WHAT ARE THE LONG-TERM CONSEQUENCES OF PEER VICTIMIZATION? CHANGES IN BIOLOGICAL FUNCTIONING AND ITS EFFECTS ON PSYCHOLOGICAL AND PHYSICAL HEALTH

by

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Abstract

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Prior research has documented long-term psychological problems (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 peer victimization has been related to psychological health, limited research has looked at the association between peer victimization and physical health problems. Even fewer studies have investigated the relationship between being bullied and health outcomes over time. This dissertation examined whether peer victimization in early adolescence lead to changes in neuroendocrine functioning (as assessed by cortisol) and physical health outcomes several years later. Adolescents and their parents (N = 120) participated in this two phase study occurring over an approximate 2.5 year period. At the first assessment, adolescents and their parents answered questions about the child's social experiences, and psychological and physical health. In addition, the adolescent collected saliva samples (to assess cortisol levels). At the second assessment, adolescents and their parents completed identical surveys and adolescents completed an additional two days of cortisol samples. As expected, being peer victimized at the first assessment was

related to poorer psychological and physical health problems as the second assessment. Moreover, this study found that peer victimization at time 1 was related to altered diurnal patterns of cortisol over time. Finally, this dissertation assessed whether changes in cortisol mediate the relationship between victimization and health.

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Chapter 1

Introduction

The relationship between early adverse experiences and its implications on subsequent negative psychological and physical health outcomes like deficits in cognitive behavior, alterations in endocrine system functioning, brain activation, and the formation of attachments have gained increased interest. Orphanages involving extreme social and resource deprivation have served as models to study these implications long-term. Within the last 20 years, studies have followed the children who lived in these orphanages during the early parts of their lives and were later placed in foster care or were adopted into permanent homes (Smyke et al., 2007). Specifically, randomized controlled trials were used to parallel the developmental trajectories between those children that were abandoned and raised within orphanages in Romania versus those children that were institutionalized and then were later moved to foster care. The cognitive development (i.e. IQ, mental and motor development) of all children in this study was followed until they reached 54 months of age (Nelson et al., 2007). The results from this study found, as expected, that the cognitive outcomes for children who continued to live in orphanages were severely lessened as compared to children who were never institutionalized or who were later placed in foster care. Most interesting was the developmental trends for the children who were previously in orphanages and were later moved into foster care. Although placed within loving and enriching environments within the first few years of life, the cognitive developmental outcomes for this group of children never reached the developmental trajectory of non-institutionalized children. This study indicates that there is a critical period of development that occurs very early in life (Nelson et al., 2007).

Even more telling may be the changes in both the structure and function of the brains for those children in the orphanages. Specifically, the times these children spent in

the orphanages, resulted in overall lower quality brain activation in comparison to those children who were adopted out earlier or who were never institutionalized (Vanderwert, Marshall, Neson, Zeanah, & Fox, 2010). Children who were institutionalized for any period of time evidenced altered endocrine system functioning (i.e., diurnal cortisol patterns; Carlson & Earls, 1997; Gunnar, Morison, Chisholm, & Schuder, 2001). Specifically, 22% of Romanian orphanage children even though adopted into permanent homes, exhibited increased diurnal cortisol that was two standard deviations higher than the average. Moreover, the longer beyond 8 months that a child remained institutionalized before adoption, the higher their overall cortisol levels were six and half years after adoption (Gunner et al., 2001).

This extreme exemplar provides compelling evidence that chronic stress experienced early in life may lead to relatively permanent changes in an individual's psychological and physical health. Although being bullied is not nearly as severe a stressor as early deprivation, to the extent that being bullied by one's peers in adolescence is a chronic stressor, it should also lead to relatively permanent changes in biological functioning and health outcomes. As such this dissertation sought to examine the influence of being bullied on health outcomes and endocrine system functioning over a 2.5 year period.

Indeed, more commonplace stressors, such as low SES or poor parenting, have been linked to changes in biological functioning. Graham, Christian, and Kiecolt-Glaser (2006) found that experiences of early life stress such as poor marriage quality can increase the likelihood of maladaptive immune responses to stress in later life. Maltreated also children show relatively permanent changes in their biological functioning and health when they are older (Cicchetti & Hinshaw, 2002, Cicchetti & Rogosch, 2001). Finally, Ouellet-Morin et al. (2008) found both genetic and environmental contributions to cortisol

reactivity when taking into account adverse family environments (e.g. maternal smoking during pregnancy, low family income, low birth weight, low maternal education level, single parenthood, young motherhood, and maternal hostile or reactive behaviors). That is, high familial adversity (poor familial environments) partially accounted for differences in cortisol reactivity, especially for children who were genetically at risk for poorer developmental outcomes. Taken together, this research suggests biological systems are malleable and can be negatively influenced by harmful environmental factors.

Following the above line of thought, the plasticity of the developing biological system may put an individual at risk when exposed to harmful environments (Cicchetti & Tucker, 1994). More specifically, individuals' environmental factors can modify the traditional course of development and thus, cause them to be more vulnerable to future life experiences (Cicchetti & Walker, 2003; Dersh, Polatin, & Gatchel, 2002).

With the widespread attention devoted to peer victimization, one may not argue with the notion that being chronically peer victimized can severely impact an individual. Indeed, numerous research studies have documented the long-standing and often detrimental effects of peer victimization on an individual's psychological health. The stress experienced as a result of chronic exposure to peer victimization is a somewhat new area of study. Even less studied, is the relationship that peer victimization has to overall health as well as understanding the biological mechanisms that underlie this relationship longitudinally.

My model (Figure 1) follows the general format of many biobehavioral investigations of health and illness (Dougall & Baum, 2012). As seen in Figure 1, stress is a mechanism by which stressors set in motion the kinds of changes that can affect both physical and psychological health long-term. Using this model, this dissertation will attempt to fill in the gaps of the current literature by examining the association between

peer victimization, neuroendocrine functioning, and physical health in a group of adolescence over time. This study will also examine whether victimization influences diurnal patterns of cortisol (as a biological measure of stress) in individuals longitudinally. Finally, the current dissertation will examine whether changes in cortisol patterns are associated with changes in health over time, and more specifically whether cortisol levels mediate the victimization-health link longitudinally.

Definition of Peer Victimization

Recently, it seems that bullying has become a well-followed social problem; however, the concept of bullying is not something new. Dan Olweus in the 1970s conducted the largest wide scale study of peer victimization in Norway. His study included 80,000 children between the ages of 7 to 16 years old and revealed that approximately 15% of children in his sample reported experiencing bullying (Olweus, 1991). Boulton and Smith (1994) also assessed peer victimization in England's schools with their sample of 6,000 students. Approximately 27% of their sample reported experiencing peer victimization. These percentages are similar to peer victimization reported in secondary schools in Australia (K. Rigby & Slee, 1999) . A study looking at the prevalence of school bullying in Korea reported that 40% of all children in the school were apart of school bullying in some way. Within this sample, approximately 14% were victims of chronic peer abuse, 17% were perpetrators of that abuse, and an additional 9% were both victims and perpetrators of bullying (Kim Y, 2004). Current rates of peer victimization in the United States vary from 10-30% at school (Nansel et al., 2001). The above research suggests the uniformity and consistency of peer victimization regardless of the culture and decade in which it is studied.

Prevalence of Peer Victimization

Recently, it seems that bullying has become a well-followed social problem; however, the concept of bullying is not something new. Dan Olweus in the 1970s conducted the largest wide scale study of peer victimization in Norway. His study included 80,000 children between the ages of 7 to 16 years old and revealed that approximately 15% of children in his sample reported experiencing bullying (Olweus, 1991). Boulton and Smith (1994) also assessed peer victimization in England's schools with their sample of 6,000 students. Approximately 27% of their sample reported experiencing peer victimization. These percentages are similar to peer victimization reported in secondary schools in Australia (K. Rigby & Slee, 1999) . A study looking at the prevalence of school bullying in Korea reported that 40% of all children in the school were apart of school bullying in some way. Within this sample, approximately 14% were victims of chronic peer abuse, 17% were perpetrators of that abuse, and an additional 9% were both victims and perpetrators of bullying (Kim Y, 2004). Current rates of peer victimization in the United States vary from 10-30% at school (Nansel, et al., 2001). The above research suggests the uniformity and consistency of peer victimization regardless of the culture and decade in which it is studied.

Types of Peer Victimization

Peer victimization is an umbrella term used to encompass several different facets or types of abuse and has come to include a broad spectrum of actions and behavior from overt, noticeable, physical or verbal acts of aggression, to a more understated, subtle, consistent occurrence of a verbal or relational act of aggression (Feinberg, 2003). A child being pushed by another child into a locker repeatedly and a child having malicious rumors continuously being spread about them are both experiencing peer victimization. What differs is the delivery of victimization. Indeed, peer victimization has

often been categorized into several subtypes, and can manifest in terms of either direct abuse or indirect abuse (Crick & Grotpeter, 1995) and can be either physical, social, or verbal in nature (Marion K. Underwood, 2003).

Peer victimization can be experienced in any of these forms, or in combination thereof (Crick & Bigbee, 1998; Mynard & Joseph, 2000) and research suggests that all types of victimization have psychological consequences (Paquette & Underwood, 1999). For completeness, several forms of bullying will be examined in this dissertation. However, it is anticipated that all types of victimization will be associated with biological functioning and health outcomes in similar ways.

Consequences of Peer Victimization

Bullying has often been associated with lower self-esteem, impaired concentration, truancy, and suicidal thoughts (Bond, Carlin, Thomas, Rubin, & Patton, 2001). Additionally, research indicates that being bullied is linked to 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). These patterns have been noted cross-culturally. Bullied children between the ages of 9 -12 years old in Greece (Andreou, 2001) and South Korea (Schwartz, Farver, Chang, & Lee-Shin, 2002) reported lower feelings of self-worth and academic functioning in comparison to their non-bullied peers. Additionally, both studies noted a positive correlation between chronic peer victimization and behavioral problems. Research conducted in England (Mynard, Joseph, & Alexander, 2000) and Australia (K. E. N. Rigby, 2000) also found that victimization was related to increased psychological distress (e.g., depression, loneliness, and anxiety).

Peer Victimization and Internalizing Problems

Internalizing problems are one class of psychological outcomes that have been repeatedly associated with victimization. Internalizing problems consists of factors related to personal distress as well as self-control issues, and behavioral inhibition (e.g. anxiety, depression, loneliness, 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 et al., 1999; Crick & Grotpeter, 1996; Janosz et al., 2008). Kochenderfer-Ladd and Skinner (2002) found that peer victimized school age children were at increased risk for developing the internalizing problems of childhood depression, loneliness, and anxiety.

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). A recent meta-analysis conducted by Iyer, Scielzo, and Jensen-Campbell (under review) assessed the association between peer victimization and the internalizing problems of depression, anxiety, and loneliness. Results indicated a significant positive relationship between peer victimization and all three types of internalizing problems. Specifically, the meta-analysis indicated that there was a positive relationship between peer victimization and anxiety ($\rho corr = .29$), depression ($\rho corr = .35$), and loneliness ($\rho corr = .39$).

Olweus (1992) conducted a longitudinal study on mental health of young adults in Sandinavia. He found a significant relationship between peer victimization and scores of depression. The association between victimization and depression seems to persist 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 vicious cycle of bullying and depression for these children (e.g., Egan & Perry, 1998).

Perhaps more ominous is the fact that depression is thought to be associated with poorer physical health outcome. Indeed, some researchers argue that depression precedes physical health problems and as such, may even cause health problems (Fekkes et al., 2006). Aneshensel, Carol, Ralph, Frerichs, and Huba (1984) found that increases in depressive symptoms, over and above initial levels of depression, were significantly related to increased reports of physical health problems. He also noted the reciprocal relationship between depression and physical health. Along these lines, Beekman et al. (1997) found a co-morbid relationship between depression and poor physical health. Individuals who were depressed were more likely to report poorer physical health, and those with poorer physical health were more likely to report being depressed. This highlights the relationship depression can have to symptoms of physical illness. In this dissertation, it is expected that victimized adolescents will report more internalizing problems than those that are not recipients of chronic peer victimization. Moreover, it is anticipated that victimized adolescents at time 1 (T1) will report increases in internalizing problems at time 2 (T2).

Victimization and Health

In addition to negative psychological health outcomes, peer victims also suffer deficits in their physical health. Numerous research studies have found an association between peer victimization and physical health problems (Greco, Freeman, & Dufton, 2007; Williams, Chambers, Logan, & Robinson, 1996). Specifically, in their sample of Australian adolescents, Rigby and Slee (1999) found those that experienced chronic peer victimization reported both poorer mental and physical health three years later in

comparison to adolescents who had not experienced chronic peer victimization. Based on the above, it is possible that abnormal cortisol levels may mediate the relationship between peer victimization and other health problems. Knack, Jensen-Campbell, and Baum, (2011) found that victimized children were likely to show differences in neuroendocrine functioning which, in turn, predicted poorer health outcomes. Specifically they reported increases in self-reports of poorer physical health by peer victims in comparison to adolescents who did not experience peer victimization. It is anticipated that victimized adolescents will report poorer health over time then adolescents who are not victimized.

Stresses Effect on the Neuroendocrine System

Cicchetti and Tucker (1994) proposed that the plasticity of biological systems is responsible for the negative 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 negative 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 poor physical health. 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.

The hypothalamic-pituitary-adrenal (HPA) system controls reactions to physical and social stress (Dickerson & Kemeny, 2004; Eisenberger, Taylor, Gable, Hilmert, & Lieberman, 2007; Purvis & Cross, 2006). Activation of the HPA axis 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 adrenocorticotropic hormones (ACTH) into the bloodstream. ACTH then begins its descent to the adrenal glands to release cortisol (McEwen et al., 1997).

Cortisol is a hormone that is an end product of hypothalamic-pituitary-adrenal (HPA) axis activation and is associated with both regulatory roles (e.g. metabolism) as well as with stress reactions (Lovallo & Thomas, 2000). Its production is essential to the management of daily stressors. However, increased production and exposure to high levels of cortisol over long periods of time can be detrimental and has been related to degeneration in hippocampal dendrites (Sapolsky, Uno, 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, 2012; Gump & Matthews, 1999).

In addition to diurnal patterns of cortisol, research has also begun to pay close attention to the cortisol awakening response, also known as CAR. Tops, Riese, Oldehinkel, Rijsdijke, and Ormel (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. Specifically, the peak in cortisol is thought to prepare us for the day. Changes noted in peaks or flattening of CAR seems to be related to several poor physical and psychological health problems. Knack et al. (2011) found that children who were recipients of chronic peer victimization had altered cortisol production in waking cortisol and CAR. More specifically, bullied children showed a flattened CAR, which was similar to what has been found in individuals diagnosed with PTSD as well as other detrimental health conditions like chronic fatigue syndrome (Wessa, Rohleder, Kirschbaum, & Flor, 2006; Fries, Dettenborn, & Kirschbaum, 2009). Similarly, 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; Topps et al., 2008). Likewise, 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. This finding was coupled with a flatter cortisol awakening pattern for adults dealing with high levels of stress from experiencing more marital conflict than those adults that were not. Finally, research has also indicated that individuals who were recipients of workplace bullying had lower levels of cortisol and subsequently poorer health outcomes (Hansen et al., 2006). It is expected that adolescent victims will show altered cortisol as specifically assessed via the cortisol awakening response and overall cortisol.

Link between Peer Victimization and Physical Health

A recent study of 154 12-year-olds found that both occasional and frequent verbal abuse by peers produced altered HPA activity in comparison to those adolescents who were not victims of peer abuse (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., 2006). Research has also provided links between lower awakening cortisol and posttraumatic stress disorder (PTSD) as well as with chronic fatigue (Hansen et al., 2006). Finally, Knack et al., (2011) found that victimized children were likely to show differences in neuroendocrine functioning which, in turn, predicted poorer health outcomes in a concurrent sample of adolescents.

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 system functioning, which leads to poorer health problems (Cicchetti & Rogosch, 2001; DeBellis et al., 1999). As such, peer victimization should be associated with differences in neuroendocrine functioning as well as differences in overall self-reports of health. It is expected that within this dissertation, victimized adolescents will report worse physical health outcomes over time as compared to those that were not victimized.

Long-term Health Consequences of Peer Victimization

Research is beginning to document the long-term effects peer victimization has on physical health outcomes. Knack, Iyer, and Jensen-Campbell (2012) found that fall reports of victimization in a group of college undergraduates predicted poorer physical health in the spring whereas poor physical health in the fall did not predict increased victimization in the spring. Peer victimization has also been shown to produce long-lasting health consequences. Specifically, Rigby & Slee (1999) reported that victimization status in adolescences predicted elevated scores of depression more than six years later. More recently, Kivimaki et al. (2004), found that individuals who were in high stress work

environments and who were also recipient of workplace bullying, were more likely to be diagnosed with fibromyalgia. Moreover, Rigby and Slee (1999) conducted a longitudinal study in Australian middle schools looking at peer victimization and self-reported measures of health. They found that victimized children reported poorer health than their non-victimized counter-parts three years after an initial assessment. Interestingly, Rigby & Slee (1999) found that experiencing victimization during earlier years was more significantly associated with poorer physical health three years later than those reporting victimization later in life. This indicates long-term implications of peer victimization. More specifically, this study suggests that repeated exposure to peer victimization during critical time periods of early development might have long-lasting consequences for physical and psychological well-being. However, although research has looked at physical health over time, virtually no study has looked at the possible mechanisms responsible for changes in biological functioning and its later impactions in changes in health outcomes. Moreover, limited research has assessed possible gender differences in this relationship. Based on the above literature, it is anticipated that being victimized will lead to overall psychological and physical health problems over time.

Gender differences in Peer Victimization

Initially, research indicated that boys in general tend to be more aggressive in nature than girls (Coie & Dodge, 1998). It has since been found that girls and boys may evidence different types of aggression with boys using attempting to hurt their peers' status while girls use forms more intended to harm their peers' relationships (Crick & Bigbee, 1998). A recently conducted meta-analysis found gender differences associated with females engaging in more social forms of victimization. However, the results indicated that gender did not actually moderate the relationship between social or

physical forms of aggression and subsequent adjustment outcomes (Card, Stucky, Sawalani, & Little, 2008).

Most interesting perhaps is that peer victimization may affect boys and girls differently. Underwood (2003) suggests that although the impact of the relationship between gender and social aggression may be small, the significance of such differences may affect boys and girls in essential ways. That is, she suggests differences in social aggression is evidenced by differences in social processes, functions, and consequences that each gender experiences (M.K. Underwood & Rosen, 2011). Further, Craig (1998) found that adolescent girls tended to report more depression as a result of peer victimization than males. These results are similar to that found in a meta-analysis by Iyer (under review). Specifically, girls experienced more depression and anxiety than did boys when bullied. Interestingly, this relationship was not found in reports of loneliness.

Further, Paquette and Underwood (1999) established that female adolescents often reported 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. As such, it may be exceedingly important to assess the differences in consequences for boys and girls who experiencing peer victimization. It is anticipated that there may be possible gender differences noted between boys and girls in their reports of psychological and physical health over time.

