still possible that 5HTTLPR could moderate the association between cortisol

and depression. As such, moderated mediation analyses were conducted following the

procedures outlined by Preacher and Hayes (Preacher & Hayes, 2004). Model 1 was used to

determine if 5HTT moderates the relationship between peer victimization and biological

functioning.   Model 2 was used to determine if 5HTT moderates the relationship between

biological functioning and depression. Finally, model 3 was used to determine if 5HTT moderates the relationship between peer victimization and biological functioning, as well as the relationship between biological functioning and depression. Analyses were run separately for boys and girls given their differences in CAR. In all models, there was no evidence that biological functioning (i.e., CAR) mediated the relationship between victimization and depression. Furthermore, the 5HTTLPR did not moderate the proposed mediation. That is, there was no mediation for some variants of 5HTTLPR compared to other variants (see Table 3.11, 3.12, 3.13, 3.14, 3.15, and 3.16 for details).

Table 3.11 Conditional indirect effects at specific values of the moderator (Model 1) for CAR.

DNA_5HTT Alleles	Indirect Effect	SE	Z	P > I Z I
Boys				
S,S	0.04	0.06	0.59	0.56
S,L	-0.01	0.02	-0.34	0.73
S,L	-0.05	0.08	-0.62	0.54
Girls				
S,S	-0.02	0.42	-0.37	0.71
S,L	-0.01	0.24	-0.31	0.76
S,L	0.00	0.02	0.04	0.97

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 3.12 Conditional indirect effects at specific values of the moderator (Model 2) for CAR.

DNA_5HTT Alleles	Indirect Effect	SE	Z	P > I Z I
Boys				
S,S	0.00	0.03	0.08	0.93
S,L	-0.01	0.02	-0.51	0.61
S,L	-0.03	0.04	-0.71	0.48
Girls				
S,S	-0.00	0.03	-0.14	0.89
S,L	-0.00	0.02	-0.08	0.94
S,L	0.00	0.03	0.04	0.97

Table 3.13 Conditional indirect effects at specific values of the moderator (Model 3) for CAR.

DNA_5HTT Alleles	Indirect Effect	SE	Z	P > I Z I
Boys				
S,S	-0.02	0.08	0.23	0.82
S,L	-0.01	0.02	-0.29	0.78
S,L	-0.11	0.10	-1.10	0.29
Girls				
S,S	-0.04	0.07	-0.60	0.56
S,L	-0.01	0.02	-0.30	0.76
S,L	0.00	0.03	0.02	0.10

Table 3.14 Conditional indirect effects at specific values of the moderator for AUCg (Model 1)

DNA_5HTT Alleles	Indirect Effect	SE	Z	P > I Z I
Boys				
S,S	0.01	0.06	0.13	0.90
S,L	0.00	0.04	0.12	0.90
S,L	0.00	0.05	0.04	0.96
Girls				
S,S	0.01	0.06	0.13	0.90
S,L	0.00	0.40	0.12	0.91
S,L	0.00	0.05	0.04	0.96

** Correlation is significant at the 0.01 level (2-tailed).
 * Correlation is significant at the 0.05 level (2-tailed).

Table 3.15 Conditional indirect effects at specific values of the moderator (Model 2) AUCg.

DNA_5HTT Alleles	Indirect Effect	SE	Z	P > I Z I
Boys				
S,S	0.03	0.11	0.25	0.80
S,L	0.01	0.04	0.18	0.86
S,L	-0.01	0.06	-0.20	0.84
Girls				
S,S	0.03	0.11	0.25	0.80
S,L	0.01	0.04	0.18	0.86
S,L	-0.01	0.06	-0.20	0.84

Table 3.16 Conditional indirect effects at specific values of the moderator (Model 3) AUCg.

