INFLUENCE OF PEER VICTIMIZATION AND SOCIAL SUPPORT ON CORTISOL PRODUCTION

by

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ABSTRACT

INFLUENCE OF PEER VICTIMIZATION AND SOCIAL SUPPORT

ON CORTISOL PRODUCTION

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The current dissertation sought to examine whether there was an association between peer victimization, neuroendocrine functioning, and physical health outcomes. Adolescents and their parent (N = 107) participated in a two-part study. In the first phase, adolescents completed a series of questionnaires either at school or online to assess levels of peer victimization, social support, and health outcomes. They then came into the laboratory with a parent for the second part of the study which consisted of two sessions. In session one, adolescents completed additional questionnaires and learned how to collect saliva samples (to assess cortisol levels); parents completed measures of their child's social experiences (i.e., victimization and social support) and health. Adolescents collected 4 samples of their saliva over each of 2 non-sports school days (for a total of 8 daily samples). Upon returning to the laboratory, adolescents completed the Trier Social Stress Test in which they prepared and delivered a 5-minute speech on why they would make an ideal class president. Cortisol samples were collected before and after the speech. As expected, peer victimization predicted negative health outcomes. This link did not differ between boys and girls. Moreover, the results suggest that peer victimization alters the hypothalamic-pituitary-adrenal (HPA) axis (as assessed by cortisol levels) which in turn

predicts negative health outcomes. This dissertation was an important first step in understanding how peer victimization impacts biological functioning.

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CHAPTER 1

INTRODUCTION

Researchers have long argued that humans have a fundamental need to belong and to have ongoing relationship bonds (Bowlby, 1973; Baumeister & Leary, 1995; Maslow, 1968, Taylor, 2007; MacDonald & Leary, 2005). Maintaining social relationships and avoiding the possibility of damage to those relationships may have been vital to ancestral survival (MacDonald & Leary). Indeed, social exclusion from a group elicits feelings of distress, negative emotions, and threatened needs such as belongingness, control, meaningful existence, and self-esteem (e.g., Williams, 2001; Waldrip & Jensen-Campbell, 2007). Even recalling instances of broken relational bonds or social pain (e.g., social exclusion, being bullied or victimized, lose of a loved one, divorce) impairs subsequent self-regulation and elicits more negative emotions than recalling instances of physical pain, important material loss, or a typical Monday morning (Chen, Williams, Fitness, & Newton, 2008; Knack & Jensen-Campbell, 2007).

Although there are many ways in which relationships may be damaged, the focus of this dissertation is on one prevalent form that occurs in early adolescence, namely peer victimization, which involves being the habitual target of peers' aggression. It has been estimated that 10 - 30% of American children report being repeatedly victimized by their peers (Grills & Ollendick, 2002; Haynie et al., 2001; Limber & Small, 2003; Nansel et al., 2001; Perry et al., 1998). The number of victimized children is concerning, especially when the long-term effects of being repeatedly victimized are considered. Chronic peer victimization peaks in early adolescence and is associated with numerous psychological problems including internalizing (e.g., anxiety, depression) and externalizing (e.g., aggression, rule-breaking behaviors) problems, low self-esteem, higher depression, and greater loneliness (Kupersmidt, Coie, &

Dodge, 1990; Parker & Asher, 1987; Crick & Grotpeter, 1995, Egan & Perry, 1998; Hodges & Perry, 1996; Olweus, 1978). It is striking that chronic peer victimization is also associated with physical health outcomes. For example, victimized children report more headaches, stomach pain, sore throats, colds, and mouth sores than nonvictimized children (Rigby, 1998; Williams, Chambers, Logan, & Robinson, 1996; Wolke, Woods, Bloomfield, & Karstadt, 2001).

These differences in physical and psychological health between victimized and nonvictimized children are long lasting. Rigby and Slee (1999) found that three years after an initial assessment, victimized children continued to have poorer health outcomes than their nonvictimized peers even after controlling for initial differences in health. Much evidence has demonstrated that early peer relationships continue to influence psychological behavior into adulthood. For example, higher levels of peer rejection during preadolescence predicted poorer overall adjustment later in life than lower levels of peer rejection (Bagwell, Newcomb, & Bukowski, 1998). Individual differences in who is victimized usually stabilize by late elementary school (Perry, Hodges, & Egan, 2001; Hodges & Perry, 1996) so that by the end of elementary school the same children tend to be victimized each year. Indeed, Pedersen, Vitaro, Barker, and Borge (2007) found that early rejection was a strong predictor for later rejection. Thus, these children who are chronically victimized are likely to be at greater risk for both immediate and later problems.