Why Study Adolescents?

Although individuals are developing throughout the lifespan, it is important to address the time period of adolescence. Friendships during adolescences are extremely important. Individuals who have an inability to maintain close friendships maybe at increased risk for suffering from the negative outcomes associated with peer victimization. Indeed, those children without a best friend and are peer victimized, are

more likely to evidence both internalizing and externalizing problems than those individuals with a best friend (Hodges, Boivin, Vitaro, & Bukowski, 1999) who are also peer victimized.

Although people can be bullied at any point in the lifespan, adolescence is a particularly important developmental period to study. As of 2009, adolescents were a part of the largest group in the world at 1.2 billion, accounting for approximately 18% of the world population (Unicef, 2011). The sheer number of adolescents sheds light on the significance of studying this age group. Of greater consequence, early adolescence is associated with significant changes in biological, cognitive, and social functioning that are rivaled only by those changes seen in infancy (Larson & Richards, 1994). For example, early adolescent children undergo a number of major physiological changes including puberty, changes in the endocrine and immune systems, and rapid brain maturation (Golub, 2008; Walker, Walder, & Reynolds, 2001). Adolescents also undergo a number of changes in their social environment (Larson & Richards, 1994). Peers and friends begin to play an increasingly important socialization role (Hartup, 1996). As friendship intimacy increases during adolescence and supplements intimacy with parents, adolescents also begin to rely more on friends to satisfy needs and solve problems that arise (Furman & Buhrmester, 1985). Both longitudinal and cross-sectional studies have found that peer victimization also peaks during this time (Nansel et al., 2001; Nylund, Asparouhov, & Muthen, 2007). Additionally, the onset of mental illnesses, such as depression, anxiety disorders, and mood disorders, increases substantially during adolescence as a result of a greater vulnerability to stress that occurs as the adolescent goes through major physiological changes (e.g., Pause et al., 2008). Adolescent anxiety and depressive disorders show an approximate two to three-fold increased risk for adulthood anxiety (Pine, Cohen, Gurley, Brook, & Ma, 1998). Depression is the second

leading cause of disability adjusted life years (DALYs; i.e., productive years lost due to premature death or physical disability) starting at 15 years (Murray & Lopez, 1997; WHO, 2011) and is often an early symptom of other physical health problems (e.g., Henningsen, Zimmermann, & Sattel, 2003; Penninx, Leveille, Ferrucci, van Eijk, & Guralnik, 1999).

Additionally, a longitudinal study conducted by Reinherz et al., (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 physical health 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 physical health, the current dissertation will investigate the long-term consequences of peer victimization on physical health. In particular, this study will examine whether victimization influences diurnal patterns of cortisol (as a biological measure of stress) over-time.

Current Study

Recent research has highlighted some of the detriments of chronic peer victimization. However, very few studies have documented the long-term influence of victimization on physical health outcomes. No study to date has examined changes in biological functioning and health simultaneously. Research is needed to determine whether there are long lasting health consequences for those experiencing peer victimization. As such, this dissertation sought to understand the physical and psychological consequences of peer victimization over a two-year period.

This study will also examine if HPA activation (as assessed via cortisol) undergoes changes over several years and may ultimately serve as a mechanism for understanding long-term poor health effects. Specifically, cortisol was assessed in terms of CAR and overall daily cortisol levels. Health outcome measures included: (1) internalizing problems (i.e., depression, and symptoms associated with PTSD); (2) physical health problems; and (3) abdominal pain. Specifically, it was hypothesized that peer victimization would influence the neuroendocrine system which in turn, would negatively influence health outcomes.

The following aims were addressed as part of this dissertation.

Aim 1: To examine whether peer victims (T1) have poorer physical and psychological health problems at the second assessment (T2). Specifically, this dissertation assessed whether adolescents who are victimized at T1 reported more frequent health problems, more severe health problems, more frequent medical visits, and more abdominal pain at T2 than adolescents who are not recipients of chronic peer victimization. Secondly, I examined whether changes in victimization (from T1 to T2) lead to poorer health outcomes at the second assessment (T2). Initial levels of health and cortisol functioning were controlled for to examine possible changes in biological functioning and health over time.

Aim 2: To examine whether being peer victimized (T1) was related to abnormal basal cortisol levels and a flatter CAR (T2). Again, initial levels of cortisol were used to examine changes in biological functioning from T1 to T2. I expected that victimized (T1) adolescents would evidence altered diurnal patterns of cortisol at T2 in comparison to their non-victimized counterparts. In addition, I examined whether being victimized leads to continued changes over time.

Aim 3: To examine whether cortisol levels mediated the victimization-health link over time. That is, I expected chronic victimization to lead to abnormal diurnal patterns, which would in turn lead to poorer psychological and physical health outcomes at T2. Additionally, I examined whether depression mediates the relationship between victimization and health over time.

Aim 4: To examine gender differences in bullying outcomes. Specifically, I evaluated whether girls are more negatively impacted by bullying than are boys. That is, bullied girls were expected to show more internalizing problems and health problems than were bullied boys. Aim 4 will be examined as part of the analyses for Aim 1 through Aim 3.

Chapter 2

Methods

Participants

Participants were 9th to 12th grade adolescents (N1 = 125) who are took part in a previous study on peer social relationships and health and came back to complete identical questionnaires approximately two years later. I needed a sample of 95 participants for a predicted effect size of r = .25, α = .05, two tailed, and a power of .70 (Cohen, 1988). However, as I wanted to assess interactions in my data set, I attempted to collect more participants (N = 120; MacKinnon, 2008).

This study was not originally set up as a longitudinal study. That is, participants did not know they would be asked to come back to the lab. Even with these limitations, we had a reasonable return rate. A total of 58% of our sample returned during T2. (These rates of attrition are similar to several longitudinal studies regarding child development published within the year (e.g., 52%, Gershoff, Lansford, Sexton, Davis-Kean, & Sameroff, 2012, 70%, Sabol & Pianta, 2012, and 64%, Chan et al., 2013). Of the 42% from Time 1 who did not participate in Time 2 data collection, 12.6% moved out of the area or had non-working numbers, 3.7% requested to not be called again, 5.1% were scheduled but did not show up for their appointment, and 20.9% dropped out and/or did not return calls for a follow up study. Participants who previously participated in the research study on peer relationships and health were contacted from a phone list. Adolescents were paid a total of $60.00 and their parents a total of $40.00 for participating in a two part session of the current project.

[1] Although 125 participants continued in the study, five participants had time 1 victimization data missing from either the child self report or the parent report. As such, when data was combine for time 1 victimization, these participants were not part of further analyses where N = 120.

The ethnic composition of the original sample was fairly diverse with 56.2% Caucasian, 15.1% African American, 2.3% Asian, and 21.5% Hispanic or Latino, .9% American Indian or Native American, and .9% other. Participants in the original study were 5th to 8th grade adolescents (N = 216, Mage = 12.35 years, SD = 1.10; age range: 10 to 15 years). In the current study, ethnic composition was virtually identical (i.e., 57.6% Caucasian, 13.6% African American, 4.0% Asian, and 20.0% Hispanic or Latino, 1.6% American Indian or Native American, and 1.6% other). In T2 participants were approximately two years older, (Mage = 14.97 years, SD = 1.24; age range: 12 to 17 years), and were in 9th through 12th grade (N = 125).

Given that 42% did not return for this follow-up phase, analyses were run to examine possible differences between groups (i.e. those that dropped out versus those that remained in the study) for all major measures (see tables 1 – 5). Attrition analyses revealed that there were no differences on any of the measures between those the dropped out of the study in comparison to those that remained in the study. Specifically, there were no differences between groups for combined parent and child reports of physical victimization $F(2, 199)= .12$, $p = .73$, verbal victimization, $F(2, 199)= 1.80$, $p = .18$, indirect victimization, $F(2, 199)= 5.50$, $p = .20$, and combined total victimization, $F(2, 199)= 2.73$, $p = .10$. Additionally, there were no differences in combined reports of the psychological health measures of anxious depression, $F(2, 214)= .66$, $p = .13$, withdrawn depression, $F(2, 214)= .69$, $p = .41$, and symptoms associated with PTSD, $F(2, 214)= 1.25$, $p = .27$. Moreover, there were no differences between groups for combined parent and child reports of frequency of health $F(2, 214)= 2.13$, $p = .15$, severity of health, $F(2, 214)= 0.90$, $p = .34$, or visits to the doctor or nurse, $F(2, 214)= 3.60$, $p = .06$. Finally, there were no differences in ethnicity, χ^2 (2, 212 = 8.50, $p = .23$ or gender, χ^2 (2, 211) = 1.35, $p = .25$ between those that continued in the study and those that did not.

Assessment of Victimization

Direct and Indirect Aggression Scales – Victim Version (DIAS). The DIAS (Bjorkqvist, Lagerspetz, & Osterman, 1992) assessed how frequently one experiences 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. Additionally, a parent version of this scale was also used to gauge parental perceptions of peer abuse experienced by their child (e.g., “How often is your child hit by others?,” etc.).

Physical and Psychological Health Assessments

Achenbach – Child Behavior Checklist (CBCL) and Youth Self Report (YSR).

The CBCL and YSR (Achenbach & Rescorla, 2001) were used to assess the adolescent’s withdrawn depression (9 items), anxious depression (18 items), and symptoms associated with PTSD (4 items). Questions were asked on a Likert-type scale, from 0 = not true to 2 = very true. Sample questions include for anxious depressed, “ I am nervous or tense,” withdrawn depressed, “would rather be alone,” and PTSD symptoms, “ I have nightmares.” Parents completed the CBCL (Child Behavior Checklist) and the adolescent completed the YSR (Youth Self Report). Additional subscales that were not part of my focal hypotheses were collected but not examined as part of this dissertation.

Health Survey. The Health Survey, which was filled out by both the adolescent and their parent, assessed how frequently adolescent-participants experienced particular health problems. Fourteen questions assessed both frequency and severity of health problems were asked using a Likert-type scale ranging from 1 (not al all) to 4 (all the

time) for frequency and 1 (does not hurt at all) to 4 (unbearable pain) for severity. Additionally, this survey was used to assess how frequently participants visit their school nurse or doctor. See Appendix 1 for a copy of the survey.

Abdominal Pain Index. This scale consisted of five Likert-type questions used to assess abdominal pain over the past two weeks. This questionnaire was used to examine the frequency, length, and intensity of abdominal pain experienced by participants as reported by both participants and their parents and is available in Appendix 1.

Procedure

As children were involved, a two-step consent process took place. Parents gave consent for their child to participate, and children gave their assent to participate in a study concerning their relationships, school performance, and health in adolescents over time. During the re-contacting period, parents were told about additional measures that were not part of this dissertation (e.g., saliva and blood collection), as well as the surveys their child would be asked to fill out (reference Figure 2 for timeline of project).

The questionnaires that were used for the current study were filled out approximately two years ago and were completed again approximately 2.5 years later (M = 24.5 months, Min = 10 months, Max = 44 months). In phase one of the current study, adolescents and their parents completed self-report victimization measures (DIAS – Victim Version; Bjorkqvist et al., 1992), as well as measures on psychological (Achenbach) and physical health (health outcomes and abdominal pain index). Participants and their parents also completed several additional measures that were not part of this study (e.g., bullying and social support).

Adolescents and their parent were then taught methods of proper collection and storage of the adolescent's cortisol via saliva. Adolescents were instructed to collect four samples of their saliva over two non-sport school days. Samples were collected

immediately when the adolescent wakes, 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. After learning how to collect their saliva samples, adolescents were then taught how to record their completion via paper copies of surveys from Survey Monkey. Adolescents gave an additional sample of their saliva for DNA (not part of this dissertation). Adolescents and their parent returned to the laboratory for the second session within three to nine days. Participants brought back their cortisol samples during this phase, and both parents and adolescents completed several additional questionnaires about their child's personality as well as Achenbach-YSR and CBCL. After this, participants gave a small sample of their blood (not used as part of the current study) (reference Figure 3 for constructs of interest and their measures).

Overview of Data Analysis

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.

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 the Health and Chronic Illness Center 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.

Handling of Missing Data

Data contained some missing values. That is, there were few instances where participants failed to, or missed answering a question. In order to determine how problematic these missing values are, the pattern at which the values are missing should be closely inspected. Although only a few values were missing, the pattern of how the data is missing influences the means by which it is handled (Tabachnick & Fidell, 2007). To analyze more completely the pattern of missing data, a missing value analysis (MVA) in SPSS was run to evaluate the pattern of my missing data points. The Little MCAR tests were not significant, ($\chi 2$ = 2278.66, 55.43, .06 dfs = 2169, 225, 1 ps = 0.06, 1.00, .82, for health and DIAS, Achenbach, and cortisol respectively) thereby indicating that the missing data points were missing completely at random (MCAR; Little, 1988) (for T2 measures missing data was as follows: child-reported victimization, .01%; child physical health child, .02%; child-reported psychological health, .04%; parent-reported victimization, .02%; parent-reported physical health, .03%; parent-reported psychological

health, .07%, cortisol, 8.65%. As participants were paid prior to the return of their cortisol in some cases, an increased number of missing values for cortisol was present in comparison to the other measures. As missing data was evident, expectation maximization (EM) methods, which substitute estimated expectations of missing data for the missing data points to maximum likelihood estimation, was imputed. Once convergence was attained, the finalized data set (with the imputed values) was saved. This overall complete data set with no missing values was used for analyses.

Chapter 3

Results

Descriptive Statistics

The reliabilities and descriptive statistics for the major measures for T1 can be found in Tables 6-8 and for T2 in tables 12-14. Tables 9-11 show the inter-relationships among the victimization, psychological health, and physical health measures at T1 and in Tables 15-17 for T2. As can be seen in Table 9, the inter-correlations among the victimization measures at T1 were moderately large (rs = 0.31 – 0.78). Moreover, parent and child reports of victimization are highly related both at T1 (see Table 9). However, since I was not interested in differences between self- versus parent- report or types of victimization, I z-scored all victimization measures and averaged together to create a more reliable overall measure of peer victimization (Shakoor et al., 2011). These victimization measures were also highly related at T2 (rs = 0.40- 0.83) as can be seen in Tables 15. Again parent and child reports of victimization were highly correlated with one another at T2 (see Table 15). As such, I again z-scored all victimization measures and averaged them together to create a composite measure of victimization at T2. In addition, the correlations among raters (i.e., parent-rated and child-rated) were moderate at both T1 and T2 for both psychological and physical health measures. As such, scores on these scales were averaged to create combined measures of severity and frequency of health, visits to the nurse or doctor, anxious depression, withdrawn depression, and PTSD (see Tables 10-11 for T1; Tables 16-17 for T2).

Peer victimization was treated as a continuous variable in all focal analyses. Additionally, although most participants came back within a similar time frame, time between assessments was centered and used as a covariate to control for possible differences between assessments (M = 24.5 months, SD = 8.26 months). Moreover, I

assessed victimization groupings (see cluster analysis after Aim 1c) at T1 versus T2. At T1, there were a total of 21 victims and 99 non-victims. At T2, there were a total of 20 victims and 100 non-victims. Based on victimization scores at T1 versus T2, participants were coded into one of four groups: (1) those that were non-victims throughout (N = 91); (2) those that escaped victimization between assessments (N = 9); (3) those that became new victims from T1 to T2 assessments (N = 8); and (4) those that were persistent victims throughout the study (N = 12).

Changes in Psychological & Physical Health

Aim 1a: Was Victimization at T1 Related to Health Problems at T2?

I wanted to examine whether peer victims (T1) have poorer physical and psychological health problems at the second assessment (T2). Specifically, I examined whether adolescents who are victimized at T1 reported more frequent health problems, more severe health problems, more frequent visits to the doctor/ nurse, and more abdominal pain at T2 than adolescents who are not recipients of chronic peer victimization at T1. I also assessed whether the sex of the adolescent moderated this victimization health relationship (Aim 4). Specifically, I examined whether bullying more negatively impacted health for girls than for boys.

To test these hypotheses, regression analyses were run. Victimization (T1) was centered and treated as a continuous variable. Gender was recoded using unweighted effects codes (Aiken & West, 1991). Finally, the cross-product between victimization and gender was created. Victimization (T1), the unweighted effects codes for gender, their cross products, and the centered difference in time from assessment one to two was entered into the equation. Dependent measures for Aims 1a, 1b, and 1c included frequency and severity of health, visits to the doctor, anxious depression, withdrawn

depression, and symptoms of PTSD. All dependent variables used were averaged scores across raters as stated previously.

Physical Health Outcomes.

Victimization (T1) was related to more frequent (B = 2.68, t(119) = 4.82, p < .001, sr2 = 0.05) and severe (B = 1.54, t(119) = 3.09, p < .001, sr2 = 0.07) health problems, as well as significantly more trips or visit to the doctor or nurse (B = 0.34, t(119) = 3.94, p < .001, sr2 = 0.11) at T2. That is, participants who had higher victimization scores at T1 reported more physical health problems at the second assessment. Results also indicated that there were no significant interactions between victimization (T1) and gender for frequency of health (B = 0.44, t(119) = 0.79, p =.43, sr2 = 0.00) or visits to the doctor or nurse (B = 0.15, t(119) = 1.72, p =.09, sr2 = 0.01) at T2. There was a significant interaction between victimization (T1) and gender for severity of health at T2 (B = 1.06, t(119) = 2.12, p =.04, sr2 = 0.03. As anticipated, bullied girls (T1) reported more severe health problems at T2 compared to non-bullied girls, (B = 2.60, t(119) = 3.68, p <.001, sr2 = 0.10). There was no relationship between bullying (T1) and health severity (T2) for boys (B = 0.48, t(119) = 0.67, p =.50, sr2 = 0.00).

Psychological Health Outcomes.

Participants who were victimized (T1) also reported being more anxious depressed (B = 0.81, t(118) = 3.22, p = .002, sr2 = 0.08), withdrawn depressed (B = 0.60, t(118) = 3.70, p < .001, sr2 = 0.10), and having more PTSD symptoms (B = 1.30, t(118) = 5.16, p < .001, sr2 = 0.18) at T2 than participants who did not report being victimized at T1. Similar to physical health symptoms, bullied adolescents reported higher levels of psychological problems two years later than did their non-bullied peers. There were no significant interactions between victimization (T1) and gender for anxious depression (B = 0.28, t(119) = 1.08, p =.28, sr2 = 0.01), withdrawn depression, (B = 0.30, t(119) = 1.83, p

=.07, sr2 = 0.03, or symptoms of PTSD (B = 0.33, t(119) = 1.29, p =.20, sr2 = 0.01) at T2. Both boys' and girls' psychological health was equally affected by peer victimization contrary to Iyer et al., (under review).

Aim 1b: Was Victimization at T1 related to Health Problems at T2 (controlling for T1 health)?