DNA_5HTT Alleles	Indirect Effect	SE	Z	P > I Z I
Boys				
S,S	0.03	0.15	0.19	0.85
S,L	0.01	0.04	0.13	0.90
S,L	-0.00	0.08	-0.05	0.96
Girls				
S,S	0.03	0.15	0.19	0.85
S,L	0.01	0.04	0.13	0.90
S,L	-0.00	0.08	-0.05	0.96

CHAPTER 4

DISCUSSION

The current thesis was a first step in trying to more fully understand the link between peer victimization and depression and anxiety. More importantly, this study focused on individual differences in susceptibility to depression and anxiety. First, I examined whether adolescents who are recipients of chronic peer victimization have abnormal cortisol levels and greater levels of depression and anxiety. Second, I tested whether variations in one functional polymorphism, namely 5HTT (serotonin transporter gene), led to higher levels of depression and anxiety. Third, I examined whether 5HTTLPR moderates the relationship between peer victimization with depression and biological functioning. Finally, I examined whether biological functioning mediated the link between victimization and depression. Additionally, I tested whether 5HTTLPR moderates the above predicted mediated relationship. The main goal of this thesis was to examine whether individual differences in 5HTTLPR predispose some individuals who are bullied to develop depression and anxiety as well as differences in endocrine functioning.

4.1 The Link Between Peer Victimization and Depression

Peer victimization has been a topic of increased interest in recent months with many news channels highlighting individuals who have been the targets of horrible peer victimization by their classmates. In fact, prior research has found that approximately 10-30% of children in this country are recipients of repeated peer victimization (Grills & Ollendick, 2002; Haynie et al., 2001; Nasel et al., 2001). In the current thesis samples, approximately 22.4% of participants

reported being bullied[5]. Given that approximately one out of every five adolescents is bullied, it is exceedingly important to look at the long term consequences of experiencing peer victimization. Research has found that those who are recipients of chronic peer victimization are much more likely to experience depression and anxiety (Bjorkqvist et al., 1982; Kochenderfer-Ladd & Ladd, 2001; Olweus, 1993). This thesis attempted to replicate as well as extend the findings between mental health problems and peer victimization.

As prior research has suggested and as expected, peer victimization was positively related to both depression and anxiety accounting for 43% and 3.8% of the variance respectively. Victimized adolescents in this thesis had greater levels of both depression and anxiety compared to their non-victimized counterparts.

Most notably, 5HTTLPR moderated the link between victimization and depression suggesting some children are more at risk for the negative outcomes associated with being the target of bullying. Adolescents with at least one s variant of the 5HTTLPR were more susceptible to depression when victimized. My finding matches previous work that has found that persons with at least one s-variant of 5HTTLPR were more vulnerable to internalizing problems when exposed to traumatic experiences. For example, Caspi and his colleagues (2003) found that individuals who experienced a stressful life event after their 21st birthday, and had a least one s variant of 5HTTLPR, had an increase in depressive symptoms between the ages of 21 to 26. Based on my findings, it would seem that an individual may not be buffered from stressful life events at any age. That is, even as early as adolescence, individuals with at least one s variant reported higher levels of depression when they experienced bullying. Moreover, peer victimization has often been considered a social stressor, and it would seem that this social stressor is profound enough to mimic similar responses to those seen for other major stressful life events (e.g. death of a significant other, loss of a job, etc).

For children with the l-l variant, I found no evidence that victimization was related to depression. Caspi and colleagues (2003) found a similar gene X environment interaction such that, individuals who experienced a stressful life event with the l-l variant did not show increases in depressive symptoms. This may suggest that having the l-l variant may have a buffering effect that protects an individuals from negative outcomes associated with major stressors.

Although the exclusion of our outliers did not seem to alter the data significantly, it did make the relationship between victimization and depression stronger. Moreover, the relationship between victimization and anxiety became significant, revealing results that mirrored what was predicted (Figure 4.1).