While there are an abundance of studies examining the psychological risks associated with being victimized, there is a shortage of studies examining the biological ramifications of being victimized. As such, this dissertation examined how peer victimization in adolescence influences neuroendocrine responses in the hypothalamic-pituitary-adrenal (HPA) axis and how the HPA axis is associated with both physical and psychological health outcomes. Since cortisol is the end product of HPA activation, cortisol levels were measured and served as an index for HPA activation. It was anticipated that adolescents who are chronically victimized will evidence differential HPA activation than adolescents who are not chronically victimized. It was expected

that greater HPA activation would be linked with increased health problems (e.g., more illnesses, school absences) and more psychological problems (e.g., adjustment problems). Furthermore, this study sought to identify potential moderators in the chronic victimization-health link. It was anticipated that social support from friends and parents would be particularly important if the adolescent was victimized. That is, support should act as a buffer against the negative influences of victimization.

1.1 Understanding Social Pain Processes by Examining Physical Pain Processes

An important question involves *why* peer victimization should not only influence psychological outcomes, but also biological and health outcomes. One possibility is that there is an interplay between social and biological functioning. Understanding this overlap may allow for the development of more thorough theoretical models and thus enhance our understanding of social processes. Recent research has begun to examine the similarities and differences between physical pain and social pain. Physical pain is commonly defined as an injury to the physical body (Craig, 1999) whereas social pain is commonly defined as an injury (or perceived injury) to one's social relationships (Eisenberger & Lieberman, 2004; MacDonald & Leary, 2005). The pain associated with social loss, rejection, exclusion, victimization, and ostracism has collectively been termed social pain. As noted above, there is evidence of a link between social pain (e.g., peer victimization) and physical pain (e.g., abdominal pain, headaches, sore throats, and colds). However, researchers have just recently begun to consider the similarities and differences between the two pain types. It is likely that the extensive literature on physical pain can help inform us about social pain processes.

Physical pain is typically defined in terms of two components: (1) the sensory-discriminative component (i.e., pain sensation), which consists of information such as the location, duration, and intensity of the painful stimulus and (2) the affective-motivational component (i.e., pain affect), which consists of information such as emotions caused by the painful stimulus or the unpleasantness associated with the painful stimulus (Craig, 1999; Price,

2000). Physical pain is believed to be comprised of both the sensory-discriminative component and the affective-motivational component of pain whereas social pain is believed to be comprised solely of the affective-motivational component (Craig, 1999; Price, 2000; MacDonald & Leary, 2005).

Evidence suggests that the neural substrates associated with the affective experience of social pain may overlap with those of physical pain (MacDonald & Leary, 2005; Eisenberger & Lieberman, 2005). Eisenberger, Lieberman, and Williams (2003) found that being socially excluded from an on-line ball tossing game differentially activated the anterior cingulate cortex (ACC) compared to periods of being included in the game. Notably, increased ACC activation was significantly correlated with increased self-reported personal distress. The ACC has previously been dubbed as the "neural alarm system," which sounds at the detection that something has gone wrong and has repeatedly been found to be activated during experiences of physical pain. Thus, Eisenberger et al.'s work provides early evidence that instances of exclusion compared with inclusion elicit not only heightened reports of distress but increased ACC activation. Jensen-Campbell, Knack, and colleagues (in preparation) also used the on-line ball tossing paradigm to compare persons who were high and low on need to belong and found that persons higher on need to belong evidenced greater insula activity during exclusion periods than during inclusion periods. Insula activity has previously been found during experiences of physical pain; finding similar activation during episodes of social pain adds to the growing literature suggesting an overlap between neural substrates of physical and social pain. Furthermore, Eisenberger et al. (2006) found sensitivity to physical pain predicted sensitivity to social pain (i.e., being excluded in the ball tossing game). In addition, they found evidence that increased reports of social distress were associated with increased reports of pain unpleasantness. MacDonald, Kingsbury, and Shaw (2005) also reviewed a series of studies demonstrating corresponding pain sensitivity between social and physical pain. That is, when persons are highly sensitive to one pain type they are also sensitive to the other pain type.

Furthermore, there is evidence that social pain experiences (e.g., maltreatment in childhood) may alter biological processes (e.g., Gunnar, Morison, Chisholm, & Schuder, 2001). These findings provide further evidence of a possible overlap between the neural substrates of physical pain and social pain.