Next, I examined whether victimization (T1) predicted health problems at T2 after controlling for T1 health problems. By controlling for health problems at T1, the outcome measure is actually assessing changes in health. Traditionally, change scores are obtained by calculating a simple gain from assessments. However, calculating simple gain scores in this way can lead to erroneous conclusions due to the influence of systematic random error measurement (Cronbach & Furby, 1970). In order to ensure a more accurate overall psychological or physical health change assessment, I regressed T1 health scores on to T2 health scores and saved the standardized residuals (Appelbaum & McCall, 1983) for frequency of health, severity of health, visits to the doctor or nurse, withdrawn depression, anxious depression, and symptoms of PTSD. Higher scores indicate increases in the above measures between assessments. Specifically, by assessing changes in health in this way, I am able to more rigorously test my hypothesis by controlling for T1 health. The model in aim 1b was identical to aim 1a with the exception of the DVs, which were now change scores (i.e., unstandardized residuals) for each outcome measure.

Health Problems

As anticipated, victimization (T1) was significantly related to changes in frequency (B = 0.34, t(115) = 4.43, p < .001, sr2 = 0.14) and severity (B = 0.40, t(115) = 5.17, p < .001, sr2 = 0.19) of health problems, as well as visits to the doctor or nurse (B = 0.21, t(114) = 2.44, p = .02, sr2 = 0.05). Children who were victimized at T1 reported

increasingly worse physical health outcomes over time. There were no significant interactions between victimization (T1) and gender for changes in frequency of health (B = -0.02, t(115) = -0.26, p =.80, sr2= 0.00), severity of health (B = -0.12, t(115) = -1.51, p = .13 sr2 = 0.02), or visits to the doctor or nurse (B = 0.02, t(114) = 0.02, p =.98, sr2 = 0.00). Both boys and girls reported declines in health when they were bullied.

Psychological Health Outcomes

Participants who were victimized (T1) reported significant increases in anxious depression (B = 0.19, t(118) = 2.25, p = .03, sr2 = 0.04), and symptoms of PTSD (B = 0.33, t(118) = 4.15, p < .001, sr2 = 0.12), but not more feelings of being withdrawn depression (B = 0.14, t(118) = 1.61, p = .11, sr2 = 0.02). As with physical health, children who were victimized at T1 reported increases in psychological problems over time. Again, there were no significant interactions between victimization (T1) and gender for changes in anxious depression (B = 0.28, t(119) = 1.08, p =.28, sr2= 0.00), withdrawn depression, (B = 0.12, t(118) = 1.41, p =.16, sr2 = 0.02), or symptoms of PTSD (B = 0.12, t(118) = 1.53, p =.13, sr2 = 0.02). Contrary to my predictions, both boys and girls reported similar increases in internalizing problems when peer victimized.

Aim 1c: Did Changes in Victimization from T1 to T2 Predict Health Outcomes at T2?

Victimization scores at T1 were significantly related to scores at T2 (see table 18). However, I wanted to examine whether changes in victimization (from T1 to T2) led to poorer health outcomes at the second assessment (T2). A change score for victimization was created for use in analyses. Just as with the change in health measures, I regressed victimization (T1) on to victimization (T2) to create a change score (i.e., unstandardized residuals) (Appelbaum & McCall, 1983). Higher numbers indicate increases in victimization. Centered change in victimization, the unweighted effects codes

for gender, their cross products, and the centered difference in time from assessment one to two were entered into the equation.

Change in victimization was not related to more frequent (B = 0.02, t(119) = 0.89, p = .37, sr2 = 0.01) or severe (B = -0.01, t(119) = -0.51, p = .61, sr2 = 0.00) health problems, or more visits to the doctor or nurse (B = 0.08, t(119) = 0.89, p = .37, sr2 = 0.01). Changes in victimization was also not related to being more anxious depressed (B = -0.00, t(118) = -0.06, p = .96, sr2 = 0.00), withdrawn depressed (B = 0.07, t(118) = 1.40, p = .16, sr2 = 0.02), or having increased symptoms of PTSD (B = 0.06, t(118) = 1.80, p = .07, sr2 = 0.03) at T2. Additionally, there were no significant interactions between gender and changes in victimization for frequency (B = 0.01, t(119) = 0.93, p =.36, sr2 = 0.01), or severity of health (B = 0.03, t(119) = 1.42, p = .16 sr2 = 0.02), visits to the doctor or nurse (B = 0.02, t(119) = 0.23, p =.82, sr2 = 0.02), anxious depression (B = 0.02, t(119) = 0.65, p =.52, sr2 = 0.00), withdrawn depression, (B = 0.63, t(118) = 1.28, p =.20, sr2 = 0.12, or symptoms of PTSD (B = 0.00, t(118) = 1.14, p =.99, sr2 = 0.00) at T2.

Aim 1C: Supplementary Analyses.

The lack of significant findings between difference scores in victimization at T1 and T2 is not surprising considering that the number of victims in T1 versus T2 was fairly similar suggesting that victimization between these two assessments was fairly stable. Victimization (T1) was highly correlated with reports of victimization (T2) (r = .60, see Table 27). However, it is possible that some victims escaped victimization while others became new victims. That is, there could be changes in victimization status between assessments. A variable centered approach, which treats victimization as a continuous variable, does not directly provide information on how status change may influence health outcomes over time. As such, an alternative way using a person-centered approach

would allow me to examine how changes in status influence health outcomes. Several other researchers argued the importance of a person-centered approach and classified their data based on distinctive groups (e.g., Nagin, 1999; Soberg & Olweus, 2003; Vaillancourt et al, 2008). Additionally, viewing data in this way can help us see if there are subgroups of victims that appear over time. As such, cluster analyses were conducted to explore victimization status at T1 and T2, as well as victimization status change between assessments.

To assign groups to victims and non-victims, I used a two-step classification process, which is thought to provide more valid and robust patterns (Steele & Aylward, 2007). To begin, an agglomerative hierarchical cluster analyses was conducted, which clusters individual scores based on proximity. This type of analyses begins by looking at each participant separately and over multiple iterations combing individuals into one large cluster. In this approach, examining the dendogram and agglomeration coefficients concludes the number of cluster groups. Specifically, the percentage of change from each step of clustering was noted. The distance between the cluster centers is indicated by a large jump in changes of agglomeration clusters. Ward's method is most recommend and as such, was chosen for analysis (Steele & Aylward, 2007). The appropriate number of clusters was determined by examination of the dendogram and agglomeration coefficients, which suggested a two-cluster solution for victimization scores at T12. Based on the hierarchal cluster analysis, there were approximately 18 victims in my sample and 102 non-victims. The separate parent and child reports for the standardized scale of physical, verbal, and indirect victimization from the DIAS scale was used for classification of victimization status.

[2] I also examined the three-factor solution for completeness. The fit of this solution was not acceptable based on the K-mean cluster results and McNemar's test.

I then used a k-cluster means analysis (with normalized Euclidean distance as the distance metric) to verify my original solution. In k-means clustering participants are classified by minimizing the SSwithin within each cluster (i.e. the distance to the cluster center). The exact cluster centers for each of the three types of victimization as were found in the hierarchical cluster analysis were used for the initial cluster centers for the k-means clustering. The results from the k-cluster solution converged in three iteration suggesting that a 2-cluster group membership suited the data the best. Using the k-cluster solution approximately 21 participants in the sample were victims and 99 were non-victims. A McNemar's test, which assessed the changes in participant's scores from each of the cluster methods, was significant at T1; χ^2 (2, 118) = 80.82, p = 0.03. However, there were only 6 cases that differed in group classification between the two types of cluster analysis. The k-cluster solution group assignments were used for the supplemental analysis as the percentage of victims that were found using this process of classification match more closely with previous studies we have run.

Identical cluster tests were run at T2. Analysis indicated via hierarchical and verified through k-cluster analysis that again a two-cluster solution was best for the T2 victimization data. Hierarchical cluster analysis indicated that the data at T2 contained 12 victims and 108 non-victims, and the k-cluster analysis found that there were 20 victims in the data and 100 non-victims. McNemar's test, was again significant at T2; χ^2 (2, 118 = 66.67, p = 0.008. However, similar to T1 data, there were only 8 cases out of 120 that were classified differently. As such, based on previous finding, the number of victims using the k-means cluster analysis was used in subsequent analyses. Additionally, the differences between victimization membership at T1 versus T2 was coded into one of four groups; those that were non victims throughout the study (72.8%), those that

became new victims at T2 (6.4%), those that escaped victimization at T2 (7.2%), and finally those that were continuous victims from T1 to T2 (9.6%).

Additional analyses were conducted to further assess victim status (persistent victims (N = 12), non-victims (N = 87), new victims (N = 8), escaped victims (N = 9)) influenced psychological and physical health over time. Group classification names were used from Smith et al. (2004); similar types of classifications can also be noted in Dempsey, Fireman, and Wang, (2006) and Kochenderfer-Ladd & Wardrop (2001). It should be noted that these results are preliminary and should be interpreted with caution due to the small N in my victim sub-groups.

To evaluate different health trajectories based on victimization group classification, a repeated-measures univariate GLM analysis was run. The within-subject factors were labeled as change in frequency, severity, visits to the doctor or nurse, withdrawn depression, anxious depression, and symptoms of PTSD respectively. Victimization group classification was entered as the between subjects factor in all supplemental analyses. Additionally, post-hoc pair-wise comparison using a Bonferroni correction on estimated means was conducted to probe all significant main effects. Finally, Profile plots were provided to illustrate the changes in health measures based on victimization statue.

Physical health problems.

Results indicated that there was a main effect for frequency of health problems , $F(3,116) = 11.91$, $p < .001$, $\eta p2 = .24$. Non-victim and new victim groups differed from each other; as did non-victim and persistent victim groups in frequency of health problems (see Figure 4 for frequency of health between T1 and T2 for each of the groups; see Table 19 for means and standard errors for all groups).

Results also indicated a main effect for severity of health problems, $F(3,116) = 8.00$, $p < .001$, $\eta p2 = .17$. There was a significant difference between non-victim and new victim groups as well as between non-victim and persistent victim groups on severity of health problems (see Figure 5 for severity of health between T1 and T2 for each of the groups; see Table 19 for means and standard errors for all groups).

Finally, there was a main effect for visits to the doctor or nurse, $F(3,116) = 7.48$, $p < .001$, $\eta p2 = .16$. Non-victim and persistent victim groups differed in visits to the doctor or nurse (see Figure 6 for visits to the doctor or nurse between T1 and T2 for each of the groups; see Table 19 for means and standard errors for all groups).

Psychological health problems.

There was a main effect for anxious depression , $F(3,115) = 7.33$, $p < .001$, $\eta p2 = .16$. Non-victim and persistent victims again differed from one another, as did non-victim and new victim groups in anxious depression (see Figure 7 for anxious depression between T1 and T2 for each of the groups; see Table 19 for means and standard errors for all groups).

Additionally, there was a main effect for withdrawn depression, $F(3,115) = 4.32$, $p = .006$, $\eta p2 = .10$, . Again, non-victims and persistent victims differed in changes for withdrawn depression (see Figure 8 for withdrawn depression between T1 and T2 for each of the groups; see Table 19 for means and standard errors for all groups).

Finally, there was a main effect for symptoms of PTSD, $F(3,115) = 11.09$, $p < .001$, $\eta p2 = .22$. Non-victim and persistent victim as well as non-victim versus new victims, and escaped victims versus persistent victims differed on symptoms associated with PTSD. Specifically, participants who were persistent victims reported significantly more symptoms associated with PTSD then non-victims, additionally, new victims reported significantly more symptoms associated with PTSD then non-victims, and finally,

persistent victims are reporting significantly more symptoms associated with PTSD than escaped victims (see Figure 9 for PTSD symptoms between T1 and T2 for each of the groups; see Table 19 for means and standard errors for all groups).

Overall, sub-group classifications between assessments followed several interesting patters (see Figures 4-9). As would be expected, persistent victims tended to have the worst outcomes on all the major measures between assessments. Also as expected, non-victims had the best outcomes on all the major measures over time. Interesting results were found between the new victims and the escaped victims. Those that were escaped victims tended to show improvements in their psychological and physical health. Although, these improvements very rarely caught up to those that were non-victims throughout the study. (Note that escaped victim scores were not significantly different than non-victims but were also not significantly different than either persistent victims or new victims and fell between these two groups). Most interesting perhaps, may be that the new victims had increases in many of the major measures between assessments. However, this group in some cases reported having worse outcomes compared to the persistent victims. Even though these results should be interpreted with caution, these preliminary findings suggest interesting trends for each sub-group that warrant further investigation.

Cortisol Patterns

Aim 2: Does Peer Victimization (T1) influence Cortisol Patterns (T2)?

Cortisol was assessed as: (1) the average cortisol level and (2) the cortisol awakening response (CAR) or the difference between wakening and wake (+ 30 minutes). Time points were first averaged across days and then calculated for each of the assessments above. Additionally, the data were run as mg per deciliter. Before calculations occurred, the data was first converted to mg per mililiter by multiplying each

cortisol sample by 100 (to allow for natural log transformations). As data points were skewed, samples were also log-transformed3 in order to normalize assessments (See Table 20 for correlations between T1 and T2 time measures of CARg as well as AUCg).

When looking at the relationship between recurrent assessments of endocrine functioning or any other physiological variable, typically the changes are calculated using an area under the curve (AUC). However, this type of calculation fails to take into account the differences in collection methods between labs (i.e. there is a lack of standardization across those measuring it). To accommodate differences in collection methods, a calculation looking at the changes among each sample for every participant, is an improved means of assessing changes. Based on this knowledge, cortisol in the present study was calculated as area under the curve in respect to ground (AUCg). (See Pruessner, Kirschbam, Meinlschmid, & Hellhammer, 2003 for a detailed description).

To create the measure of cortisol awakening response in respect to ground, CARg, was calculated using the first two time points of AUCg. As CARI is a measure of the dynamic cortisol response rather than a total cortisol secretion, it was left out of analyses (Hinkelmann et al., 2013).

As in the previous model, victimization was centered and treated as a continuous variable. Gender unweighted effects codes were also created (Aiken & West, 1991). In addition, the interaction between sex of the participant and victimization was entered into the model. Finally, time between assessments and the T1 cortisol measurement were centered and entered as control variables. Following significant interactions between victimization and gender, simple effects analyses for victimization were conducted using the appropriate dummy codes for sex (see Aiken and West, 1991, pp. 130-133, for a review).

[3] Analyses were also run using non-transformed data. Results revealed virtually identical results.

As my data for both victimization and cortisol were skewed, bootstrapping was performed on the proposed regression analyses. Bootstrapping is a resampling strategy, which creates regression weights through many replications or replacements from the original sample. In this case, all bootstrapping results in this document were based on 5000 bootstrap samples (Tibachnik & Fidell, 2007). Moreover, bootstrapping can be used when the original sampling distribution is not normal. For example, much as the case in this dissertation, when estimation occurs using a regression coefficient produced by an ordinary least square, which evidences a skewed residual. Additionally, bootstrapping may be used when the distribution sampled does not have any known analytic solution (i.e. the differences reported between medians in a sample). When this occurs, instead of a more traditional use of confidence intervals, the use of a bias-corrected percentile can be employed as was done in this case (Mooney, Duval, & Duvall, 1993).

Furthermore, as my sample was not homogenous, a stratified bootstrapping method for victimization groups, gender, and ethnicity was implemented. Victim groups involved those that were non-victims versus those that were victims at a minimum of one time point. Additionally, ethnicity was recoded to include only 4 groups; White/Caucasian, Black/African American, Hispanic/Latino, and other in all analyses. This was done so that victim and ethnic groups would be large enough to do the stratified bootstrapping correctly. In this paper, for the use of analyses, samples were independently drawn for bootstrapping based on the strata they were apart of (Biecke & Freedman, 1984).

Does Victimization (T1) predict overall daily cortisol levels as assessed by AUCg at T2? The overall model for AUCg was significant, $F(4, 97) = 6.89$, $p < .001$. Moreover, there is a significant main effect for victimization, $B = -69.08$, $t(101) = -1.53$, $p = .02$, bootstrapping; BCa LL 95% = -127.36.10, BCa UL 95% = -17.26. Results indicate that

victimized children are producing lower daily cortisol at T2. These results have been found in other studies as well (e.g. Knack et al., 2011, Vaillancourt et al., 2008). Additionally, there was a significant T1 victimization x gender effect, B = 61.35, t(101) = 1.36, p = .04, bootstrapping; BCa LL 95% = 4.73, BCa UL 95% = 119.63. Specifically, victimized boys are producing less daily cortisol at T2, B = -130.70, t(100) = -2.09, p = .002, bootstrapping; BCa LL 95% = -212.89, BCa UL 95% = -55.38. For girls there is no relationship between being bullied and daily cortisol levels at T2, B = -11.33, t(100) = 0.17, p = .79, bootstrapping; BCa LL 95% = -93.38, BCa UL 95% = 64.30 (see Table 21 for regression summary statistics for all variables).

When AUCg at T1 was used as a control, the overall model for AUCg was still significant F(5, 95) = 6.67, p < .001. Specifically, there was a marginal main effect for victimization, B = -59.23, t(101) = -1.33, p = .06, bootstrapping; BCa LL 95% = -120.63, BCa UL 95% = -2.45. Victimization was related to decreases in cortisol over the two year time period. Moreover, there was still a significant T1 victimization x gender effect, B = 69.11, t(101) = 1.56, p = .03, bootstrapping; BCa LL 95% = 8.09, BCa UL 95% = 127.87. For boys, bullying was associated with lncreases in daily cortisol from T1 to T2, B = -126.07, t(101) = -2.07, p = .01, bootstrapping; BCa LL 95% = -212.36, BCa UL 95% = -43.45. For girls, there was no relationship between being bullied and daily cortisol level, B = 9.63, t(101) = 0.15, p = .87, bootstrapping; BCa LL 95% = -86.97, BCa UL 95% = 100.27 (see Table 21 for regression summary statistics for all variables).

Does Victimization (T1) predict a flatter CAR? The multiple regression model with all five predictors produced a non-significant overall effect for the cortisol awakening response in regards to ground, CARg, when T1 CARg was used as a control, F(6,96) = 0.61, p = .69, R2 = .03 There was however a significant victimization main effect for the cortisol awakening response in regards to ground, CARg, B = -14.29, t(101) = -0.88, p =

.04, bootstrapping; BCa LL 95% = -23.93, BCa UL 95% = -5.29. Moreover, there were no significant victimization (T1) x gender effect, for the CARg, B = -0.07, t(101) = -0.73, p = .37, bootstrapping; BCa LL 95% = -0.22, BCa UL 95% = 0.08 The significant differences noted in the cortisol awakening response in respect to ground, for children who are victimized, follows much previous literature that documents how victimized children produce a blunted cortisol awakening response. Additionally, the difference in overall cortisol over time suggests changes in biological function in the long-term as a result of experiencing peer victimization.

Mediation Model

Aim 3: Do cortisol levels mediated the victimization-health link over time.