Susceptibility to Anxiety Without Outliers
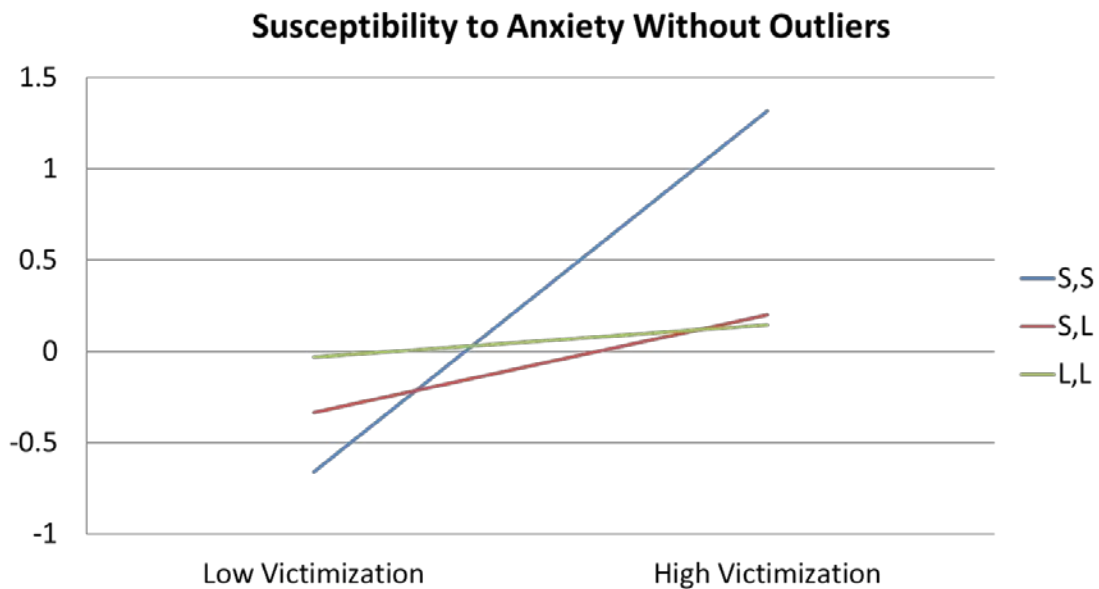
Figure 4.1. Participants' susceptibility to anxiety (outliers removed) as a result of peer victimization based on allele variant.

Additionally, examination of these 4 outliers may provide some key insight into reports of depression. Nolen-Hoeksema and Girgus (1994) found that after an individual reaches 15 years of age, girls are twice as likely as boys to be depressed. It may be possible that girls are more likely to report depression than boys, even if the boys are experiencing depression. Additional

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[5] This percentage is based on cluster analyses. Two groups of adolescents clustered based on

research will also need to examine whether other genes interact with 5HTTLPR to influence the victimization-depression link. For example, genes including MAOA and DRD1–DRD4 have also been implicated in mood disorders, especially when childhood adversity/stressful life events are present(Caspi et al., 2002). . Way & Taylor (2010) found that individuals having the low expressing allele of MAOA-uVNTR, when put in an fMRI, were seen to have the largest response within the dACC which is associated most often with self-reports of distress and hypersensitivity to threat. Interestingly, MAOA is sex-linked such that boys only receive one copy of the gene and are more likely to inherit a weaker version of the gene (Caspi et al. 2002).

4.2 Peer Victimization and Biological Functioning

As previously discussed, cortisol was assessed at: (1) the average cortisol level (AUCg); (2) waking cortisol (T2) (3) evening cortisol; and (4) the cortisol awakening response (CAR) or the difference between wakening and wake (+ 30 minutes). As previous research has reported gender differences in cortisol responses for victimized children (e.g., Knack, Jensen-Campbell, & Baum, in press; Vaillancourt et al., 2008), a model that included sex of participant as a moderator was first assessed. There was a gender X victimization interaction for overall daily cortisol levels. For boys, there was no evidence that victimization was related to overall cortisol. For girls, it appeared that victimization was negatively related to overall daily cortisol levels. Similar results were found for waking cortisol and CAR, where there was no evidence that victimization was related to waking cortisol for boys, and for girls, victimization was negatively related to waking cortisol.