The overlap between physical and social pain systems is theoretically relevant to the current dissertation because it highlights the important relationship between social interactions and psychological and biological functioning. It also suggests that the literature pertaining to physical pain can help further understanding about the underlying mechanisms occurring during instances of social pain (e.g., victimization). Thus, it is important to consider the extensive literature on physical pain. Early theories of physical pain attributed pain-related symptoms to a specific disease state (Turk & Monarch, 2002). These biomedical models failed to take into account the various psychological and affective components of pain. The Gate-Control Theory (GCT) was an attempt to include psychological and affective components and is still widely accepted today (Melzack & Wall, 1965; Turk & Monarch). The GCT also identified the central nervous system as being an important aspect in pain processes and pain perception. More recently, Melzack (1999, 2005) proposed a neuromatrix theory of pain in which genetic components as well as the neural and hormonal components of the stress response play a pivotal role in understanding pain. Evidence of a connection between stress and physical pain can be traced back to researchers such as Selye (1950) who noted that pain caused a disruption in homeostasis. This disruption is stressful and activates a network of neural, hormonal, and behavioral mechanisms in an attempt to return to a homeostatic state.

1.2 Effects of Stress

Stress and pain have an interconnected and often bidirectional relationship. As noted above, pain itself is often experienced as a stressor. On the other hand, chronic stress can lead to experiences of various pains. Before further examining the relationship between pain and stress it is important to consider the difficulty in defining stress. In some cases, stress is

considered the actual stimulus that causes a reaction whereas in other cases stress is the response that occurs due to a particular stimulus. Typically, events that are threatening or harmful are considered to be stressful. In addition, stress is typically associated with negative or unpleasant feelings (Dougall & Baum, 2001). Most researchers agree that stress has a direct effect on a number of physiological systems including the sympathetic and parasympathetic nervous system, endocrine system, and hypothalamic-pituitary-adrenal (HPA) axis (e.g., Cannon, 1929; Selye, 1956; Mason, 1971; Carver, 2007; Dougall & Baum).

Indeed, the body is equipped to react to stress by activating a number of different systems in an attempt to adapt and return the body to a homeostatic state. A number of factors contribute to how successfully an individual will handle a particular stress (McEwen, 1998). Two of the most common factors include how the person perceives the situation (i.e., is the situation perceived as stressful?) and the person's overall health state. When a stressor is detected, the sympathetic nervous system (SNS) and HPA axis are activated. Typically, these systems are shut off after the stress is reduced (McEwen, 1998). However, when the stress is chronic or repeated the SNS and HPA systems do not shut off which leads to overexposure of stress hormones (e.g., glucocorticoids).

There is a great deal of research illustrating the link between stress and health problems (see Carver, 2007). For example, chronic stress has been linked with decreased immune functioning, specifically a decreased number of antibodies produced in response to a vaccination (Glaser, Sheridan, Malarkey, MacCallum, & Kiecolt-Glaswer, 2000; Miller, Cohen, Pressman, Barkin, Rabin, & Treanor, 2004). Miller, Cohen, and Ritchey (2002) found chronic stress experienced by cancer patients also decreased immune functioning by impairing the body's anti-inflammatory signals. Increased blood pressure is also associated with work-related stress (i.e., high status positions, high psychological demand) as well as merely recalling work-related stress (Carels, Sherwood, & Blumenthal, 1998) and marital disagreements (Smith, Gallo, Goble, Ngu, & Stark, 1998). Kiecolt-Glaswer, Newton, Cacioppo, MacCallum, Glaser, and

Malarkey (1996) found that marital conflict was related to neuroendocrine functioning in wives (but not husbands). Specifically, they found that wives evidenced higher cortisol levels when there was a high probability that their husband would withdraw after a negative interaction. Furthermore, McBeth et al. (2005) suggested that an impaired HPA axis response may be the underlying mechanism in predicting the onset of chronic widespread pain in persons experiencing psychological distress.

1.3 Stress and the Neuroendocrine System

It is well accepted that physical and social stress engages the HPA axis (Dickerson & Kemeny, 2004; Eisenberger et al., 2007; Purvis & Cross, 2006). The activation of the HPA axis consists of the hypothalamus signaling the release of corticotrophin releasing hormone (CRH) which in turn activates the anterior pituitary gland and stimulates the adrenal cells to release adrenocorticotropic hormone (ACTH) into the bloodstream. ACTH travels to the adrenal glands to release cortisol (McEwen et al., 1997). Cortisol, one of the end products of HPA activation, can be beneficial and protective in response to acute stressors. Indeed, the production of cortisol helps engage the body to deal effectively with the stressor at hand. However, chronic exposure to high levels of cortisol can be maladaptive and is associated with degeneration in hippocampal dendrites (Sapolsky, Uno, Rebert, & Finch, 1990; Uno et al., 1989; Sapolsky et al., 1990), disrupted learning and memory (McEwen, 2000), and decreased immune functioning (Miller, Cohen, & Ritchey, 2002).