Before assessing if endocrine functioning mediates or partially mediates the link between peer victimization and health measures as presented in Figure 1, the relationship between cortisol and both psychological and physical health outcomes was explored (see Table 23). AUCg and CARg was examined in the final mediation analyses since only those measures were related to victimization in the previous analyses, correlation analyses examined these cortisol measures with all heath constructs at T2. As differences between the boys and girls were noticed in several initial analyses, data was split by gender and then correlated with all the major health measures. As can be seen in Table 23, there were significant relationships between several of the health measures and the assessments of cortisol. Specifically, boys' health constructs of severity and frequency of health, visits to the doctor, anxious depression, withdrawn depression, and symptoms of PTSD at T2 assessments were significantly correlated with AUCg. Moreover, CARg was significantly correlated with the simple difference in measures of frequency of health between T2 assessments. These same relationships are

not necessarily seen with girls (see Table 23). Once again, these results indicate that different relationships between both psychological and physical health, and biological functioning are present between boys and girls. However, it is possible that cortisol could mediate the relationship between victimization and health outcomes and that gender could moderate the association between victimization and cortisol, and between cortisol and health outcomes (both psychological and physical). As such, moderated mediation analyses were conducted following the process model outlined by Hayes (2013).

The conditional process analysis put forth by Hayes (2013), is most often used when there is a desire to explain the "conditional nature of the mechanism or mechanisms by which a variable transmits its effect on another (pg. 10)." Specifically, mediation analysis can be used to assess both the direct and indirect relationship of an independent variable (X) on an outcome variable (Y) through a third variable (M). Conversely, moderation analyses demonstrate how the effect of the independent variable (X) on the dependent variable (Y) is contingent upon some other variable (W). The use of the conditional process analysis is beneficial when the research goal is to assess both of these methods simultaneously by paying specific attention to the "estimations and interpretation of the conditional nature (moderation component) of the indirect and/or direct effects (mediation component) of X on Y in a causal system (pg. 10)."

The first model run, model 1 as seen in Figure 10, was run with victimization at T1 (X) predicating health at T2 (Y) looking at the mediating process of AUCg T2 (M). Additionally, gender (W) was assessed as a moderator in the above relationship. Specifically, gender (W) was evaluated as a moderator between victimization T1 (X) and AUCg (M) and also as a moderator between AUCg (M) and health outcomes (T2) (Y). Results indicated that AUCg (M) did mediate the relationship between victimization T1 (X), and all of the psychological health outcomes at T2 (Y) for boys (W). This relationship

was not seen for physical health outcomes at T2 for boys or overall for girls (W) (see Table 24 for details). Analyses, as used in model 1, were run again separately for boys and girls given the differences noted in the measures of cortisol (see Figure 11). Results indicated that when the files were run separately, AUCg (M) mediated the relationship between victimization at T1 (X) and health at T2 (Y) for frequency of health, severity of health, withdrawn depression, anxious depression, and symptoms of PTSD for boys only. No mediating relationship was noted for girls (see Table 25).

Next, CARg replaced AUCg in model 1. Specifically, the mediating process of CARg (W) between the relationship of victimization T1 (X) and psychological and physical health (Y) was explored. Again, gender (W) was evaluated as a moderator between victimization T1 (X) and CARg (M) and also as a moderator between CARg (M) and health outcomes (T2) (Y). Results indicated that CARg (M) did not mediate the relationship between victimization T1 (X), and all of the psychological health outcomes at T2 (Y) for boys or girls (W) (see Table 26 for details). Analyses, as used in model 1, were run again separately for boys and girls give the differences noted in the measures of cortisol (see Figure 11). Results indicated that when the files were run separately, CARg (M) did not mediate the relationship between victimization at T1 (X) and health at T2 (Y) for either boys or girls (see Table 27).

Observing AUCg4 as a mediator in the relationship between victimization and psychological health outcomes led to an additional mediation model (model 2, see Figure 12) assessing if bullying indirectly influences cortisol via depression or symptoms associated with PTSD. Indeed, some researchers argue that psychological health

[4] As significant mediating or moderating relationships were not initially found with any of the constructs and CAR_g, this model was dropped from further analyses.

outcomes such as depression may in fact be a precursor to physical health problems (Henningsen et al., 2003; Penninx et al.,, 1999).

In the subsequent analysis (see Figure 12) the psychological health outcomes of withdrawn depression, anxious depression, and symptoms of PTSD (M) (both at T1 and T2) were assessed as mediators in the relationship between peer victimization at T1 (X) and AUCg cortisol at T2 (Y). Moreover, gender (W) was again entered into model 2 to assess moderation. Results indicated that gender did moderate the relationship between anxious depression T1 ($B = 39.96$, $t(103) = 2.79$, $p = .01$), withdrawn depression T1 ($B = 52.94$, $t(103) = 2.29$, $p = .02$), symptoms of PTSD T1 ($B = 30.59$, $t(103) = 2.42$, $p = .02$) , and AUCg at T2 respectively. Identical significant moderation by gender was found between anxious depression T2 ($B = 51.34$, $t(103) = 3.99$, $p < .001$), withdrawn depression T2 ($B = 60.02$, $t(103) = 2.18$, $p = .03$), symptoms of PTSD T2 ($B = 41.50$, $t(103) = 2.81$, $p = .01$), and AUCg at T2 as well. These results suggest that gender moderates the positive increase in the relationship between psychological health and AUCg cortisol.

Again model 2 (see Figure 13), was assessed separately for boys and girls. The psychological health outcomes of withdrawn depression, anxious depression, and symptoms of PTSD (M) (both at T1 and T2) were assessed as mediators in the relationship between peer victimization at T1 (X) and AUCg cortisol (Y) at T2 for boys and girls separately. Results indicated the psychological health outcome of PTSD (M) at both (T1 and T2) mediated the relationship between T1 victimization (X) and T2 AUCg (Y) for both boys. No other significant mediating relationships were noted (see Table 28).

To further assess the mediating role of psychological health, specifically, frequency of health, model 3 was run (see Figure 14). The psychological health outcomes of withdrawn depression (M), anxious depression (M), and symptoms of PTSD

(M) (both at T1 and T2) were assessed as mediators in the relationship between peer victimization at T1 (X) and frequency of health T2 (Y). Results indicated that gender did moderate the relationship between peer victimization (T1) (X) and withdrawn depression T1 (M) (B = 0.43, t(120) = 2.43, p = .02) and symptoms of PTSD T1 (M) (B = 0.59, t(120) = 2.32, p = .02). A marginally significant moderation by gender (W) was found between peer victimization (T1) (X) and anxious depression T1 (M) (B = 0.48, t(120) = 1.84, p = .07).

Model 3 was also run to look at T2 measures of frequency of health (M). A significant moderation by gender (W) was found between peer victimization (T1) (X) and withdrawn depression T2 (M) (B = 0.32, t(119) = 2.00, p = .05). There was no significant interaction between peer victimization (T1) (X) and PTSD T2 (M) (B = 0.34, t(119) = 1.37, p = .17), or anxious depression T2 (M) (B = 0.30, t(119) = 1.17, p = .24).

Model 3 was adapted for the outcome variable of severity of health (see Figure 14). Specifically the model was run to assess the mediating role of withdrawn depression (M), anxious depression (M), and symptoms of PTSD (M) (both at T1 and T2) in the relationship between peer victimization at T1 (X) and severity of health T2 (Y). Results indicated that gender did moderate the relationship between peer victimization (T1) (X) and withdrawn depression T1 (M) (B = 0.43, t(120) = 2.43, p = .02) and symptoms of PTSD T1 (M) (B = 0.59, t(120) = 2.32, p = .02). A marginally significant moderation by gender (W) was found between peer victimization (T1) (X) and anxious depression T1 (Y) (B = 0.48, t(120) = 1.84, p = .07).

The adapted model 3 (see Figure 14) was also run to look at T2 measures of severity of health (M). Moderation by gender was found between peer victimization (T1) (X) and withdrawn depression T2 (M) (B = 0.58, t(119) = 2.32, p = .02), symptoms of PTSD T2 (M) (B = 0.43, t(119) = 2.43, p = .02). There was no significant moderation by

gender (W) found between peer victimization (T1) (X) and anxious depression T2 (M) (B = 0.30, t(119) = 1.17, p = .24). These results suggest that gender in some cases does moderate the relationship between psychological health and severity of health.

Additionally model 3 for frequency of health (see Figure 15) was used to analyze the psychological health outcomes of withdrawn depression (M), anxious depression (M), and symptoms of PTSD (M) (both at T1 and T2) as mediators in the relationship between peer victimization at T1 (X) and frequency of health T2 (Y) for boys and girls separately. Results indicated the psychological health outcome of PTSD T2 (M) and anxious depression T2 (M) did mediate the relationship between T1 victimization (X) and T2 frequency of health (Y) for boys. Withdrawn depression (M), anxious depression (M), and symptoms of PTSD (M) (both at T1 and T2) significantly mediated the relationship between peer victimization T1 (X) and frequency of health T2 (Y) for girls (see Table 29).

The adapted model 3 for severity of health (see Figure 15), was used to analyze the psychological health outcomes of withdrawn depression (M), anxious depression (M), and symptoms of PTSD (M) (both at T1 and T2) were assessed as mediators in the relationship between peer victimization at T1 (X) and severity of health T2 (Y) for boys and girls separately. Specifically, results indicated that psychological health outcome of PTSD T2 (M) did mediate the relationship between T1 victimization (X) and T2 severity of health (M) for boys. Withdrawn depression (M), anxious depression (M), and symptoms of PTSD (M) (both at T1 and T2) significantly mediated the relationship between peer victimization T1 (X) and severity of health T2 (M) for girls (See Table 30). Taken together, these results indicate that psychological health in many cases is a precursor to biological and physical health outcomes specifically for girls.

Chapter 4

Discussion

The current dissertation was an initial step in beginning to unfold and understand the long term psychological and physical health outcomes experienced by chronic victims. First, I examined whether those that were classified as peer victims at the first assessment had poorer health consequences at the second assessment. Second, I examined if victimization at T1 was related to psychological and physical health problems at T2 while controlling for health at T1. Third, I looked at whether changes in victimization from T1 to T2 were related to poorer health outcomes at T2. Fourth, I examine whether being a peer victimized (T1) was related to abnormal basal cortisol levels, a flatter CAR, and different diurnal patterns of cortisol levels (T2). Finally, I examine whether cortisol levels mediated the victimization-health link over time.

The link between peer victimization and health consequences at the second assessment. Current rates of peer victimization in the United States vary from 10-30% at school (Nansel, et al., 2001). In the current study, it was found that 18% of adolescents (n = 21) were victims at T1 and 17% of adolescents (n = 20) were victims in T2.

These percentages match those found in many current studies on victimization (Grills & Ollendick, 2002; Haynie et al., 2001; Nasel et al., 2001). More importantly, this number corresponds with current research conducted on peer victimization around the world. Rigby (2000) conducted a study to look at bullying in Australian schools. His sample consisted of 38,000 school children between the ages of 7 to 17 years. He found that nearly 16% of his sample reported experiencing chronic peer victimization. A study looking at the prevalence of school bullying in Korea reported that approximately 14% of their sample were victims of chronic peer abuse, 17% were perpetrators of that abuse, and an additional 9% were both victims and perpetrators of bullying (Kim Y, 2004).

Given that approximately one out of every five to six 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). Research however has yet to look at these consequences over time. Specifically, this dissertation attempted to fill in this gap by looking at the long-term health consequences experienced by chronic peer victims.

I examined whether peer victimized adolescents at T1 had poorer physical and psychological health problems at T2. I found that adolescents who were peer victimized in early adolescence (T1), did indeed report having more frequent and severe health problems, as well as increased visits to the doctor or nurse approximately 2.5 years later (i.e., in middle to late adolescence). Additionally, adolescents who were victimized in early adolescence also reported being more anxious depressed as well as withdrawn depressed as they moved into mid- to late adolescence. Moreover, these adolescents also reported having more symptoms associated with PTSD at T2 than adolescents who did not report being victimized at T1. Just as with physical health, children who were peer victimized reported higher levels of psychological problems two years later. These findings follow closely with the notion that during rapid periods of maturation, stressful life experiences can lead to long-term health consequences. As literature has reported, the rapid maturation in biological, cognitive, and social functioning is resembled only by those changes seen in infancy (Larson & Richards, 1994). The major changes in pubertal development and to the endocrine and immune systems during adolescence can leave these systems vulnerable when chronic or persistent stress is experienced. The additional importance placed on friendships that surface during adolescence continues to

reiterate the importance of experiencing positive social relationships, and the long-lasting consequences faced when those positive social relationships are not met (Hodges, et al., 1999).

The negative consequences experienced by peer victims are not something new. Research has found an association between peer victimization and physical health problems (Greco et al., 2007). Moreover, a study by Knack et al. 2012 found that even after controlling for personality differences in a group of college students, peer victimization predicted poorer health outcomes at both an initial assessment and over time. Rigby & Slee (1999) found those that experienced chronic peer victimization reported both poorer mental and physical health three years later in comparison to adolescents who had not experienced chronic peer victimization. Peer victims have also been found to exhibit increases in internalizing behaviors (i.e. loneliness, anxiety and depression) than those children who were not victimized by their peers (Callaghan & Joseph, 1995; Hodges & Perry, 1996; Slee, 1994; Swearer et al.,2001). The results from this study continue to suggest that the consequences of peer victimization are often persistent and not just something that will be “gotten over. “

Additionally, I examined if victimization at T1 was related to health problems at T2 while controlling for T1 health. Specifically, I examined the change scores between T1 and T2 scores for all psychological and physical health outcomes. Victimization at T1 was related to changes in significantly more frequency and severe health problems. Additionally there was a significant positive increase in visits to the doctor or nurse after experiencing victimization at T1. Participants also reported feeling increased changes in anxious depression and symptoms of PTSD. These results suggest that the effects of peer victimization are not only long lasting, but continue to get worse.

Moreover, the findings of this dissertation seem to indicate that bullying is rather persistent over time. Specifically, within my analyses, I did not find that changes in victimization from T1 to T2 predicted poorer health outcomes at T2. My results indicate that victimization is indeed a fairly stable trait. Other studies have found similar indications (Camodeca, Goossens, Terwogt, & Schuengel, 2002; Pelegrini & Bartini, 2000). However, victimization status seems to influence both psychological and physical health. The supplemental analysis that was conducted to examine victimization status between T1 and T2 assessments revealed some very interesting findings. Particularly, my analyses indicated that persistent victims have the worst psychological and physical health outcomes on all the major assessments over time. More promising maybe the fact that escaped victims may recover to a certain extent over time. This however is not seen on all outcomes. Further research still needs to be conducted on the victimization trajectories. Nagin (1999) urges a study between the estimation of behaviors in childhood to its links to long-term well-being in adulthood. Specifically, he states that creating and plotting trajectories would create a forum to observe the link between developmental patterns and frequently linked behaviors. By using trajectory assessments with victimization, we can begin to anticipate long-term consequences for each victim group and begin to create and learn about group characteristics.

Indeed, very few studies have actually looked at the trajectory of being bullied over time. Menesini, Modena, and Tani (2009) looked at the groups of bullies, victims, bully/victims, and uninvolved students. The authors looked at changes in groups between assessments and coded participants into 8 overall groups. They found that stable victims and stable bully/victims tended to report overall a higher feeling of anxiety and withdrawal then did those in the other groups. Although not identical, this study suggests that stable victims are indeed suffering the worst consequences when experiencing victimization

over time. Similarly, a study by Boivin, Petitclerc, Feng, and Barker (2010) found that over time, victimization was found to be less associated with aggression and progressively more associated with withdrawal. The mentioned studies, much as was found in this dissertation, indicates that the psychological health associated with being a peer victim is often a very frequent detriment of victimization. Even though these results should be interpreted with caution, the preliminary findings found in this dissertation suggests interesting trends for each sub-group that warrant further investigation.

Is being a peer victim related to endocrine functioning over time? In order to investigate the long-term consequences on children's biological system, cortisol was assessed as: (1) the average cortisol level (AUCg) and the cortisol awakening response (CAR) or the difference between wakening and wake (+ 30 minutes). Cortisol was assessed in this study, as it is the end product of the endocrine system. As previously stated, cortisol is associated with both regulatory roles (e.g. metabolism) as well as with stress reactions (Lovallo & Thomas, 2000). Overall production of cortisol is deemed essential to the management of daily stressors; however, over production of cortisol is related to several adverse consequences. Specifically, over production of cortisol is related to disturbed learning and memory (McEwen, 2000) and diminished immune functioning (Miller et al., 2002).

I found that being victimized at T1 influenced cortisol patterns at T2. Specifically, I found that being victimized at the first assessment predicted differences in overall daily cortisol. Additionally, I found that boys were producing less daily cortisol. There were no differences found for girls. 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. My results however are suggesting that the diurnal patterns of cortisol are

changing over time as a result of being the recipient of chronic peer abuse. I also explored the role of victimization on CARg also known as the cortisol awakening response. This time period is calculated as the increase in cortisol that occurs between the initial sample of cortisol and roughly 30 minutes after waking. Typically cortisol peaks about 30 minutes after waking. This peak has been termed the "cortisol mobilization response" or "cortisol awakening response." Typically this assessment is thought to serve as a preparatory response for the day (Tops et al., 2008). Research has indicated that individuals under severe stress actually have a blunted cortisol awakening response (Wessa et al., 2006; Fries et al., 2009). Moreover, several researchers have noted blunted cortisol awakening responses for bullied adolescents (Knack et al., 2011; Vaillencourt et al., 2008). Based on the importance of the cortisol awakening response, I assessed if victimization at T1 predicted a flatter CAR at T2. I found that victimized children at T1 did produce a blunted cortisol awakening response at T2. That is, bullied children are still producing a blunted CAR approximately 2.5 years later. Knack et al. (2011) found that being victimized produced changes in the hypothalamic-pituitary-adrenal (HPA) axis, which in turn predicted negative health outcomes. The current results provide compelling evidence that the changes in endocrine functioning are not only persistent, but are affecting the HPA axis long term. Taken together, these results again suggest that being peer victimized during a critical period of development is leaving lasting consequences on one's biological system.

Does cortisol mediate the link between victimization and health long term? Overall assessments of psychological and physical health were significantly correlated with measures of AUCg particularly for boys versus girls. Specifically, boys' health measures of severity and frequency of health, visits to the doctor, anxious depression, withdrawn depression, and symptoms of PTSD at both T1 and T2 assessments were

significantly correlated with AUCg and CARg. These same relationships were not noted with girls.

Moderated mediation analyses were conducted following the process model outlined by Hayes (2013) to assess the relationships of cortisol, victimization, health and gender. The model in Figure 10 was used to determine if gender moderated the relationship between peer victimization and biological functioning, as well as the relationship between biological functioning and health outcomes. Results indicated that there were no moderators by gender in Figure 10. However, AUCg (T2) did mediate the relationship between victimization (T1) and measures of psychological health at the second assessment for boys. There was no mediating relationship noted for girls. Additionally, CARg (T2) did not mediate the relationship between victimization (T1) and measures of psychological health at the second assessment for boys or girls.

As depression is often an early symptom of physical health problems, (Henningsen et al., 2003; Penninx et al., 1999) an additional model, Figure 12, was used to determine if psychological health mediated the relationship between victimization at the first assessment and AUCg at the second assessment. Results indicate that gender did moderate the relationship between all the psychological health (both T1 and T2) and AUCg (T2). Additionally, when files were run separately by gender, mediation was found for symptoms associated with PTSD for boys. This relationship was not seen for girls.