Similar findings were reported by Knack et al (in press.) who found that cortisol diurnal patterns differed for adolescent boys and girls and further, they found that peer victimized girls had a damped CAR. Vaillancourt and colleagues (2008) also found differences in overall cortisol responses between boys and girls who were bullied. For boys, it appeared that verbal

their victimization scores: a non-victimized group (N = 97) and a victimized group (N = 28).

bullying was associated with greater cortisol production. Conversely, for girls, verbal bullying was associated with lower cortisol production. Interestingly, Vaillancourt et al. (2008) proposed that differences in cortisol production that occur as a result of types of victimization may be due to the fact that for males, social goals tend to value dominance, while female social goals tend to value social inclusion. Further, Paquette and Underwood (1999) found that female adolescents often reporte more negative thoughts and feelings associated with being victimized than do males. These findings suggest that adolescent boys and girls may be affected differently by bullying, which results in bullied girls producing less cortisol (Vaillancourt et al., 2008). It should be noted that lower cortisol levels, or hypocortisolism, have been linked to traumatic events such as PTSD (Heim et al., 2000). Breslau et al. (1999) found that women were at a higher risk for developing PTSD even after controlling for type of traumatic event. This finding is supported by other research and may provide a reason for differences noted between gender in our sample.

<u>4.3 Peer Victimization, Biological Functioning, and Genetic Polymorphisms</u>

5HTTLPR was also expected to moderate the association between victimization and biological functioning and once again that this pattern would be different for boys than it would be for girls. Serotonin (5HT) has been implicated in many physiological processes including neuroendocrine rhythms and functioning. For overall cortisol, there was a sex X 5HTTLPR x victimization. Bullied girls with the s-s variant of 5HTTLPR had lower daily cortisol levels. Conversely, there was no association between victimization and daily cortisol levels for girls with the s-l and l-l variants. There was also a sex X 5HTTLPR X victimization interaction for waking cortisol and CAR. Again, bullied girls with the s-s variant had lower waking cortisol and a flatter CAR.

Differences in diurnal patterns are not uncommon. Cichetti and Rogosch (2001) found that children who had experienced physical maltreatment had lower levels of waking cortisol than did those children who had not experienced physical maltreatment. Knack et al. (in press)

found that children who were recipients of chronic peer victimization had altered cortisol production in waking cortisol and CAR. Additionally, Tops et al. (2008) put forth the "cortisol mobilization response" which outlines the basic patterns of cortisol throughout the day. Usually individuals have an increase in cortisol which happens around 30 minutes after waking. The waking (+30 minute) assessment is usually the peak in a person's overall diurnal pattern. Typically cortisol levels tend to gradually drop throughout the rest of the day. The spike seen in the waking (+30 minute) assessment may possibly reveal something to us about prevalence of stress for an individual. Tops et al. found that those women who had a high fear of negative social evaluation and thus had higher levels of social stress, had lower cortisol awakening responses than did those women who had a lower fear of negative social evaluation (i.e., lower levels of social stress). Similarly, a study conducted by Barnett, Steptoe, and Garieis (2005) found that adults particularly women, reported higher levels of stress from dealing with more marital conflict than those adults who were not dealing with marital conflict, which was coupled with a flatter cortisol awakening pattern. The above research may provide an explanation for the finding that bullied girls with the s-s variant have lower levels of cortisol and a flatter CAR. Once again, something about having the s-s variant may put bullied girls at risk for developing abnormal cortisol patterns, which could lead to a other health problems besides depressive symptoms (e.g., Knack, Jensen-Campbell, & Baum, in press). Hanse et al. (2002) found that individuals who were recipients of workplace bullying had lower levels of cortisol and subsequently poorer health outcomes.

### 4.4 Moderation of 5HTT in Peer Victimization and Biological Functioning

Model 1 was used to determine if 5HTT moderates the relationship between peer victimization and biological functioning. Model 2 was used to determine if 5HTT moderates the relationship between biological functioning and depression. Finally, model 3 was used to determine if 5HTT mediates/moderates the relationship between peer victimization and

biological functioning, as well as the relationship between biological functioning and depression. In all models, there was no evidence that biological functioning (i.e., CAR) mediated the relationship between victimization and depression. Furthermore, the 5HTTLPR did not moderate the proposed mediation. That is, there was not mediation for some variants of 5HTTLPR compared to other variants.