Little work has examined how peer victimization influences HPA axis activation. However, looking at research which examined the link between similar relationship stressors (e.g., maltreatment in childhood or posttraumatic stress disorder) and HPA axis activity may shed light on how chronic peer victimization might influence HPA activation. Being maltreated in childhood has long term effects on the HPA axis response to stress (Gillespie & Nemeroff, 2007; Tarullo & Gunnar, 2006). For example, Hart, Gunnar, and Cicchetti (1995) found that maltreated preschoolers had lower cortisol reactivity on high social conflict days at school as

compared to low social conflict days than preschoolers who were not maltreated. More recent research has begun to examine potential moderators between maltreatment and cortisol reactivity. For example, maltreated children with internalizing and externalizing problems have differential daily salivary cortisol levels than peers without internalizing and externalizing problems or who have not been maltreated (Cicchetti & Rogosch, 2001). Children with internalizing problems evidenced hypercortisolism whereas children with externalizing problems evidenced hypocortisolism. Even less severe types of maltreatment appear to influence HPA functioning, as evidenced by alterations in cortisol levels. Bugental, Martorell, and Barraza (2003) found that infants with emotionally unavailable mothers have elevated baseline cortisol levels whereas infants whose mothers frequently use physical discipline such as spanking were more hyperreactive in the strange situation paradigm.

Patients experiencing posttraumatic stress disorders (PTSD), by definition, have experienced a significant amount of stress. Again, such a population experiencing chronic stress can inform the current work. Rohleder, Joksimovic, Wolf, & Kirschbaum (2003) found that persons with PTSD did not evidence the typical cortisol awakening response. Rather than the typical peak 30 minutes after waking, there was no significant change in cortisol levels during the first 30 minutes after waking in persons with PTSD (control participants did evidence the expected cortisol awakening response). In addition, persons with PTSD had lower overall cortisol levels and a flattened pattern over the course of the day compared with control participants not experiencing PTSD. Other research supports the link between traumatic experiences (e.g., chronic abuse, PTSD) and lower cortisol levels (e.g., Johnson, Delanhanty, & Pinna, 2008; Pervanidou, 2008).

While researchers agree that stress influences hormonal responses, it is still relatively unclear exactly how chronic versus acute social stress affects hormone production. Few studies have examined how peer victimization is associated with hormone production. Hansen et al. (2006) found that persons who were bullied by co-workers had lower morning cortisol levels,

less social support, and more internalizing problems (e.g., depression, anxiety). Vaillancourt et al. (2008) found that peer victimization in adolescence was linked with lower cortisol levels. Finally, Kliewer (2006) found that adolescents who witnessed high levels of violence had lower cortisol levels in the morning as well as during a lab stress test.

1.4 Social Stressors and HPA Reactivity

There is still discrepancy regarding how long-term maltreatment impacts cortisol reactivity. Acute social stressors have been repeatedly found to increase cortisol reactivity (Dickerson & Kemeny, 2004). In a meta-analysis of 208 studies, uncontrollable tasks that involved a social evaluative threat produced the largest cortisol change and had the longest recovery time. These tasks influenced the overall magnitude of cortisol responses as well as the recovery trajectory. The authors suggested that individuals are motivated to preserve self-esteem. Social evaluative tasks were found to be perceived as threatening. It is likely that elevated cortisol levels are evidence of a physiologically based attempt to reduce the threat.

One common laboratory social stressor, the Trier Social Stress Test (TSST), has repeatedly been found to activate the HPA axis (Kirschbaum, Pirke, & Hellhammer, 1993; Kudielka, Schmidt-Reinwald, Hellhammer, & Kirschbaum, 1999). The TSST is typically comprised of two tasks. In the first task, participants are instructed to give a convincing five minute speech (e.g., for a job application, to be class president) to a committee of two to three persons who pose as experts. Participants are given a 10 minute preparation period during which they can create written notes and prepare for their upcoming speech. However, these notes are not able to be used during the actual speech delivery. If participants stop speaking before the five minutes allocated for the speech delivery are over, the committee probes the participant to continue and asks follow-up questions. Immediately after the speech delivery, the participant is told he/she will be completing a calculation task in which he/she will count backwards by 13 from a large number (e.g., 1379). However, if any mistakes are made, they will be stopped and asked to start over; this portion of the TSST lasts about five minutes. Before

and after the TSST, salivary cortisol samples are collected. This task serves as an acute period of social stress and reliably has led to increased levels of salivary cortisol.