To further assess the mediating role of psychological health, an additional model was run looking at the outcome measures of frequency of health and severity of health respectively. For frequency of health, results indicated that gender did moderate the relationship between peer victimization and withdrawn depression and symptoms of PTSD at T1. At T2 measures of psychological health, a significant moderation by gender was found between peer victimization and withdrawn depression. For severity of health,

results indicated that there was moderation by gender found between peer victimization and withdrawn depression and symptoms of PTSD (T2). There was no significant moderation by gender found between peer victimization (T1) and anxious depression (T2). These results suggest that gender in some cases does moderate the relationship between psychological health and severity of health. More specifically, these results indicate as found by previously literature that poor psychological health behaviors are indeed contributing to poorer physical health.

Future Directions. This was an initial attempt in beginning to unfold and understand what the long term psychological and physical health outcomes experienced by chronic victims are. Specifically with this study we can begin to unfold and examine why the consequences of peer victimization maybe more long lasting for some children over others. Moreover, a strength of this dissertation was examining changes in biological functioning that are occurring during a period of rapid maturation both physically and emotionally. This dissertation was able to replicate many of the previous findings that peer victimization does relate to poorer psychological and physical health (Crick & Grotpeter, 1995; Crick & Bigbee, 1998; Crick et al., 1999; Knack et al., 2011)

Although there are many interesting and important findings, there should still be caution exercised when generalizing the results of the above study. First, the number of peer victims in this study was rather small. However, even with the small sample of victimization found in this study (18% in study one and 17% in study two), I find the relationship between peer victimization and psychological and physical health over time. Finding these relationships despite a small sample size, demonstrates the robust relationship between peer victimization and poor health outcomes over time.

Second, I found that cortisol was a mediator in the relationship between peer victimization and psychological and physical health. Specifically, due to the small

correlations between cortisol and all of health outcomes and the large correlations noted between psychological and physical health, there might be some overlap in method variance, which may be why some of these relationships were not significant (Spector 1987; Spector 2006).

Third, I have clearly shown a specific model that should be used to assess long-term peer victimization and health outcomes. This model is based on previous research that has suggested that victimization leads to greater health problems over time and not vice versa (e.g., Egan & Perry, 1998; Fekkes et al., 2006; Knack et al., 2012). The longitudinal nature of this study supports the suggested model, however, alternative models should also be assessed for completeness.

Finally, the mechanisms that bridges peer victimization and poorer psychological and physical health needs to be further examined as well as the changes and improvements in these detriments experienced by some versus others. As a next step, research should also examine how peer victimization affects health into the time period of emerging adulthood and beyond. Based on the patterns we have seen in this dissertation, it is important to also assess victimization trajectories during other periods of critical development. Research has yet to assess the victimization group trajectories in physical health and biological functioning over time.

This dissertation advances the field of psychology by demonstrating that the negative consequence of peer victimization may indeed be long lasting. 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. The current findings found in this dissertation really speak to the vulnerability of the biological and mental health systems experienced by peer victims during a time of great cognitive and biological development. The rapid maturation coupled with the extreme stress experienced by poor peer relationships clearly lead to

long lasting detriments in psychological and physical health outcomes. Most importantly, this dissertation provides compelling research to support evidence of the adverse effects that peer victim's experience. The results continue to shed light on the notion that being bullied is not a normal part of life and is something that individuals will not reconcile with time. In complete contrast, this dissertation not only finds that physical and psychological health for persistent victims do not get better with time, but that for these individuals, being bullied leaves very clear and noticeable mental and physical scars.

Appendix A

List of Tables

Table 1 Descriptive Statistics and Means for Standardized Self & Parent Reports of Victimization at Time 1 Between Participants that Remained in the Study and those that Dropped Out

Measure		Means	Std. Deviation	Std. Error	F	Sig.
Self Report						
	Remained	0.03	1.06	0.09		
Physical	Dropped out	-0.05	0.91	0.10	0.35	0.56
	Total	0.00	1.00	0.07		
	Remained	-0.01	1.08	0.10		
Verbal	Dropped out	0.01	0.89	0.09	0.02	0.9
	Total	0.00	1.00	0.07		
	Remained	-0.09	0.99	0.09		
Indirect	Dropped out	0.12	1.00	0.11	2.43	0.12
	Total	0.00	1.00	0.07		
	Remained	-0.04	1.06	0.10		
Total	Dropped out	0.06	0.91	0.10	0.56	0.46
	Total	0.00	1.00	0.07		
Parent Report						
	Remained	0.02	1.04	0.09		
Physical	Dropped out	-0.03	0.95	0.11	0.13	0.72
	Total	0.00	1.00	0.07		
	Remained	-0.09	1.01	0.09		
Verbal	Dropped out	0.14	0.98	0.11	2.55	0.11
	Total	0.00	1.00	0.07		
	Remained	-0.11	0.99	0.09		
Indirect	Dropped out	0.16	1.00	0.11	3.52	0.06
	Total	0.00	1.00	0.07		
	Remained	-0.08	1.01	0.09		
Total	Dropped out	0.13	0.97	0.11	2.11	0.15
	Total	0.00	1.00	0.07		

Table 1—*Continued*

Total Report						
	Remained	0.02	1.08	0.10		
Physical	Dropped out	-0.03	0.87	0.10	0.12	0.73
	Total	0.00	1.00	0.07		
	Remained	-0.08	1.07	0.01		
Verbal	Dropped out	0.12	0.87	0.10	1.80	0.18
	Total	0.00	1.00	0.07		
	Remained	-0.13	1.00	0.09		
Indirect	Dropped out	0.21	0.96	0.12	5.50	0.20
	Total	0.00	1.00	0.07		
	Remained	-0.09	1.07	0.10		
Total	Dropped out	0.15	0.87	0.10	2.73	0.10
	Total	0.00	1.00	0.07		

Table 2 Descriptive Statistics and Means for Self & Parent Reports of Psychological Health Measures at Time 1 Between Participants that Remained in the Study and those that Dropped Out

Measure		Means	Std. Deviation	Std. Error	F	Sig.
Self-Report						
Anxious Depressed	Remained	5.45	4.69	0.42	0.90	0.42
	Dropped out	6.00	4.98	0.55		
	Total	5.67	4.80	0.34		
Withdrawn Depressed	Remained	3.34	2.74	0.25	0.03	0.87
	Dropped out	3.40	2.39	0.26		
	Total	3.36	2.60	0.18		
PTSD	Remained	7.71	5.15	0.47	0.17	0.68
	Dropped out	8.01	5.18	0.58		
	Total	7.83	5.15	0.37		
Parent Report						
Anxious Depressed	Remained	3.10	3.49	0.31	0.15	0.08
	Dropped out	3.94	3.19	0.35		
	Total	3.44	3.39	0.24		
Withdrawn Depressed	Remained	1.98	2.41	0.22	1.83	0.18
	Dropped out	2.44	2.37	0.26		
	Total	2.16	2.40	0.17		
PTSD	Remained	3.87	3.92	0.36	2.13	0.15
	Dropped out	4.65	3.48	0.39		
	Total	4.18	3.76	0.26		
Total Report						

Table 2—*Continued*

Anxious Depressed	Remained	4.27	3.28	0.30		
	Dropped out	4.97	3.12	0.34	0.66	0.13
	Total	4.55	3.22	0.23		
Withdrawn Depressed	Remained	2.66	2.20	0.20		
	Dropped out	2.91	1.83	0.20	0.69	0.41
	Total	2.76	2.60	0.14		
PTSD	Remained	5.78	3.65	0.34		
	Dropped out	6.35	3.37	0.38	1.25	0.27
	Total	6.01	3.54	0.25		

Table 3 Descriptive Statistics and Means for Self & Parent Reports of Physical Health Measures at Time 1 Between Participants that Remained in the Study and those that Dropped Out

Measure		Means	Std. Deviation	Std. Error	F	Sig.
Self Report						
Frequency Health	Remained	44.16	9.35	0.85		
	Dropped out	46.35	9.53	0.99	2.82	0.09
	Total	45.10	9.47	0.65		
Severity Health	Remained	38.04	8.10	0.73		
	Dropped out	38.15	7.77	0.81	0.01	0.92
	Total	38.09	7.94	0.54		
Visits to Doctor or Nurse	Remained	5.50	1.27	0.12		
	Dropped out	5.80	1.31	0.14	2.90	0.09
	Total	5.63	1.29	0.09		
Parent Report						
Frequency Health	Remained	39.88	7.35	0.67		
	Dropped out	40.53	8.19	0.89	0.35	0.08
	Total	40.15	7.69	0.54		
Severity Health	Remained	35.30	7.11	0.64		
	Dropped out	36.78	7.25	0.79	2.11	0.15
	Total	35.90	7.19	0.50		
Visits to Doctor or Nurse	Remained	5.54	1.59	0.14		
	Dropped out	5.85	1.45	0.16	2.00	0.16
	Total	5.67	1.54	0.11		

Table 3—*Continued*

Total Report						
Frequency Health	Remained	42.05	6.90	0.63		
	Dropped out	43.50	7.11	0.78	2.13	0.15
	Total	42.65	7.01	0.49		
Severity Health	Remained	36.71	6.03	0.55		
	Dropped out	37.52	5.85	0.64	0.90	0.34
	Total	37.04	5.95	0.42		
Visits to Doctor or Nurse	Remained	5.52	1.15	0.11		
	Dropped out	5.83	1.05	0.12	3.60	0.06
	Total	5.65	1.12	0.08		

Table 4 Status and Chi-Square value at Time 1 By Participant Ethnicity for those that Remained in the Study and those that Dropped Out

Status	Native American	Asian	Black or African American	Hawaiian or other Pacific Islander	White or Anglo American	Hispanic or Latino	χ^2	Sig.
Remained	2	5	16	2	72	24		
Dropped out	0	0	17	1	50	22	8.50	0.23
Total	2	5	33	3	122	46		

Table 5 Status and Chi-Square value at Time 1 By Participant Gender for those that Remained in the Study and those that Dropped Out

Status	Male	Female	χ^2	Sig.
Remained	54	69		
Dropped out	32	57	1.35	0.25
Total	86	126		

Table 6 Descriptive Statistics for Self & Parent Reports of Victimization at Time 1

Measure	Mean	SD	Min	Max	Range	Possible Range	Skewness	Kurtosis	α
Self Reports									
Physical	10.88	3.98	7.00	28.00	21.00	28.00	1.63 (.17)	3.25 (.33)	0.83
Verbal	10.67	4.41	5.00	23.00	18.00	20.00	0.86 (.17)	0.03 (.33)	0.85
Indirect	22.92	8.27	11.86	52.00	40.00	48.00	1.01 (.17)	0.69 (.33)	0.89
Total	12.30	3.98	6.80	25.80	19.00	24.00	0.98 (.17)	0.59 (.33)	0.92
Parent Reports									
Physical	9.87	3.45	6.23	27.00	20.77	28.00	1.46 (.17)	2.50 (.34)	0.87
Verbal	10.07	3.56	5.00	23.00	18.00	20.00	0.93 (.17)	1.00 (.34)	0.89
Indirect	23.81	7.68	12.00	47.00	35.00	48.00	0.54 (.17)	.28 (.34)	0.92
Total	14.58	4.32	8.00	29.00	21.00	24.00	0.75 (.17)	0.30 (.34)	0.95
Combined Reports									
Physical	10.37	3.19	7.00	22.50	15.50	28.00	1.47 (.17)	2.34 (.34)	0.87
Verbal	10.41	3.41	5.00	21.00	16.00	20.00	0.83 (.17)	0.32 (.34)	0.87
Indirect	23.53	6.96	12.00	45.00	33.00	48.00	0.69 (.17)	.01 (.34)	0.92
Total	14.77	3.99	8.14	26.17	18.03	96.00	0.76 (.17)	0.03 (.34)	0.95

Standard errors are noted within the parentheses

Table 7 Descriptive Statistics for Self & Parent Reports of Physical Health at Time 1

Measure	Mean	SD	Min	Max	Range	Possible Range	Skewness	Kurtosis	α
Self Reports									
Frequency Health	45.10	9.47	27.00	79.00	52.00	52.00	0.67 (.17)	0.53 (.33)	0.81
Severity Health	38.09	7.94	27.00	68.00	41.00	52.00	0.94 (.17)	0.54 (.33)	0.86
Abdominal Pain	2.21	1.60	0.00	8.60	8.60	10.00	0.85 (.17)	0.87 (.33)	0.88
Visits	5.63	1.29	3.68	10.00	6.32	8.00	0.91 (.17)	0.86 (.33)	0.51
Parent Reports									
Frequency Health	40.15	7.69	26.00	73.00	47.00	52.00	0.74 (.17)	1.11 (.34)	0.86
Severity Health	35.90	7.19	26.00	59.00	33.00	52.00	0.99 (.17)	0.77 (.34)	0.90
Visits	5.67	1.54	4.00	12.00	8.00	8.00	0.99 (.17)	0.83 (.34)	0.68
Combined Reports									
Frequency Health	42.65	7.00	29.00	67.50	38.50	52.00	0.49 (.17)	.33 (.34)	0.89
Severity Health	37.04	5.95	27.00	57.00	30.00	52.00	0.61 (.17)	0.37 (.34)	0.90
Visits	5.65	1.12	4.00	8.58	4.58	8.00	0.67 (.17)	.08 (.34)	0.66

Standard errors are noted within the parentheses

Table 8 Descriptive Statistics for Self & Parent Reports of Psychological Health at Time 1

Measure	Mean	SD	Min	Max	Range	Possible Range	Skewness	Kurtosis
Self Reports								
Anxious Depressed	5.67	4.80	0.00	20.00	20.00	26.00	0.99 (.17)	0.43 (.34)
Withdrawn Depressed	3.34	2.60	0.00	12.00	12.00	12.00	0.83 (.17)	0.40 (.34)
PTSD	7.83	5.15	0.00	25.00	25.00	26.00	0.76 (.17)	0.18 (.34)
Parent Reports								
Anxious Depressed	3.44	3.39	0.00	18.00	18.00	26.00	1.64 (.17)	3.28 (.34)
Withdrawn Depressed	2.16	2.40	0.00	12.00	12.00	12.00	1.46 (.17)	2.43 (.34)
PTSD	4.18	3.76	0.00	22.00	22.00	26.00	1.42 (.17)	2.74 (.34)
Combined Reports								
Anxious Depressed	4.55	3.22	0.00	17.50	17.50	26.00	1.11 (.17)	1.50 (.34)
Withdrawn Depressed	2.76	2.06	0.00	10.00	10.00	12.00	1.05 (.17)	1.19 (.34)
PTSD	6.00	3.54	0.00	22.00	22.00	26.00	0.79 (.17)	1.21 (.35)

Standard errors are noted within the parentheses

Table 9 Correlations of Self and Parent Reports of Victimization at T1

Measures	Physical Self Report	Verbal Self Report	Indirect Self Report	Physical Parent Report	Physical Parent Report	Physical Parent Report
Physical Self Report	1.00					
Verbal Self Report	.74**	1.00				
Indirect Self Report	.61**	.79**	1.00			
Physical Parent Report	.55**	.46**	.38**	1.00		
Verbal Parent Report	.36**	.52**	.51**	.65**	1.00	
Indirect Parent Report	.31**	.47**	.56**	.53**	.79**	1.00

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

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

Table 10 Correlations of Self and Parent Reports of Psychological Health at Time 1

Measures	Anxious Depressed Self Report	Withdrawn Depressed Self Report	PTSD Self Report	Anxious Depressed Parent Report	Withdrawn Depressed Parent Report	PTSD Parent Report
Anxious Depressed Self Report	1.00					
Withdrawn Depressed Self Report	.62**	1.00				
PTSD Self Report	.82**	.75**	1.00			
Anxious Depressed Parent Report	.27**	0.17	.23*	1.00		
Withdrawn Depressed Parent Report	.20*	.46**	.27**	.47**	1.00	
PTSD Parent Report	0.15	0.16	.26**	.77**	.60**	1.00

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

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

Table 11 Correlations of Self and Parent Reports of Physical Health at Time 1

Measures	Frequency Health Self Report	Severity Health Self Report	Visits to Doctor or Nurse Self Report	Frequency Health Parent Report	Severity Health Parent Report	Visits to Doctor or Nurse Parent Report
Frequency Health Self Report	1.00					
Severity Health Self Report	.83**	1.00				
Visits to Doctor or Nurse Self Report	.47**	.40**	1.00			
Frequency Health Parent Report	.34**	.23*	0.17	1.00		
Severity Health Parent Report	.36**	.24**	.18*	.81**	1.00	
Visits to Doctor or Parent Report	.45**	.33**	.27**	.59**	.69**	1.00

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

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

Table 12 Descriptive Statistics for Self & Parent Reports of Victimization at Time 2

Measure	Mean	SD	Min	Max	Range	Possible Range	Skewness	Kurtosis	α
Self Reports									
Physical	9.19	3.04	7.00	28.00	21.00	28.00	2.79 (.22)	11.66 (.43)	0.83
Verbal	9.06	3.90	5.00	21.00	16.00	20.00	1.20 (.22)	0.99 (.43)	0.85
Indirect	20.35	7.42	12.00	44.81	32.81	48.00	1.28 (.22)	1.42 (.43)	0.89
Total	10.61	3.30	6.80	24.43	17.63	24.00	1.37 (.22)	2.26 (.43)	0.92
Parent Reports									
Physical	8.77	2.74	7.00	24.00	17.00	28.00	1.46 (.22)	8.13 (.43)	0.87
Verbal	8.72	3.20	4.65	23.00	18.35	20.00	2.47 (.22)	5.05 (.43)	0.89
Indirect	20.32	6.82	12.00	44.00	32.50	48.00	1.09 (.22)	1.38 (.43)	0.92
Total	19.52	2.47	6.66	21.20	14.54	24.00	1.17 (.22)	2.26 (.43)	0.95
Combined Reports									
Physical	8.98	2.45	7.00	20.00	13.00	28.00	2.08 (.22)	5.06 (.43)	0.86
Verbal	8.89	3.09	4.83	22.00	17.17	20.00	1.48 (.22)	2.91 (.43)	0.87
Indirect	20.34	6.24	12.00	44.25	32.25	48.00	1.25 (.22)	1.70 (.43)	0.93
Total	10.45	2.43	6.90	18.75	11.85	24.00	1.25 (.22)	1.42 (.43)	0.95

Standard errors are noted within the parentheses

Table 13 Descriptive Statistics for Self & Parent Reports of Physical Health at Time 2