However, it seems that 5HTT is moderating the relationship between victimization and biological functioning as well as moderating the relationship between peer victimization and depression (see Figure 4.2).
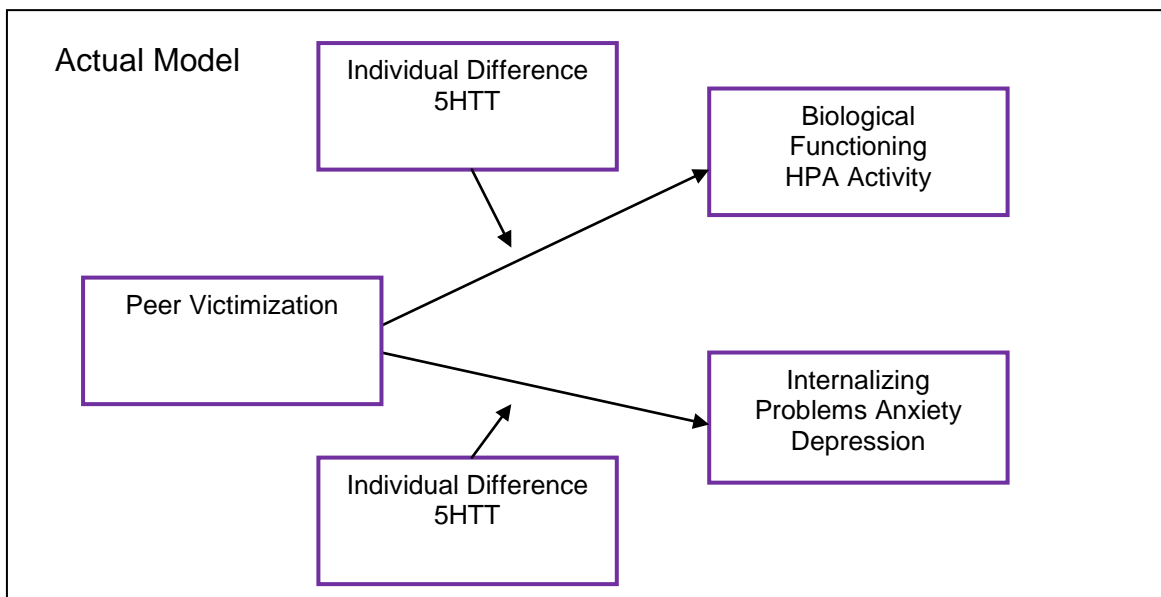
Figure 4.2. Actual Model for 5HTT

It is possible that because serotonin influences many desperate psychological (e.g., emotional states) and physiological processes (e.g., sleep, neuroendocrine functioning, food intake, etc), the social stress of bullying, especially for children with the s-s variant, leads to a myriad of problems associated with serotonin transmission that do not necessarily mediate one another. In this study, bullied children with the s-s variant had higher levels of depression as well as

44

differences in daily cortisol levels. It is possible that abnormal cortisol levels may indeed mediate other health problems that were outside the scope of this paper, namely physical health problems (e.g., Knack, Jensen-Campbell, & Baum, in press). Indeed, other studies have found associations between lower cortisol levels and health problems (Hanse et al. (2002). Future research needs to examine these possibilities.

It is also possible that some of these adolescents are developing symptoms associated with PTSD in conjunction with depression which in turn, is altering biological functioning. Interestingly, these null results in the mediation model may actually be very telling. Posttraumatic Stress Disorder (PTSD) is experienced after the exposure to an extremely traumatic event (DSM-IV). Yehuda and colleagues (1996) conducted a study to examine differences in cortisol between combat veterans with PTSD, and individuals diagnosed with major depression. They found combat veterans with PTSD had significantly lower cortisol levels than any of the other two groups. More importantly they concluded that combat veterans with PTSD in a normal setting may have an overstated sensitization, which in turn is causing them to produce lower levels of cortisol. This system differs from those individuals with just depression, whose cortisol production differences may be the result of alterations and deregulation that occur to the hypothalamic-pituitary-adrenal (HPA) axis as a result of the depression.