Indeed, these acute periods of social stress led to increased levels of salivary cortisol. The cortisol response from social stressors peaks roughly 20 to 30 minutes after the onset of the stressor (Smyth et al., 1998). Although baseline levels are elevated, maltreated children evidenced lower cortisol reactivity than comparison children on similar acute social stressor tasks (Hart, Gunnar, & Cicchetti, 1996; Cicchetti & Rogosch, 2001). Adults maltreated during childhood currently presenting with psychiatric diagnoses showed greater ACTH responses but normal cortisol levels after a task similar to the TSST (Tarullo & Gunnar, 2006). Unfortunately, studies examining hormonal responses to maltreatment do not commonly examine reactivity to acute stressors but instead focus on basal or baseline cortisol levels. However, the limited research available suggests that chronic stress such as maltreatment or peer victimization leads to altered HPA axis activation to acute stressors as evidenced by lower cortisol reactivity (Hart, Gunnar, & Cicchetti; Cicchetti & Rogosch; Tarullo & Gunnar; Bugental, Martorell, & Barraza, 2003).

1.5 The Importance of Social Support in Stressful Situations

As mentioned above, it has been argued that humans have an innate need to belong (e.g., Bowlby, 1973; Baumeister & Leary, 1995). According to Baumeister and Leary, individuals are driven to form and maintain positive, significant, lasting relationships. As such, persons should be driven to engage in frequent and positive interactions. It has been suggested that social relationships are so vital that threats to relationships (e.g., exclusion, rejection, victimization) may be processed as a basic survival threat (MacDonald & Shaw, 2005; MacDonald & Leary, 2005).

Indeed, Taylor's (2002, 2007) tend-and-befriend hypothesis of stress posits that rather than activating a fight-or-flight responses, stress situations activate an affiliative system. Once signaled, the need for social contact is met through interactions with others. Social support, in

turn, reduces biological and psychological stress responses (e.g., cortisol). Conversely, being in contact with nonsupportive others or being alone should exacerbate these stress responses (Taylor et al., 2000). Individuals who interact with supportive others should therefore evidence diminished cortisol responses in stressful situations. Eisenberger et al. (2007) found a negative association between perceived social support and ACC activity such that greater perceived social support was associated with decreased ACC activation when the individual was being excluded.

There are two main theories regarding when the beneficial effects of social support are experienced, namely the stress-buffering hypothesis and the main effects hypothesis (Cohen & Wills, 1985). According to the stress-buffering hypothesis, an individual's social network equips the individual with the needed resources to cope with stressful events and situations. Thus, the benefits of social support are only evidenced during stressful periods. The main effects hypothesis, on the other hand, postulates that social support is beneficial regardless of whether a stressful situation is being experienced. This theory of social support postulates that the more an individual participates in the social network the more benefits will be experienced. The stress-buffering hypothesis has received the most research attention and is heavily supported. The current dissertation takes a buffering approach by examining how perceived support buffers against the effects of an acute social stress.

Having the support of another can facilitate persistence in tasks, increase self-regulatory ability, and increase pain tolerance. For example, task persistence is easier when a partner is present, presumably because the partner can help divert attention from distractions (Mischel, Cantor, & Feldman, 1996). In other words, the presence of another can increase persons' self-control. In addition, the presence of others can increase an individual's tolerance to intense electrical shock (Amoroso & Walters, 1969; Buck & Parke, 1972). On the other hand, individuals who are rejected by others are more likely to have impaired self-regulatory ability

(Baumeister & Twenge, 2003; Knack & Jensen-Campbell, 2006). These studies illustrate that the mere presence of a supportive person can be beneficial.

Likewise, the mere perception that a person is supportive can be nearly as beneficial as actually receiving support (Cohen, Lakey, Tiell, & Neely, 2005). Feldman, Downey, and Schaffer-Neitz (1999) found that perceived support was related to a reduction in self-reported pain and negative affect. On the other hand, a decrease in perceived social support predicted increases in eating disorders (Jackson, Weiss, Lunquist, & Soderlind, 2005). In other words, perceiving social support even in the absence of actual support can still have buffering effects (Knack, Waldrip, & Jensen-Campbell, 2007). Conversely, having support (but not perceiving it as support) can be detrimental.