Measure	Mean	SD	Min	Max	Range	Possible Range	Skewness	Kurtosis	α
Self Reports									
Frequency Health	43.54	9.38	28.00	68.00	40.00	52.00	0.46 (.22)	.31 (.43)	0.89
Severity Health	37.95	9.13	27.00	66.00	39.00	52.00	0.97 (.22)	0.14 (.43)	0.91
Visits	5.04	1.46	4.00	12.00	8.00	8.00	2.35 (.22)	6.93 (.43)	0.63
Parent Reports									
Frequency Health	40.12	7.16	26.73	59.00	32.27	52.00	0.25 (.22)	.41 (.43)	0.87
Severity Health	35.77	6.51	27.00	59.81	32.81	52.00	1.02 (.17)	1.43 (.43)	0.88
Visits	5.49	1.43	3.40	10.00	6.60	8.00	0.80 (.22)	.11 (.43)	0.71
Combined Reports									
Frequency Health	41.83	7.22	28.50	58.50	30.00	52.00	0.43 (.22)	.53 (.43)	0.92
Severity Health	36.86	6.49	27.46	58.50	31.04	52.00	1.01 (.22)	0.99 (.43)	0.90
Visits	5.27	1.16	4.00	10.50	6.50	8.00	1.54 (.22)	3.78 (.43)	0.70

Standard errors are noted within the parentheses

Table 14 Descriptive Statistics for Self & Parent Reports of Psychological Health at Time 2

Measure	Mean	SD	Min	Max	Range	Possible Range	Skewness	Kurtosis
Self Reports								
Anxious Depressed	5.08	4.44	0.00	24.00	24.00	26.00	1.70 (.22)	3.73 (.43)
Withdrawn Depressed	3.13	2.55	0.00	13.00	13.00	12.00	1.12 (.22)	1.44 (.43)
PTSD	7.33	4.64	0.00	22.00	22.00	26.00	1.14 (.22)	1.73 (.43)
Parent Reports								
Anxious Depressed	3.07	3.34	0.00	20.00	20.00	26.00	2.20 (.22)	7.23 (.43)
Withdrawn Depressed	2.21	2.50	0.00	12.00	12.00	12.00	1.42 (.22)	1.96 (.43)
PTSD	3.85	3.60	0.00	22.00	22.00	26.00	1.74 (.22)	4.79 (.43)
Combined Reports								
Anxious Depressed	4.07	3.13	0.00	19.50	19.50	26.00	1.64 (.22)	4.78 (.43)
Withdrawn Depressed	2.67	2.01	0.00	10.00	10.00	12.00	0.97 (.22)	0.80 (.43)
PTSD	5.59	3.28	0.00	21.50	21.50	26.00	1.29 (.22)	3.52 (.43)

Standard errors are noted within the parentheses

Table 15 Correlations of Self with Parent Reports of Victimization at T2

Measures	Physical Self Report	Verbal Self Report	Indirect Self Report	Physical Parent Report	Physical Parent Report	Physical Parent Report
Physical Self Report	1.00					
Verbal Self Report	.68**	1.00				
Indirect Self Report	.50**	.80**	1.00			
Physical Parent Report	.44**	.39**	.27**	1.00		
Verbal Parent Report	.29**	.51**	.47**	.66**	1.00	
Indirect Parent Report	.20*	.44**	.54**	.57**	.79**	1.00

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

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

Table 16 Correlations of Self and Parent Reports of Psychological Health at Time 2

Measures	Anxious Depressed Self Report	Withdrawn Depressed Self Report	PTSD Self Report	Anxious Depressed Parent Report	Withdrawn Depressed Parent Report	PTSD Parent Report
Anxious Depressed Self Report	1.00					
Withdrawn Depressed Self Report	.65**	1.00				
PTSD Self Report	.88**	.74**	1.00			
Anxious Depressed Parent Report	.27**	0.11	.18*	1.00		
Withdrawn Depressed Parent Report	.19*	.26**	0.18	.54**	1.00	
PTSD Parent Report	.23*	0.11	.25**	.79**	.67**	1.00

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

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

Table 17 Correlations of Self and Parent Reports of Physical Health at Time 2

Measures	Frequency Health Self Report	Severity Health Self Report	Visits to Doctor or Nurse Self Report	Frequency Health Parent Report	Severity Health Parent Report	Visits to Doctor or Parent Report
Frequency Health Self Report	1.00					
Severity Health Self Report	.75**	1.00				
Visits to Doctor or Nurse Self Report	.47**	.51**	1.00			
Frequency Health Parent Report	.52**	.38**	.32**	1.00		
Severity Health Parent Report	.35**	.36**	.24**	.77**	1.00	
Visits to Doctor or Parent Report	.27**	.29**	.28**	.46**	.56**	1.00

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

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

Table 18 Correlations of Victimization Measures at Time 1 and Time 2

		Time 1		
	Measures	Physical	Verbal	Indirect
Time 2	Physical	0.66**	0.47**	0.34**
	Verbal	0.49**	0.58**	0.48**
	Indirect	0.35**	0.46**	0.55**

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

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

Table 19 Means and Standard Errors for Physical & Psychological Health for Each Victim Group

	Non Victims		New Victims		Escaped Victims		Persistent Victims	
Physical Health	*M*	*S.E*	*M*	*S.E*	*M*	*S.E*	*M*	*S.E*
Frequency	40.01_{a}	0.67	49.54_{b}	2.25	42.28_{ab}	2.12	49.31_{b}	1.84
Severity	35.40_{a}	0.61	43.75_{b}	2.05	37.32_{ab}	1.93	41.25_{b}	1.67
Visits to doctor/nurse	5.01_{a}	0.11	5.67_{a}	0.36	5.44_{ab}	0.34	6.42_{b}	0.29
Psychological Health								
Anxious Depressed	3.50_{a}	0.31	7.31_{b}	1.04	3.56_{ab}	0.98	6.54_{b}	0.85
Withdrawn Depressed	2.39_{a}	0.21	3.56_{a}	0.69	2.33_{ab}	0.65	4.38_{b}	0.56
PTSD	4.80_{a}	0.31	7.94_{b}	1.02	5.67_{ac}	0.97	9.46_{b}	0.84

note: a, b, c suggests that groups are significantly different from each other

Table 20 Correlations between time 1 and time 2 time measurements of AUC_g and CAR_g

	AUC_g T2	CAR_g T2
AUC_g T1	0.22*	-0.06
CAR_g T1	0.01	0.07

Table 21 Regression and summary statistics when using normal OLS regression, and robust regression procedures for all AUC Time 2 measures

Parameter	*b*	*SE*	Corrected 95% Confidence Interval Lower	Upper	*p*
Change in AUC_g					
Intercept	2074.15	329.92	142.179	2636.80	0.00
Difference in run dates	26.41	5.61	16.34	36.52	0.00
Gender	44.61	38.11	-28.10	114.51	0.24
Victim T1	-59.23	30.70	-120.63	-2.45	0.06
Victim x gender	69.11	30.86	8.09	127.87	0.03
AUCg T1	0.22	0.15	-0.04	0.53	0.14
AUC_g					
Intercept	2574.89	40.56	2496.08	2654.02	0.00
Difference in run dates	26.43	5.62	15.73	37.04	0.00
Gender	51.25	39.05	-23.96	125.36	0.19
Victim T1	-69.081	28.45	-127.36	-17.26	0.02
Victim x gender	61.35	28.65	4.73	119.63	0.04

Table 22 Regression and summary statistics when using normal OLS regression, and robust regression procedures for CAR variables at Time 2

Parameter	*b*	*SE*	Corrected 95% Confidence Interval Lower	Upper	*p*
Change in CAR_g					
Intercept	75.38	41.17	-31.17	130.72	0.20
Difference in run dates	-2.82	2.21	-8.45	0.92	0.37
Gender	-1.88	10.12	-29.06	20.32	0.85
Victim T1	-14.29	5.30	-34.24	2.77	0.04
Victim x gender	-6.49	5.47	-25.55	10.13	0.29
CAR_g T1	0.29	0.28	-0.07	0.95	0.36

Table 23 Correlations of psychological and physical health and cortisol measures for T2

Measures	AUCg T2	CARg T2
Boys		
frequency	-0.37*	0.13
severity	-0.28	0.01
visits to doctor or nurse	-0.12	0.01
anxious depression	-0.48**	-0.05
withdrawn depression	-0.37**	-0.10
PTSD	-0.38**	0.00
Girls		
frequency of health	0.17	0.02
severity of health	0.03	0.09
visits to doctor or nurse	-0.10	0.04
anxious depression	0.03	0.01
withdrawn depression	0.01	-0.11
PTSD	0.04	0.00

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

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

Table 24 Conditional indirect effects at specific values of the moderator for each outcome variable AUC_g (Figure 10)

	Health Outcomes	Indirect Effects	SE	Lower CI	Upper CI
Boys					
	Frequency Health	0.40	0.29	-0.05	1.11
	Severity Health	0.27	0.18	-0.00	0.70
	Visits to Doctor	0.01	0.04	-0.07	0.09
	Withdrawn Depressed	**0.15**	**0.07**	**0.03**	**0.33**
	Anxious Depressed	**0.31**	**0.17**	**0.08**	**0.79**
	PTSD Symptoms	**0.24**	**0.14**	**0.03**	**0.59**
Girls					
	Frequency Health	-0.03	0.27	-0.80	0.40
	Severity Health	-0.00	0.13	-0.42	0.19
	Visits to Doctor	0.00	0.02	-0.03	0.06
	Withdrawn Depressed	-0.00	0.04	-0.11	0.07
	Anxious Depressed	-0.00	0.06	-0.17	0.08
	PTSD Symptoms	-0.00	0.07	-0.23	0.09

Bold indicates significant mediation

Table 25 Conditional indirect effects at each outcome variable run separately for boys and girls for AUC_g (Figure 11)

Health Outcomes	Indirect Effects	SE	Lower CI	Upper CI
Boys				
Frequency Health	**0.52**	**0.33**	**0.05**	**1.39**
Severity Health	**0.42**	**0.22**	**0.10**	**1.02**
Visits to Doctor	0.04	0.04	-0.02	0.13
Withdrawn Depressed	**0.18**	**0.09**	**0.04**	**0.42**
Anxious Depressed	**0.36**	**0.19**	**0.09**	**0.87**
PTSD Symptoms	**0.30**	**0.15**	**0.07**	**0.72**
Girls				
Frequency Health	-0.03	0.28	-0.83	0.38
Severity Health	-0.00	0.14	-0.42	0.22
Visits to Doctor	0.00	0.02	-0.03	0.06
Withdrawn Depressed	-0.00	0.04	-0.11	0.07
Anxious Depressed	-0.00	0.06	-0.17	0.09
PTSD Symptoms	-0.00	0.02	-0.20	0.11

Bold indicates significant mediation

Table 26 Conditional indirect effects at specific values of the moderator for each outcome variable for CAR_g (Figure 10)

Health Outcomes	Indirect Effects	SE	Lower CI	Upper CI
Boys				
Frequency Health	-0.27	0.32	-1.18	0.15
Severity Health	-0.08	0.19	-0.54	0.21
Visits to Doctor	-0.01	0.02	-0.08	0.02
Withdrawn Depressed	0.01	0.08	-0.10	0.24
Anxious Depressed	-0.01	0.17	-0.28	0.45
PTSD Symptoms	-0.06	0.17	-0.42	0.29
Girls				
Frequency Health	-0.03	0.08	-0.25	0.08
Severity Health	-0.06	0.09	-0.38	0.07
Visits to Doctor	-0.01	0.02	-0.04	0.01
Withdrawn Depressed	0.02	0.03	-0.02	0.10
Anxious Depressed	-0.01	0.04	-0.13	0.02
PTSD Symptoms	-0.01	0.05	-0.23	0.09

Table 27 Conditional indirect effects at each outcome variable run separately for boys and girls for CAR_g (Figure 11)

	Health Outcomes	Indirect Effects	SE	Lower CI	Upper CI
Boys					
	Frequency Health	-0.24	0.31	-1.03	0.19
	Severity Health	-0.02	0.16	-0.35	0.36
	Visits to Doctor	-0.00	0.02	-0.05	0.03
	Withdrawn Depressed	0.02	0.08	-0.07	0.32
	Anxious Depressed	0.01	0.16	-0.22	0.53
	PTSD Symptoms	-0.04	0.16	-0.34	0.35
Girls					
	Frequency Health	-0.04	0.09	-0.31	0.07
	Severity Health	-0.07	0.11	-0.41	0.07
	Visits to Doctor	-0.01	0.02	-0.06	0.01
	Withdrawn Depressed	-0.02	0.03	-0.02	0.10
	Anxious Depressed	-0.01	0.05	-0.17	0.02
	PTSD Symptoms	-0.01	0.05	-0.17	0.06

Table 28 Conditional indirect effects at each outcome variable run separately for boys and girls for AUC_g (Figure 13)

Health Outcomes	Indirect Effects	SE	Lower CI	Upper CI
Boys				
Time 1 Psychological Health				
Withdrawn Depressed	-25.05	22.55	-91.61	1.56
Anxious Depressed	-37.54	26.82	-110.40	0.23
PTSD Symptoms	**-73.10**	**40.39**	**-175.07**	**-9.01**
Time 2 Psychological Health				
Withdrawn Depressed	-30.98	29.45	-102.46	19.50
Anxious Depressed	-43.38	28.71	-104.91	9.12
PTSD Symptoms	**-62.16**	**34.99**	**-152.65**	**-8.79**
Girls				
Time 1 Psychological Health				
Withdrawn Depressed	24.79	35.47	-37.50	109.01
Anxious Depressed	20.21	32.52	-37.84	94.39
PTSD Symptoms	4.38	55.74	-132.45	98.23
Time 2 Psychological Health				
Withdrawn Depressed	2.92	32.31	-65.48	65.45
Anxious Depressed	5.95	22.26	-26.88	70.53
PTSD Symptoms	14.63	44.73	-71.44	116.46

Bold indicates significant mediation

Table 29 Conditional indirect effects at each outcome variable run separately for boys and girls for frequency of health (Figure 15)

Health Outcomes	Indirect Effects	SE	Lower CI	Upper CI
Boys				
Time 1 Frequency of Health				
Withdrawn Depressed	0.07	0.13	-0.06	0.57
Anxious Depressed	0.23	0.23	-0.06	0.91
PTSD Symptoms	0.49	0.43	-0.15	1.62
Time 2 Frequency of Health				
Withdrawn Depressed	0.27	0.26	-0.11	1.03
Anxious Depressed	**0.51**	**0.29**	**0.02**	**1.21**
PTSD Symptoms	**1.01**	**0.42**	**0.27**	**1.95**
Girls				
Time 1 Frequency of Health				
Withdrawn Depressed	**1.93**	**0.58**	**0.97**	**3.31**
Anxious Depressed	**1.32**	**0.53**	**0.49**	**2.64**
PTSD Symptoms	**2.70**	**0.67**	**1.56**	**4.23**
Time 2 Frequency of Health				
Withdrawn Depressed	**1.84**	**0.54**	**0.89**	**3.03**
Anxious Depressed	**1.12**	**0.56**	**0.17**	**2.42**
PTSD Symptoms	**2.06**	**0.66**	**0.98**	**3.55**

Bold indicates significant mediation

Table 30 Conditional indirect effects at each outcome variable run separately for boys and girls for severity of health (Figure 15)

Health Outcomes	Indirect Effects	SE	Lower CI	Upper CI
Boys				
Time 1 Severity of Health				
Withdrawn Depressed	0.04	0.11	-0.06	0.44
Anxious Depressed	0.17	0.15	-0.01	0.59
PTSD Symptoms	0.11	0.25	-0.34	0.72
Time 2 Severity of Health				
Withdrawn Depressed	0.13	0.18	-0.07	0.71
Anxious Depressed	0.44	0.29	-0.03	1.16
PTSD Symptoms	**0.67**	**0.29**	**0.21**	**1.45**
Girls				
Time 1 Severity of Health				
Withdrawn Depressed	**0.13**	**0.07**	**0.03**	**0.31**
Anxious Depressed	**1.01**	**0.46**	**0.27**	**2.08**
PTSD Symptoms	**1.83**	**0.59**	**0.82**	**3.17**
Time 2 Severity of Health				
Withdrawn Depressed	**0.95**	**0.44**	**0.28**	**2.07**
Anxious Depressed	**0.74**	**0.44**	**0.09**	**1.85**
PTSD Symptoms	**1.37**	**0.50**	**0.57**	**2.55**