Interestingly, researchers have found that individuals who are peer victimized have some symptoms of PTSD. For example, Stroch & Esposito (2003) reported that children who were both overtly and relationally bullied, had a greater number of symptoms of posttraumatic stress. Moreover, research from Knack, Jensen-Campbell, and Baum (in press), Vaillancourt et al. (2008) and the current research study all found cortisol patterns that match the cortisol patters of individuals with symptoms of PTSD. Additionally the patterns of cortisol are different for individuals with PTSD and depression than individuals diagnosed with just depression (Yehuda et al., 2004), particularly in studies where females are involved (Meewisse et al., 2007).

45

Based on this, it is possible that some of our participants are experiencing PTSD as well as depression. As both of these disorders affect the body differently, and ultimately may produce completely different changes in biological functioning and levels of cortisol, it may not be surprising that the proposed mediation models did not work.

4.5 Future Directions

This was an initial attempt at examining why the peer victimization-depression link is so profound for some adolescence but not others, by introducing the possibility of a gene x environment interaction.

Although there are many interesting and important findings, there should still be caution exercised when generalizing the results of the above study. Although I found that the 5HTTLPR variations moderated the effects of peer victimization on depression this should not be mistaken for genes causing behavior. As often noted, many genes interact with the environment to produced specific outcomes. What we are suggesting here however, is that having the s-s variant may make an individual more susceptible to depression after being peer victimized. Moreover, my population of participants with the s-s variant (22.4%) although similar to the percentages of the overall population, is still small, and thus my findings should be replicated in a larger sample

Additionally, although I was predicting a directional model, it is important to keep in mind that this data is correlational and concurrent. As such, alternative or longitudinal models should be considered as part of future studies. Based on previous research however, it has been suggested that victimization leads to greater health problems over time and not vice versa (e.g., Egan & Perry, 1998; Fekkes et al., 2006; Knack, Iyer, & Jensen-Campbell, in press). For example, although children with low self-esteem are more likely to be victimized, victimized children show decreases in self-esteem over time (Egan & Perry).

Even with these limitations, this thesis advances the field of psychology by demonstrating that the negative consequence of peer victimization may not simply occur as a

46

result of something that is in the child's head and something that these kids will simply "get over." In fact, the research here helps combat the belief that being harassed by one's peers is merely just a normal part of one's life. Rather, the current findings provide early evidence that human genetic variation may have an effect on expression of depression and more importantly, bring us one step closer to understanding why some children are more adversely affected by bullying than are other children.