Social support also appears to influence cortisol responses. Kirschbaum, Klauer, Filipp, and Hellhammer (1995) found that men who were anticipating giving a public talk had lower cortisol levels when their girlfriend provided support as compared to support offered by a stranger or when their girlfriends were not supportive. Likewise, cortisol levels were attenuated by the social support of a best friend while preparing for giving a public speech (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). As reviewed by Kirschbaum and Hellhammer (1999) and DeVries, Glasper, and Detillion (2003), social support appears to have a significant impact on the HPA axis and in turn is likely to influence health outcomes and serve as a buffer against stress-related health problems (e.g., anxiety, high blood pressure, immune deficiency). Blair, Granger, Willoughby, Kivlighan, and the Family Life Project Investigators (2006) found that infants' cortisol reactivity was related to the degree of mother's sensitivity. When mothers were highly sensitive to infant's needs, infants evidenced lower baseline cortisol and an expected increase in cortisol levels when faced with a challenge (e.g., arm restraint); however, when mothers were not very sensitive to the infant's needs, higher baseline cortisol levels and low cortisol reactivity was observed (Bugental, Martorell, & Barraza, 2003). In other words, even as early as infancy, persons are influenced by the supportiveness of others.

1.6 Friendships as Social Support in Adolescence

Friendships are a common source of social support in adolescence. Prior to reaching adolescence, children tend to seek out parents for companionship and intimacy needs (Furman & Buhrmester, 1985). However, as children make the transition into adolescence, peers play an increasingly important role (Harris, 1995). Adolescents begin to turn more frequently to peers for companionship and support in addition to seeking out their parents (Furman & Buhrmester; Sullivan, 1953; Hartup, 1983). Furman and Buhrmester (1992) found that children in 4th grade rated their parents as being most supportive whereas adolescents in 7th grade rated their peers as being equally supportive as their parents. By the 10th grade, peers were rated as providing more support than parents. While peers begin to play an increasingly important role as children transition into adolescence, the parental role does not become any less important. For example, Liu (2006) found that adolescents' current attachments to their parents influenced the adolescents' expectations of peer interactions and perceived peer social support.

As friends begin to play an increasingly central role in each others' lives, these relationships may also begin to be an important buffer against the negative consequences of being victimized. Hodges, Boivin, Vitaro, and Bukowski (1999) found that simply having a best friend predicted a decrease in victimization throughout the school year. Furthermore, adolescents with a best friend did not evidence an increase in internalizing nor externalizing problems whereas adolescents without a best friend developed more internalizing and externalizing problems over the course of a school year (Hodges, Boivin, Vitaro, & Bukowski; Hodges, Malone, & Perry, 1997). Hodges, Malone, and Perry (1997) also examined the link between victimization and behavioral risk (e.g., internalizing problems, externalizing problems, physical strength) among children without any reciprocal friendships (n = 66) and children with at least one reciprocal friendship (n = 163). They found that children without at least one friend had significantly more internalizing and externalizing problems than peers with one or more friends. Finally, Laursen, Bukowski, Aunola, and Nurmi (2007) demonstrated that the majority of

developmental outcomes associated with peer rejection are specific to those children and adolescents without friends. That is, they found that a reciprocated friendship moderated the link between peer rejection and adjustment problems such as internalizing and externalizing problems. That is, persons without any reciprocal friends are significantly worse off than persons with at least one friend.

However, there is variation in the ability of others to provide support (Bukowski, Hoza, & Boivin, 1994; Hodges & Perry, 1996). Merely having friends does not necessarily buffer against the negative outcomes of peer victimization. Rather, it is important to consider the quality of the friend. For example, Waldrip, Malcolm, and Jensen-Campbell (2008) found that friendship quality was especially important for adolescent adjustment when the number of friends and general peer acceptance was low. In addition, Knack, Jacquot, Jensen-Campbell, and Malcolm (under revision) found that children who are low on agreeableness can experience the benefits of having a friend who is high on agreeableness (i.e., being kind, cooperative, supportive). That is, when the friend was high on agreeableness the target child evidenced fewer externalizing problems and experienced less overt and relational victimization. Likewise, Jensen-Campbell and Knack (in preparation) found that having a highly agreeable friend is negatively related to a target child's reports of loneliness at camp. Furthermore, a close friend's social competency (i.e., ability to initiate relationships, provide emotional support, assert influence, resolve conflict, and use of relationally aggressive tactics) was negatively associated with the target child being overtly victimized at camp (Knack, Rex-Lear, Bryant, Gomez, & Jensen-Campbell, 2007). Thus, having high quality friends who provide support can buffer against negative outcomes.