Bold indicates significant mediation

Appendix B

List of Figure

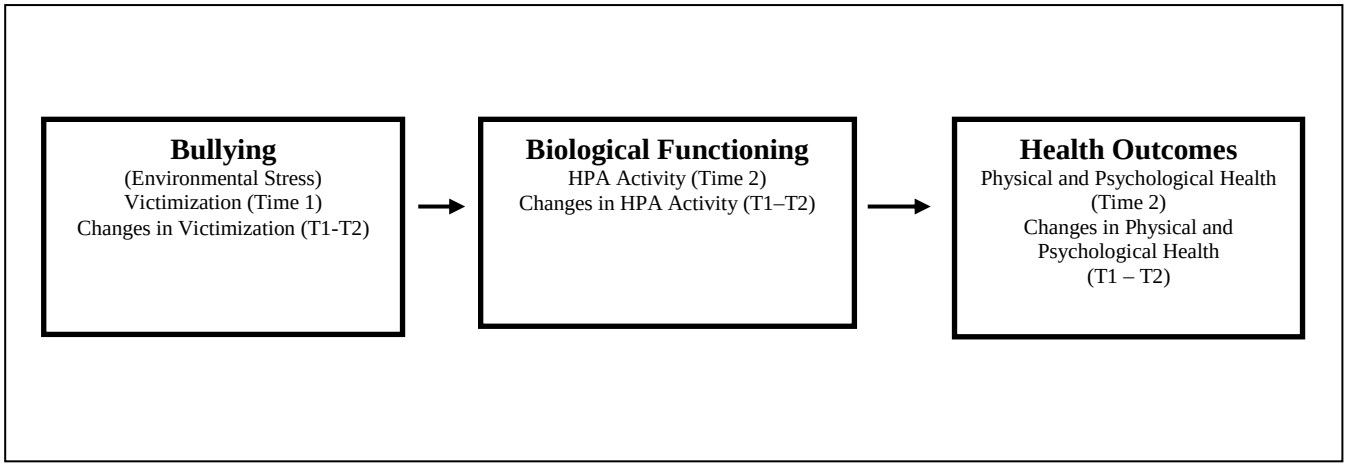

Figure 1 Theoretical Model of Peer Victimization's Influence on Health over Time

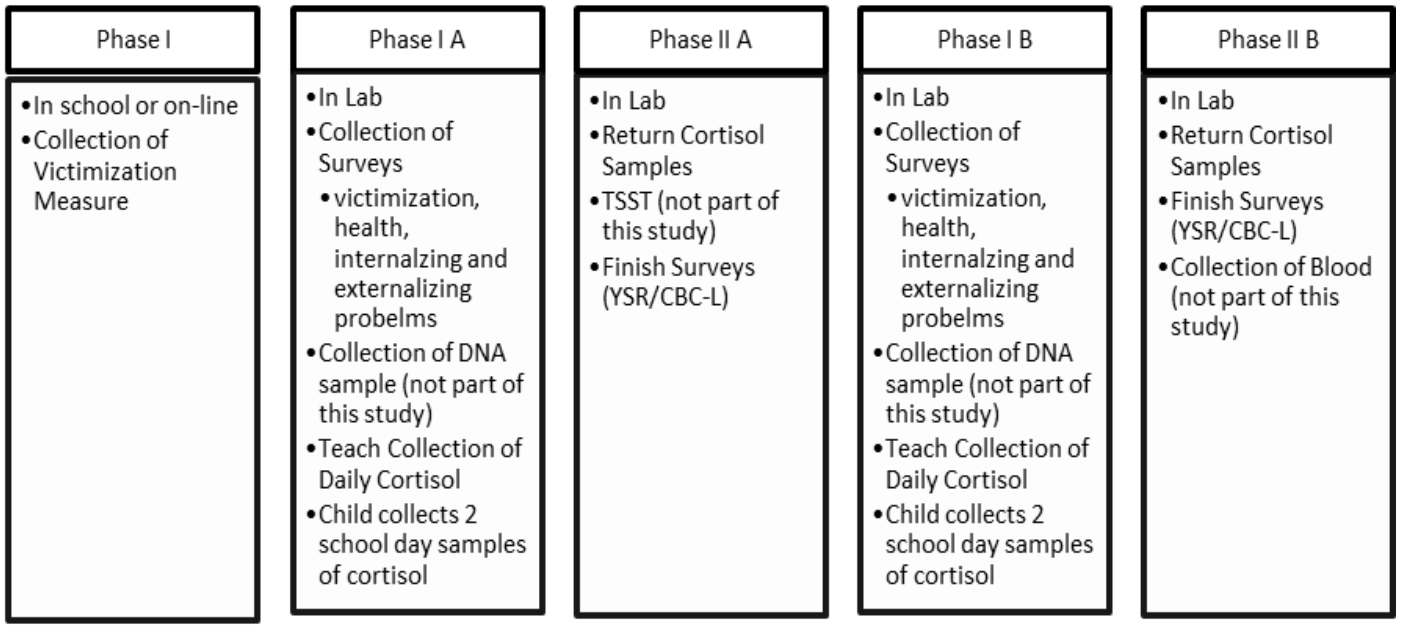

Figure 2 Timeline of Project

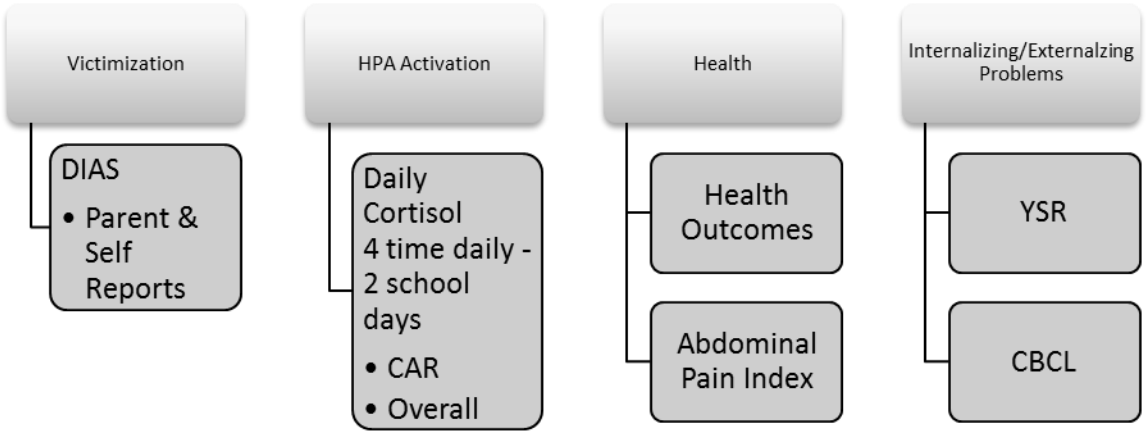

Figure 3 Constructs of Interest and Their Measures

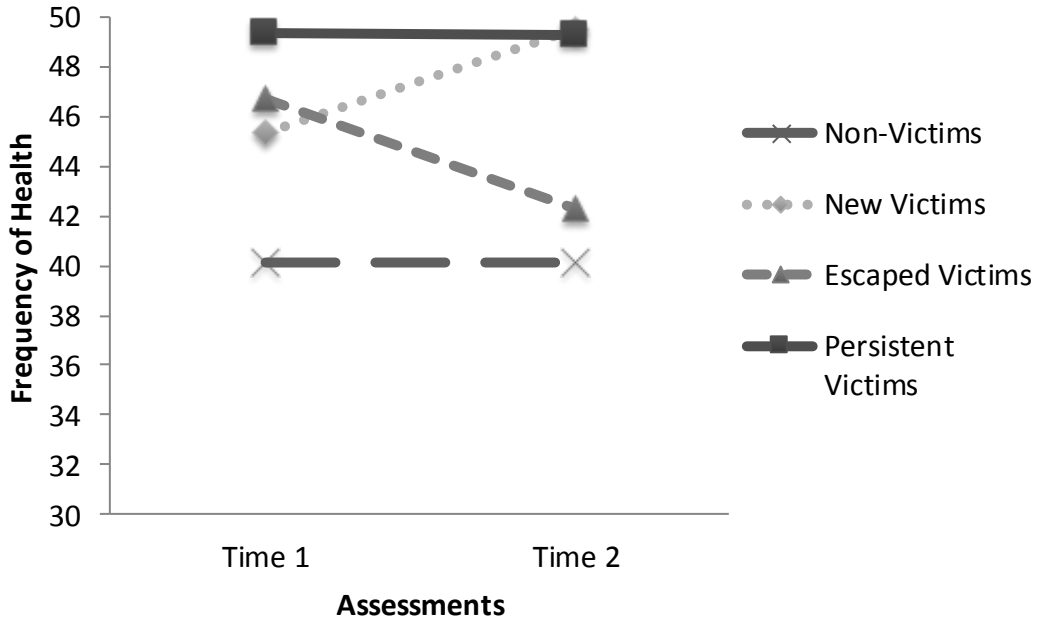

Figure 4 Changes in frequency between time 1 and time 2 by group membership

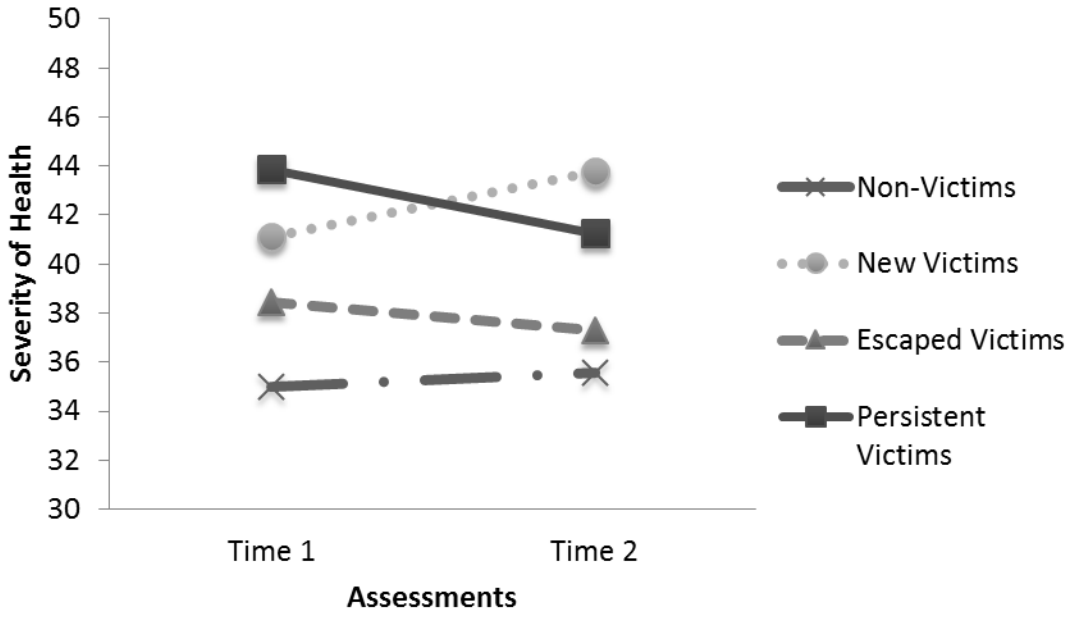

Figure 5 Changes in severity between time 1 and time 2 by group membership

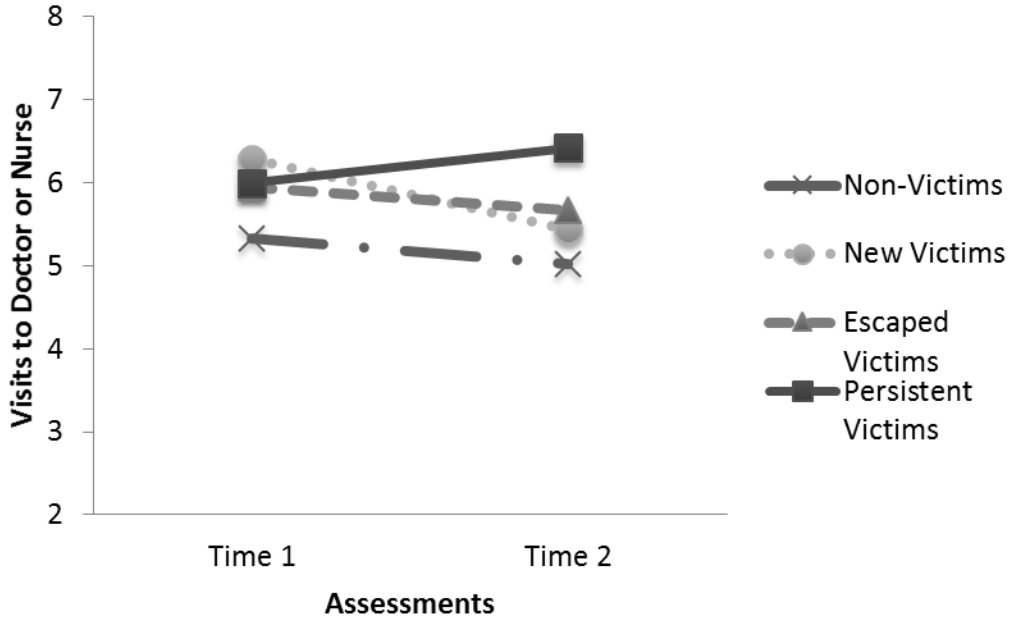

Figure 6 Changes in visits to the doctor or nurse between time 1 and time 2 by group membership

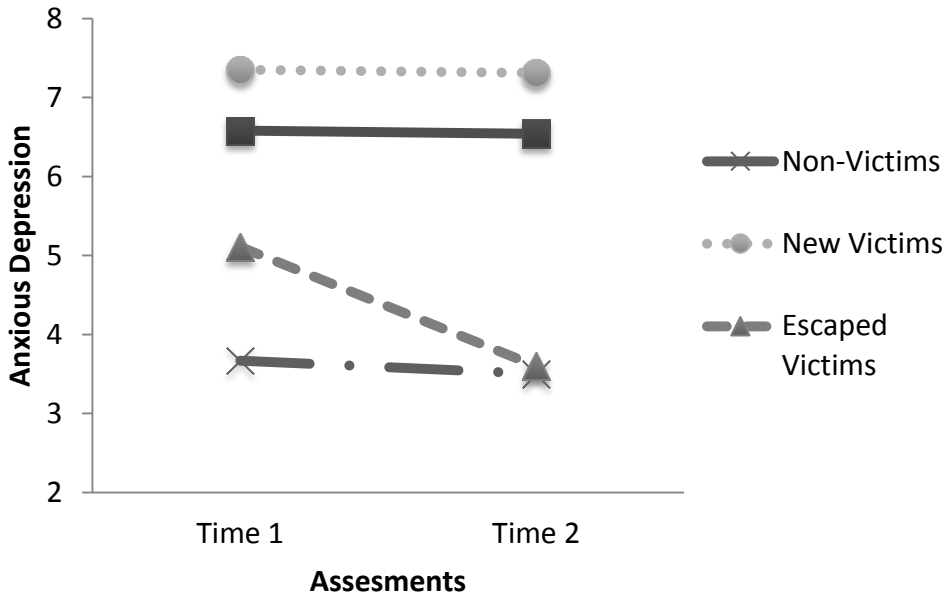

Figure 7 Changes in anxious depression between time 1 and time 2 by group membership

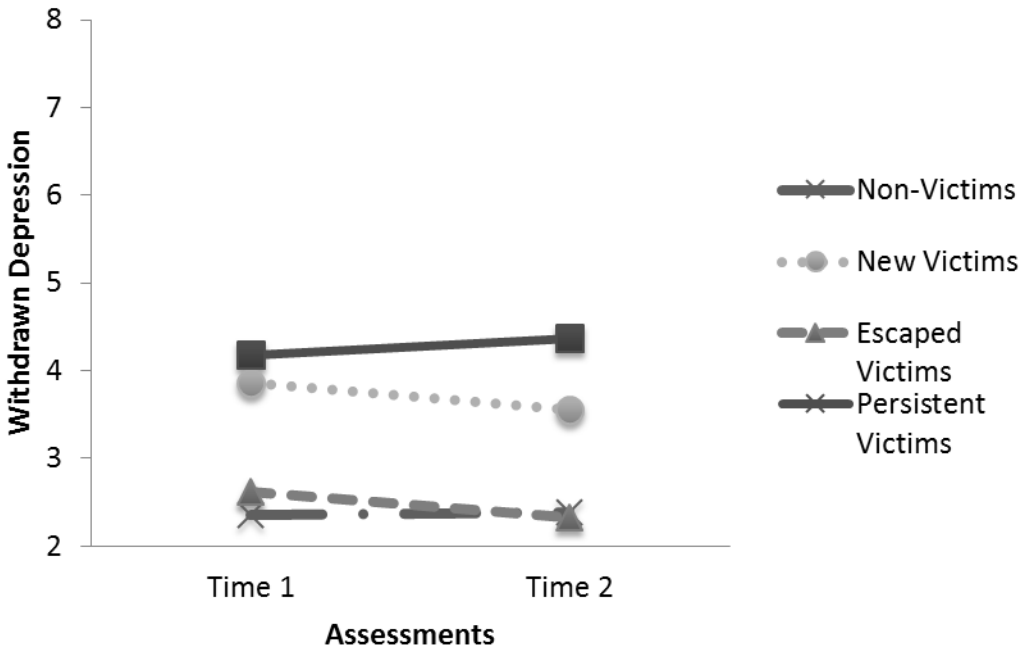

Figure 8 Changes in withdrawn depression between time 1 and time 2 by group membership

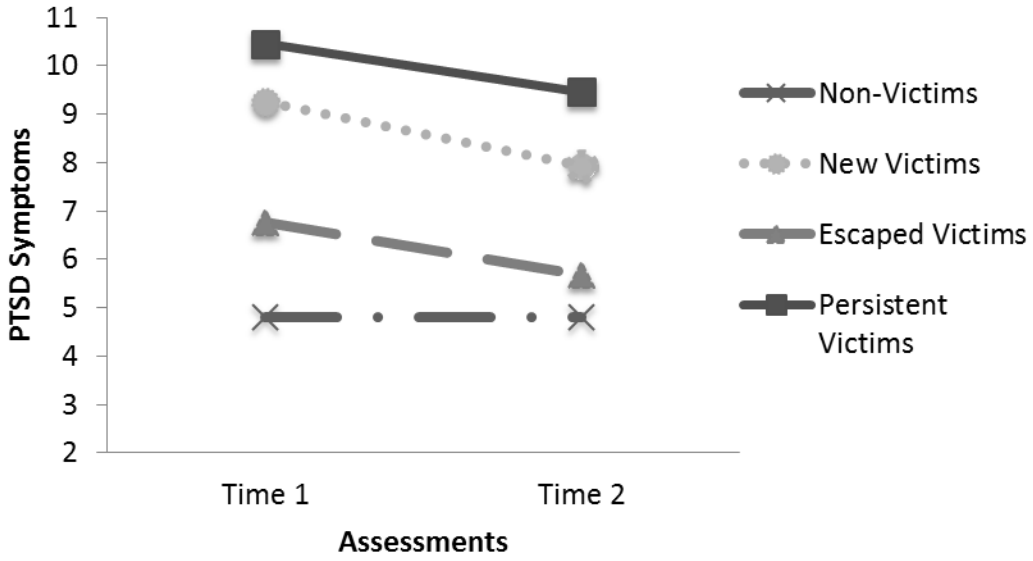

Figure 9 Change symptoms of PTSD between time 1 and time 2 by group membership

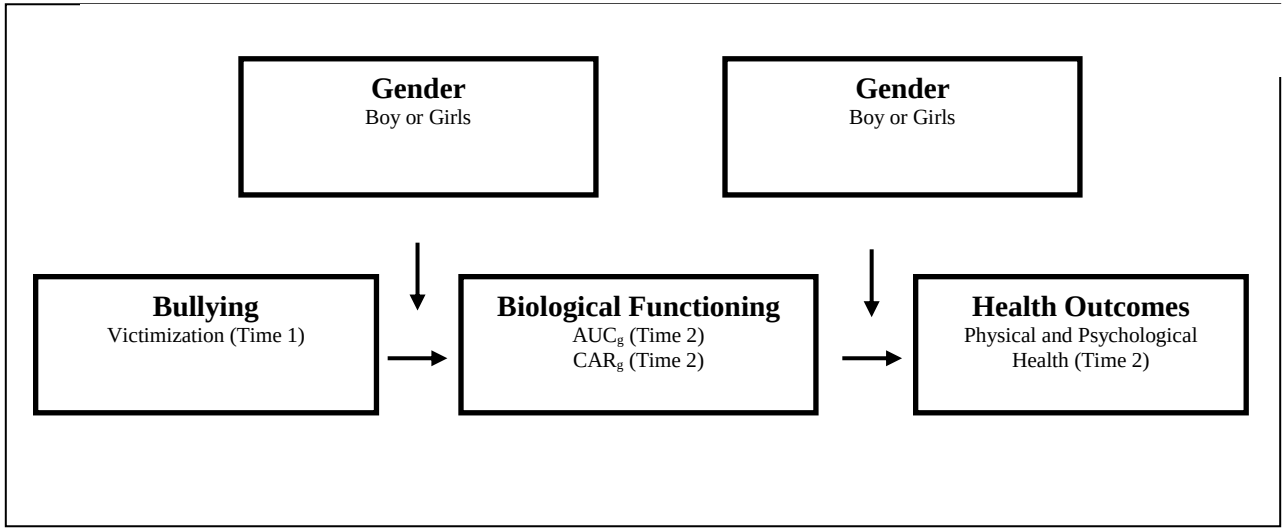

Figure 10 Mediation/Moderation model between peer victimization, AUCg or CARg, health, and gender