APPENDIX A

FIGURES
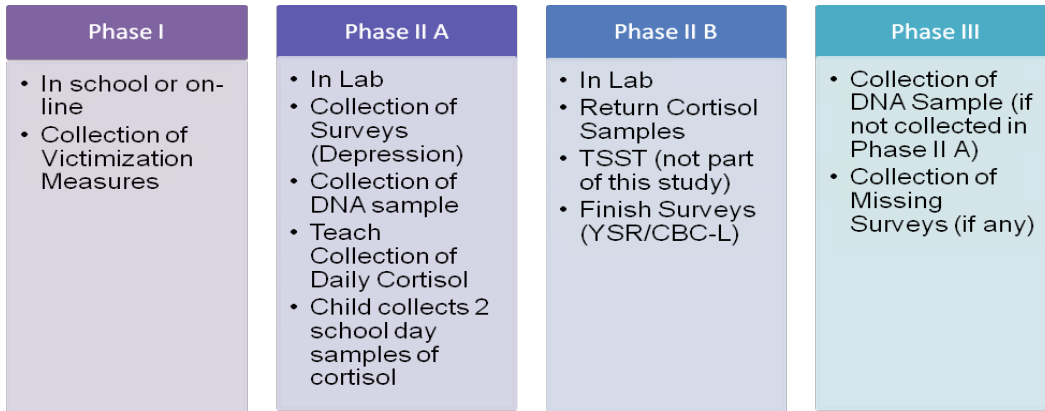
Figure 1. Timeline of Project
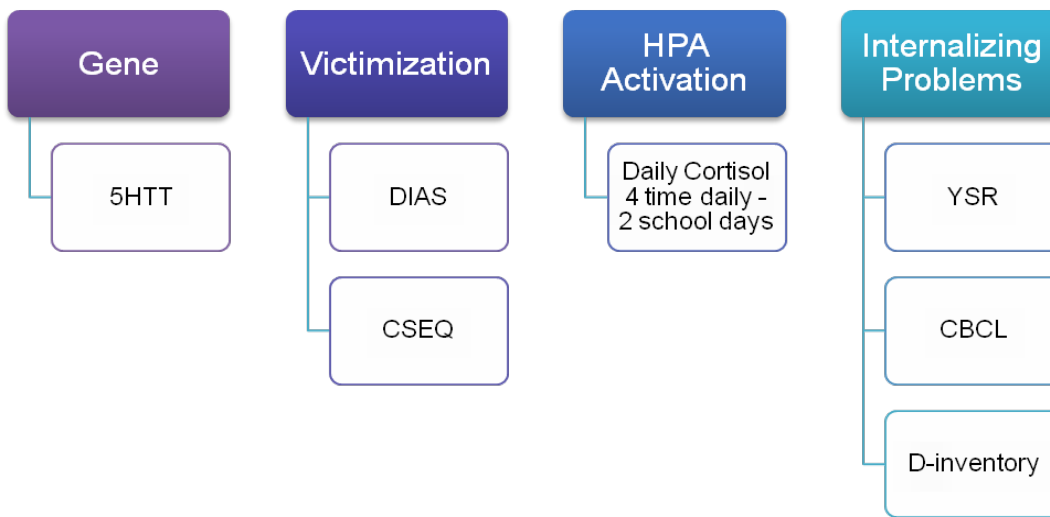
Figure 2. Constructs of Interest and Their Measures

APPENDIX B

MODELS

Model 1

```
          ┌─────────────────┐
          │   Individual    │
          │   Differences   │
          │      5HTT       │
          └────────┬────────┘
                   │
                   ▼
┌──────────────┐  ┌──────────────────┐  ┌──────────────────────┐
│ Peer         │  │ Biological       │  │                      │
│ Victimization│─▶│ Functioning      │─▶│ Internalizing Problems│
│(Environmental│  │ HPA Activity     │  │ Anxiety              │
│ Stress)      │  │                  │  │ Depression           │
└──────────────┘  └──────────────────┘  └──────────────────────┘
```

Model 2

```
                              ┌─────────────────┐
                              │   Individual    │
                              │   Differences   │
                              │      5HTT       │
                              └────────┬────────┘
                                       │
                                       ▼
┌──────────────┐  ┌──────────────────┐  ┌──────────────────────┐
│ Peer         │  │ Biological       │  │ Internalizing Problems│
│ Victimization│─▶│ Functioning      │─▶│ Anxiety              │
│(Environmental│  │ HPA Activity     │  │ Depression           │
│ Stress)      │  │                  │  │                      │
└──────────────┘  └──────────────────┘  └──────────────────────┘
```

Model 3

Individual
Differences
5HTT

Peer Victimization
(Environmental
Stress)

Biological Functioning
HPA Activity

Internalizing Problems
Anxiety
Depression

APPENDIX C

TABLES

C.1 Girls Correlations of Depression and Anxiety at each Cortisol Measure

Measures	Cortisol T1	Cortisol T2	Cortisol T3	Cortisol T4	Overall Cortisol	CAR
Affective	-0.12	-0.06	0.165	-0.06	-0.03	-0.01
Anxiety	-0.00	0.12	0.33**	-0.04	0.14	0.15
Total Depression (D-Inventory)	-0.14	-0.11	0.10	0.07	-0.03	-0.05
Depression (AD + Total Depression)	-0.14	-0.09	0.15	-0.01	-0.04	-0.03

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

C.2 Boys Correlations of Depression and Anxiety at each Cortisol Measure.