On the other hand, having low quality friends can exacerbate negative outcomes. For example, spending time with deviant peers is associated with increased deviant and antisocial behavior (e.g., Dembo & Schmeidler, 2003; van Lier, Vitaro, Wanner, Vuijk, & Crijnen, 2005). Adolescents' aggression levels can also be influenced by the quality of friends (Adams, Bukowski, & Bagwell, 2005). When an aggressive adolescent has highly aggressive friends, the

target adolescent maintains high aggression levels. However, when an aggressive adolescent has friends low on aggression, the target adolescent's aggressive behavior decreases over time. Therefore, not only is it important to determine whether adolescents have friends but also to consider who their friends are. In summary, although victimization is associated with serious psychological and physical outcomes, having a strong social support network can be beneficial and may even act as a buffer against the negative outcomes associated with victimization. As such, I expected that perceived support from a best friend and/or a parent would buffer against victimization outcomes.

1.7 Importance of Studying Adolescents

Social stressors, such as peer victimization, may be especially influential during periods of rapid developmental change. Adolescence is one such period when individuals go through extensive changes. In fact, Larson and Richards (1994) note that the transition from childhood to adolescence is one of the most complex life periods. It is no wonder that adolescence is a high-risk period for the onset of psychopathology, especially depression (Walker, 2002). During this period, persons undergo a number of physiological changes including puberty, alterations in HPA axis activation, an increase in stress hormones levels, and rapid brain maturation (Walker; Spear, 2000, Walker, Walder, & Reynolds, 2001). According to Spear, many of the basal changes in cortisol are influential in stress and emotional reactivity, including the prefrontal cortex and the limbic region. Walker, Walder, and Reynolds (2001) note that during adolescence there is an increase in HPA axis activation.

In addition to physiological changes, adolescents undergo a number of environmental changes. For example, peers begin to play an increasingly important socialization role (Hartup, 1996; Spear, 2000). Adolescence may be a time of increased vulnerability to stressful events which ultimately leads to a change in how stress is experienced (Walker, Walder, & Reynolds, 2001). Thus, it is during this period that persons may be most vulnerable to the influence of social experiences (i.e., more sensitive to rejection, social slights, and health problems).

1.8 Limitations of Previous Studies

While a great deal of research in developmental psychology has examined the behavioral and psychological outcomes related with chronic peer victimization, there is a shortage of studies that look at physiological outcomes. Likewise, there is a plethora of work studying the complex physiological reaction to stress; however these studies fail to consider important individual differences and relationship processes that might be influencing the stress response. For example, while the psychological importance of perceiving peer social support is well accepted, little is known about how peer social support influences the HPA axis during periods of stress. Furthermore, the majority of studies that do examine friendship processes focus on positive relationship features but fail to assess negative relationship features. This failure to include negative relationship dimensions is a deficit in the field. Positive and negative relationship dimensions are only weakly correlated, thus it is possible that important information could be gained by including the assessment of negative relationship qualities (Berndt, 2002; Furman & Buhrmester, 1985). The current study attempted to bridge these gaps in the literature to more fully understand how peer victimization influences the stress response (as assessed by cortisol levels) and the role that psychological processes play.

1.9 Current Study

Research is needed to begin to understand better *why* the link between victimization and poor health persists into adulthood. Whereas previous research has focused primarily on behavioral and psychological outcomes of peer victimization, the current work examined the physiological responses to peer victimization. Specifically, this dissertation examined whether HPA activation as assessed through cortisol levels may be an underlying mechanism in the long-term effects of peer victimization. It was expected that peer victimization would influence neuroendocrine responses which would in turn influence health outcomes. Furthermore, perceived social support was expected to buffer against the negative effects of victimization. See Figure 1.1 for the theoretical model.