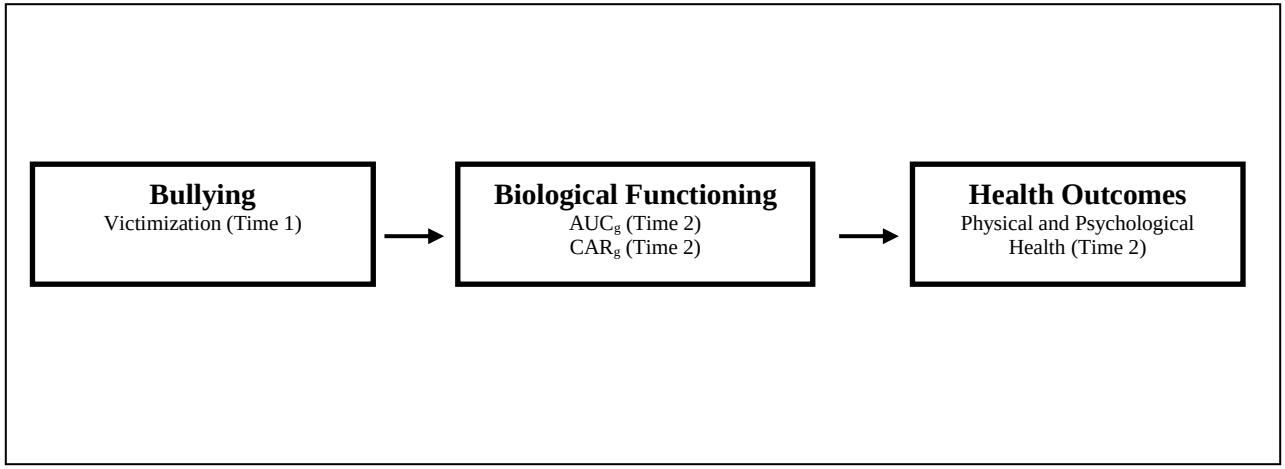

Figure 11 Mediation model between peer victimization, AUCg or CARg and health run separately for boys and girls

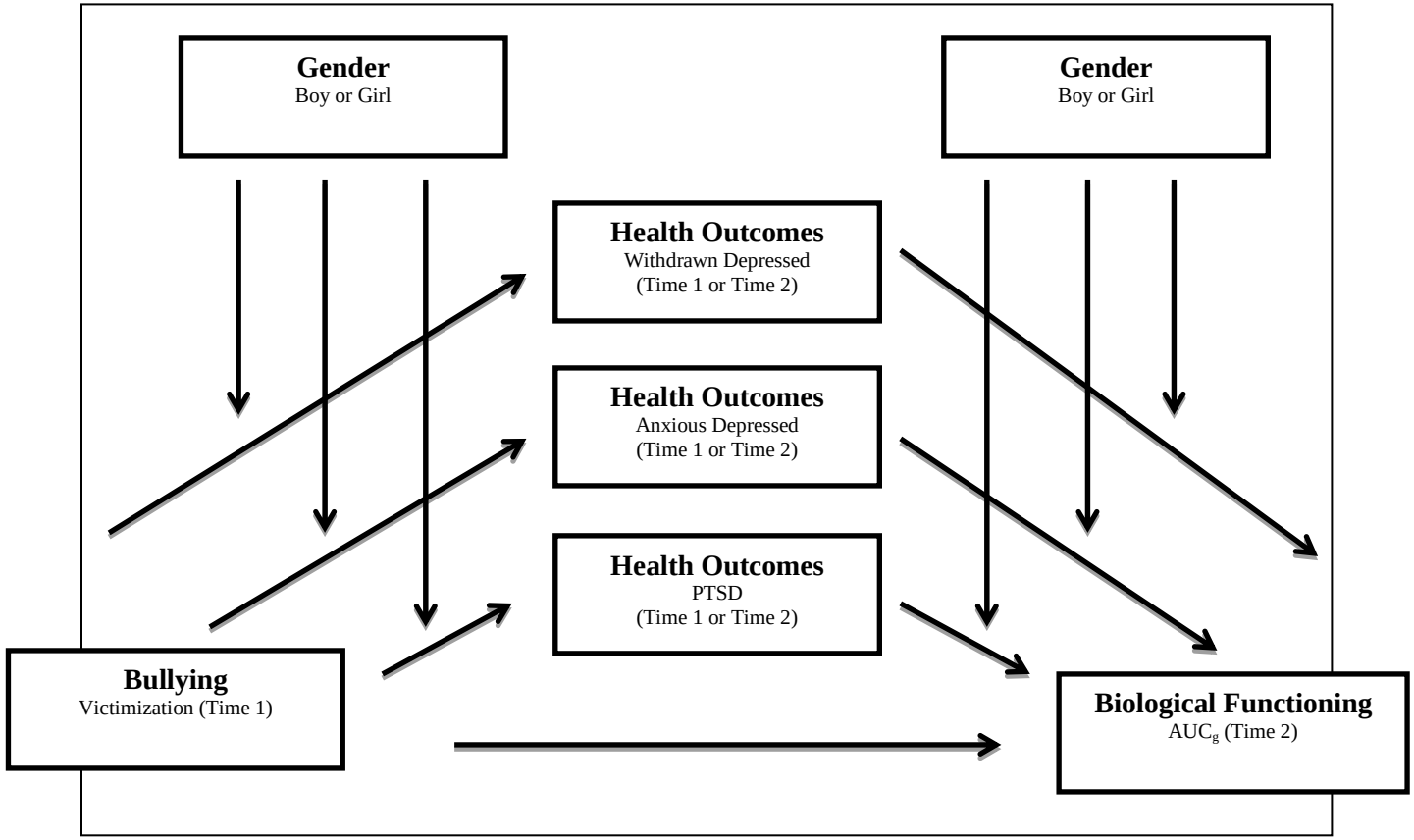

Figure 12 Mediation/Moderation model between peer victimization, AUCg, psychological health outcomes at time 1 or time 2, and gender

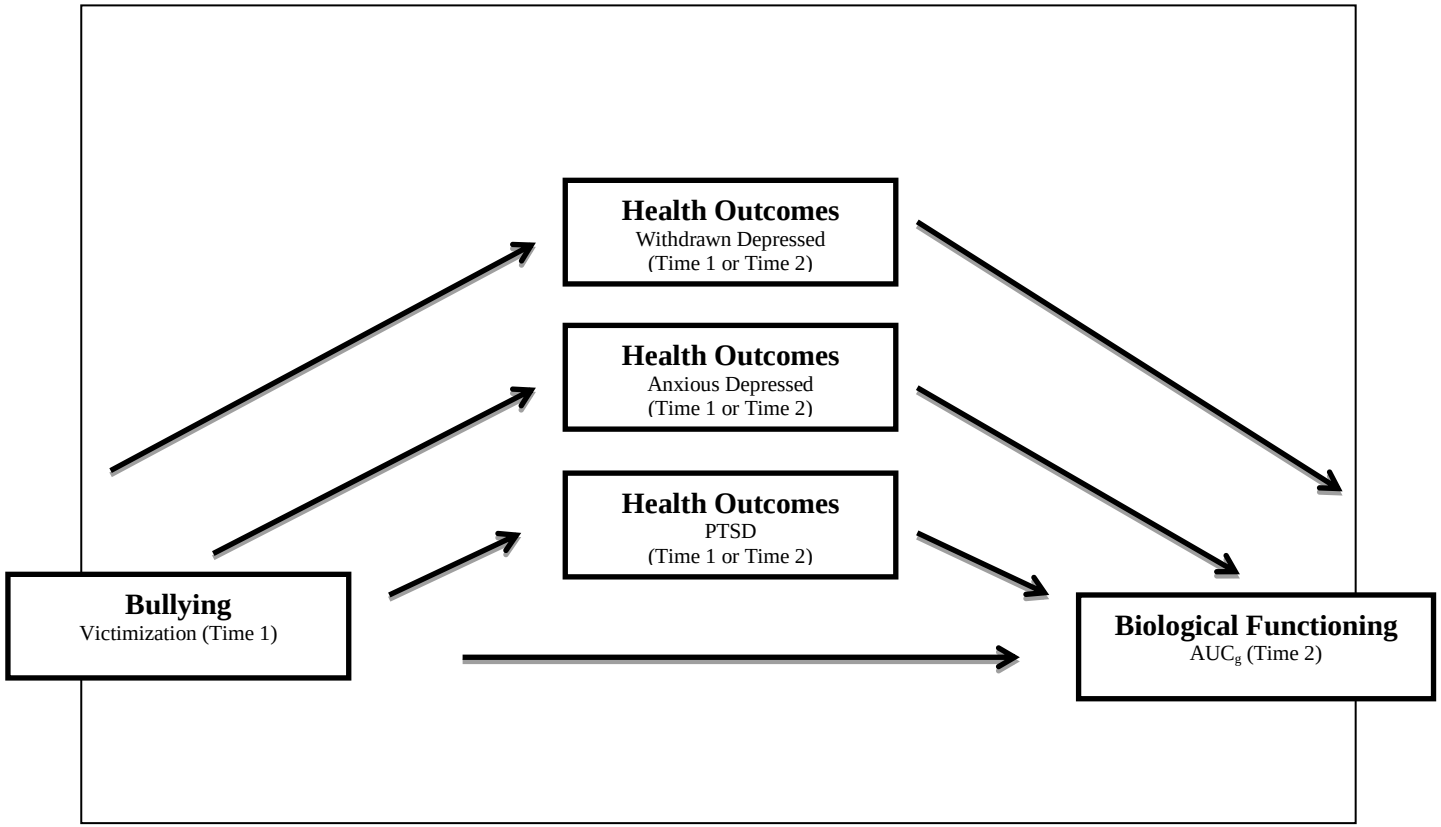

Figure 13 Mediation model between peer victimization, AUCg, psychological health outcomes at time 1 or time 2

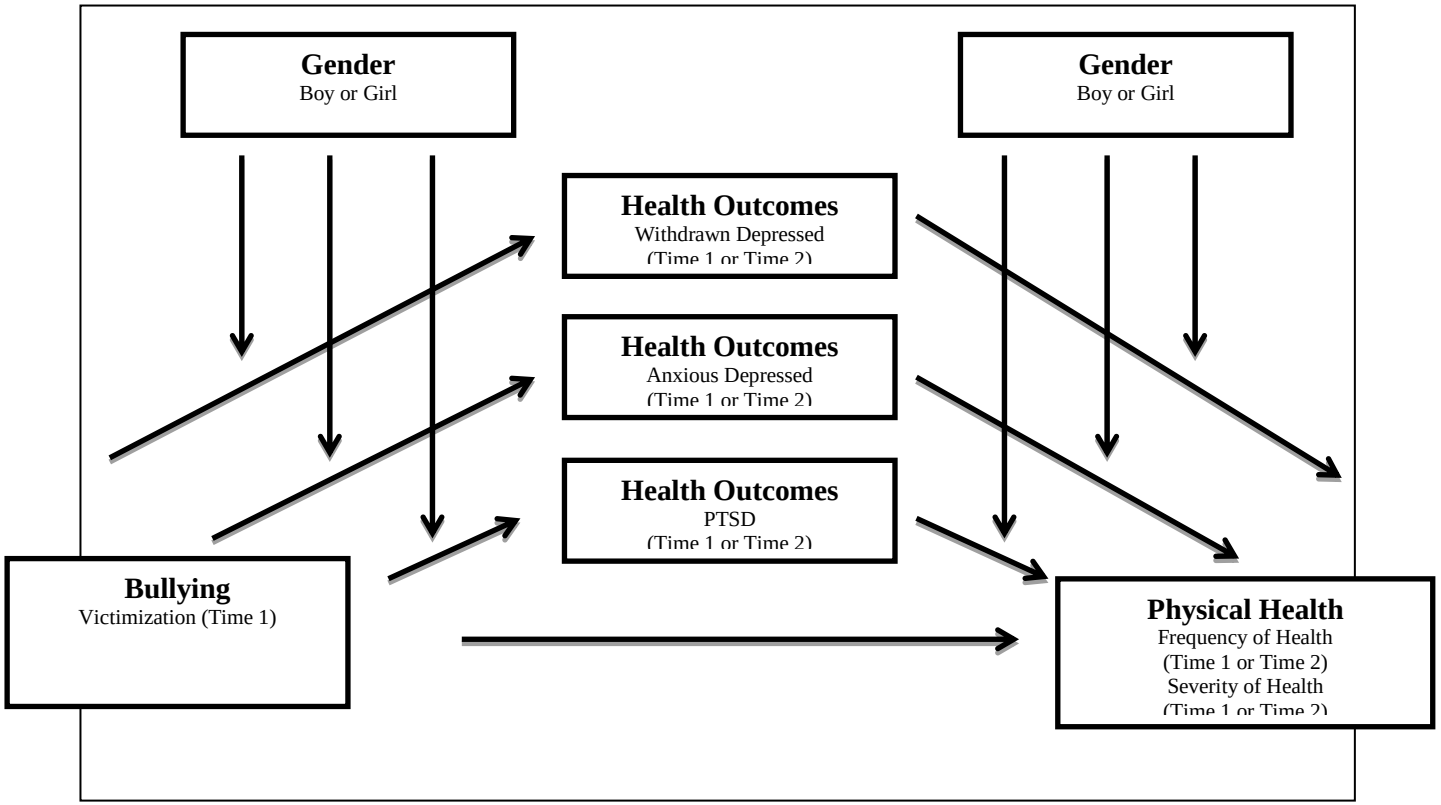

Figure 14 Mediation/Moderation model between peer victimization, frequency or severity of health, psychological health outcomes at time 1 or time 2, and gender

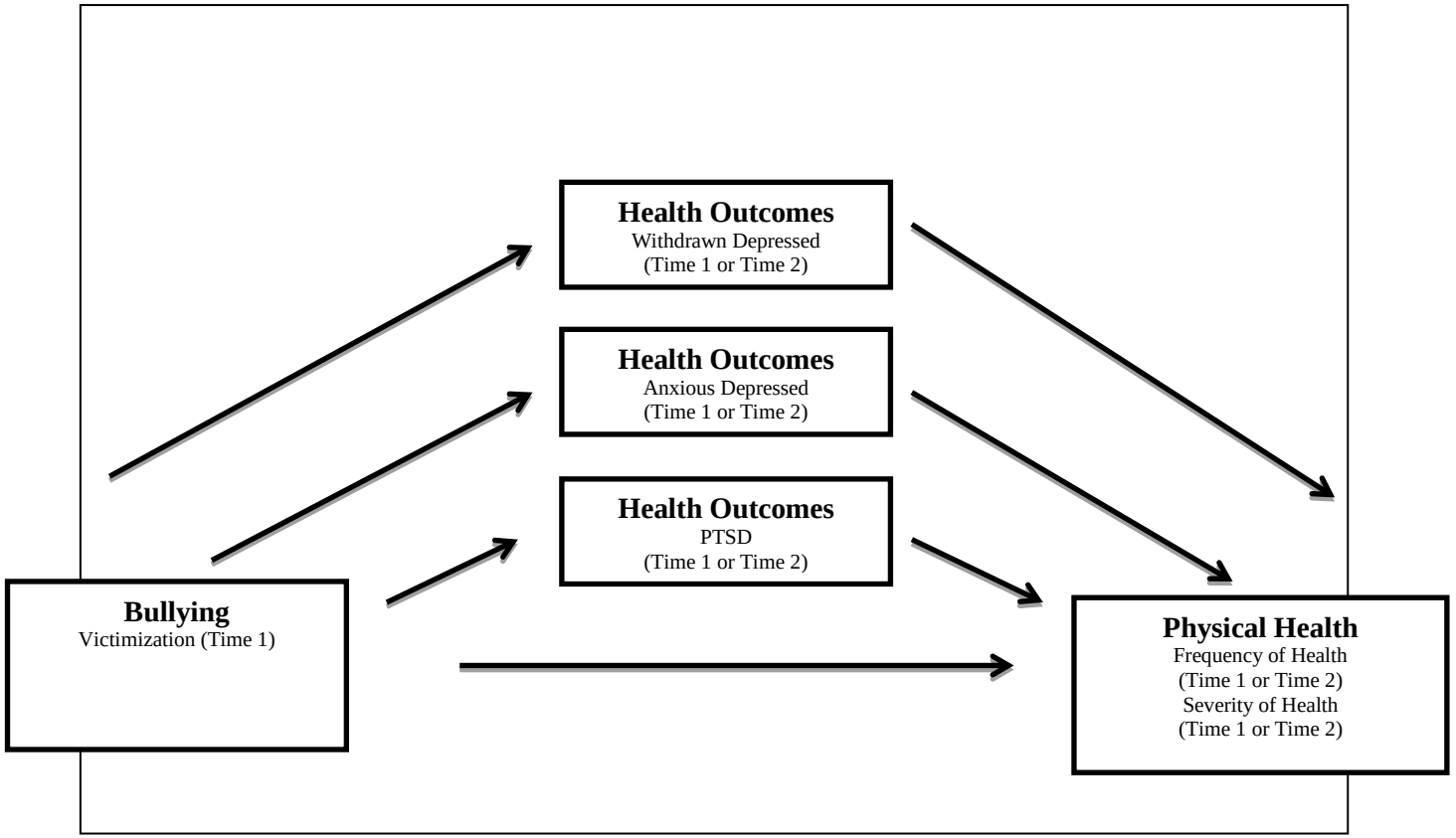

Figure 15 Mediation model between peer victimization, frequency or severity of health, psychological health outcomes at time 1 or time 2

Appendix C

Sample Questionnaires

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?

Health Outcomes

Please indicate the frequency and severity of the following physical symptoms.

1. Extreme fatigue
2. Allergic reaction
3. Sleep problems
4. Stomach ache
5. Nausea/vomiting
6. Diarrhea
7. Muscle aches and pains
8. Headaches or migraine
9. Weight change (gain or loss of 5 or more pounds)
10. Respiratory congestion
11. Runny nose
12. Coughing
13. Sore throat
14. Sneezing
15. Blocked nose
16. Fever or chills
17. Dizziness
18. Double or blurred vision
19. Trouble catching breath
20. Having a cold
21. Chest pains
22. Numbness or tingling
23. Low energy
24. Ear infections
25. Getting sick
26. Heart beating too quickly
27. Visits to the doctor
28. Visits to the school nurse

Abdominal Pain Index

1. How frequently over the past two weeks have you experienced abdominal pain?
2. In a typical day over the past two weeks, how frequently did you experience abdominal pain during the day?
3. When you experienced abdominal pain over the last two weeks, how long did it typically last?
4. When you experienced abdominal pain over the last two weeks, how intense was the pain typically?
5. When you experienced abdominal pain over the last two weeks, what was the maximum intensity of the pain?

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Priya A. Iyer was born in Mission Viejo, California and was raised in Northeast Indiana. She received her Bachelor of Arts degree in Behavioral Neuroscience Psychology from Purdue University in West Lafayette, IN. She discovered her immense love for research in personality and developmental psychology after developing her research skills under the guidance of Drs. William Graziano and Theordore Wachs at Purdue University.

Priya furthered her education at the University of Texas at Arlington in Arlington, TX by earning a Masters of Science degree in Experimental Psychology under the supervision of Dr. Lauri Jensen-Campbell in 2010. Her current research interest focuses on the influences of bullying on psychological and physical health outcomes. She is specifically interested in why some children may be more adversely affected by bullying than are other children.