Measures	Cortisol T1	Cortisol T2	Cortisol T3	Cortisol T4	Overall Cortisol	CAR
Affective	0.03	0.11	-0.12	0.14	0.07	0.09
Anxiety	0.14	0.00	-0.14	0.15	0.08	-0.11
Total Depression (D-Inventory)	-0.14	-0.7	-0.06	0.18	0.01	0.04
Depression (AD + Total Depression)	-0.14	0.01	-0.09	0.18	0.04	0.07

APPENDIX D

MEASURES

DIAS –VS

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   2 seldom   3 sometimes   4 quite often   5 very often

1. How often are you hit by other classmates?
2. How often are you shut out of the group by other classmates?
3. How often do other classmates yell at you or argue with you?
4. How often do classmates become friends with another classmate as a kind of revenge?
5. How often are you kicked by other classmates?
6. How often are you ignored by other classmates?
7. How often are you insulted by other classmates?
8. How often do classmates who are angry with you gossip about you?
9. How often are you tripped by other classmates?
10. How often do classmates tell bad or false stories about you?
11. How often do classmates say they are going to hurt you?
12. How often do classmates plan to secretly bother you?
13. How often are you shoved by other classmates?
14. How often do classmates say bad things about you behind your back?
15. How often are you called names by other classmates?
16. How often do classmates tell others "Let's not be friends with him/her!"?
17. How often do other classmates take things from you?
18. How often do classmates tell your secrets to a third person?
19. How often are you teased by other classmates?
20. How often do classmates write small notes where you are criticized?
21. How often are you pushed down to the ground by other classmates?
22. How often do other classmates criticize your hair or clothing?
23. How often do other classmates pull at you?
24. How often do classmates who are angry with you try to get others to dislike you?

Things that Happen to Me at School

(CSEQ-SR; Crick & Gropeher, 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   4 = almost all the time
2 = almost never   5 = all the time
3 = sometimes

1. How often does another kid give you help when you need it?
2. How often do you get hit by another kid at school?
3. How often do other kids leave you out on purpose when it is time to play or do an activity?
4. How often does another kid yell at you and call you names?
5. How often does another kid try to cheer you up when you feel sad or upset?
6. How often does a kid who is mad at you try to get back at you by not letting you be in their group anymore?
7. How often do you get pushed or shoved by another kid at school?
8. How often does another kid do something that makes you feel happy?
9. How often does a classmate tell lies about you to make other kids not like you anymore?
10. How often does another kid kick you or pull your hair?
11. How often does another kid say they won't like you unless you do what they want to do?
12. How often does another kid say something nice to you?
13. How often does a kid try to keep others from liking you by saying mean things about you?
14. How often does another kid say they will beat you up if you don't do what they want you to do?
15. How often do other kids let you know that they care about you?

D-Inventory (Depression Inventory)

Directions: Indicate the intensity that you experience each of the following symptoms.

0 = never   3 = always

1. I feel sad often.
2. I am often depressed.
3. I see life as mostly a negative.
4. I feel like I fail often.
5. I am unhappy with the way that I am.
6. I am often disappointed with myself.
7. I like the way that I am leading my life.
8. I wish that I was someone else.
9. I am happy with the way that I am.
10. I cry often.
11. I am often moody and cranky.
12. I am usually very happy.
13. I don't like to be around other people.
14. I have a hard time making decisions.
15. I am not happy with the way that I look.
16. I have difficulty sleeping at night.
17. I often feel tired and have no energy.
18. I do not have much of an appetite (i.e., not hungry).
19. I often feel down.
20. I often feel lucky for who I am.

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