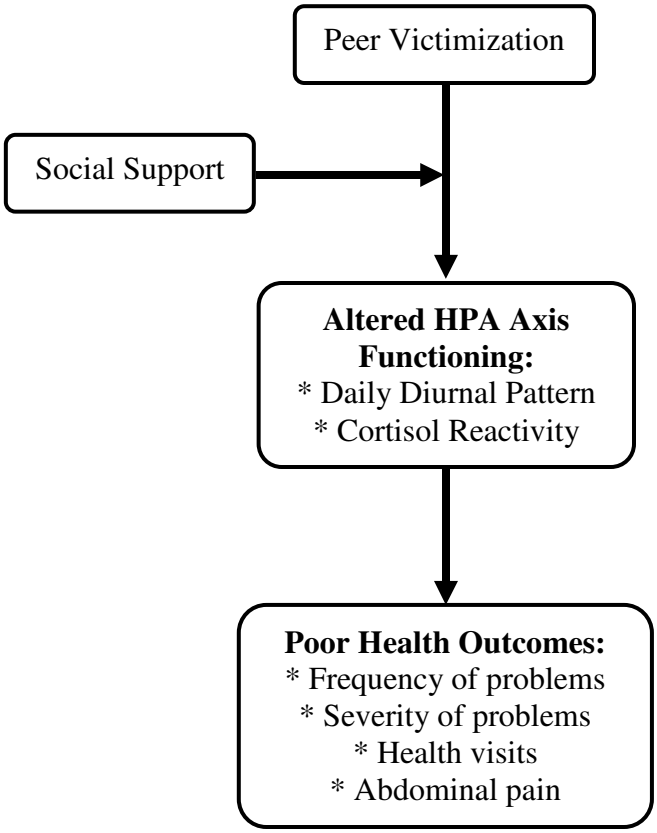


Figure 1.1 Theoretical Model

First, it was expected that peer victimization would predict health outcomes. Specifically, adolescents who were victimized were expected to report more frequent health problems, more severe health problems, more frequent visits to the doctor/nurse, and more abdominal pain than adolescents who were not chronically victimized. I also examined whether internalizing versus externalizing problems mediated the victimization-health link.

Second, possible moderators between peer victimization and health outcomes were also examined. It was expected that relationship processes would moderate the link between peer victimization and health outcomes. It was anticipated that perceived positive social support from close friends or parents would have a buffering effect against the negative health outcomes associated with peer victimization. On the other hand, perceived negative social support from close friends or parents was expected to exacerbate the link between victimization

and poor health. No relationship buffering effects from friends and parents were expected for adolescents who have not been chronically victimized.

Third, it was expected that being peer victimized would influence cortisol levels. The current dissertation examined both diurnal cortisol patterns and cortisol reactivity as an index of HPA axis activation. I first examined the link between victimization and cortisol production. I examined both diurnal patterns of cortisol production as well as cortisol reactivity during an acute social stressor. I expected that victimized adolescents would evidence lower daily patterns of cortisol than nonvictimized adolescents. In addition, I anticipated victimized adolescents would have a different cortisol reaction to the acute social stressor. Next, I predicted that these differences in cortisol would predict poorer health outcomes for victimized adolescents compared to nonvictimized adolescents. Finally, I expected that both daily cortisol levels and cortisol reactivity would mediate the victimization-health link.

CHAPTER 2

METHOD

2.1 Participants

Adolescents in 5th to 8th grade and their parent participated in a two-part research study (N = 107). Participants were adolescents in 5th – 8th grades from schools in the Dallas/Fort Worth area who ranged in age from 10 years to 14 years (*M* = 12.23, *SD* = 1.09). Both girls (56.1%) and boys (43.9%) participated in this project. The racial composition included 66.4% White, 15.9% Hispanic/Latino, 11.2% Black/African American, 3.7% Asian, 1.9% American Indian/Alaskan Native, and 0.9% Hawaiian/Pacific Islander participants. Five students did not complete the speech portion of the TSST because they were visibly distressed (e.g., crying) over the idea of giving the speech and said that they could not proceed (i.e., they prepared at least part of the speech but did not deliver it). However, cortisol was still collected from these participants at the target times. Participants also completed the same scale measures at the appropriate time points. They were included in the analyses but excluding them from the analyses did not change the overall pattern of findings.

Several methods were used for recruiting participants. First, persons were randomly selected from a mailing list obtained from Arlington Independent School District (AISD) to be contacted for possible participation in a study on friendship, peer relationships, and health. Information about the project was mailed home to these selected persons along with instructions for how to enroll in the study. Second, I went into several private Catholic schools, a Protestant school, and one public school (i.e., Bailey Junior High) in the area to talk with adolescents in large groups (i.e., either in their homeroom period or at the end of their lunch period) about the project. Third, I obtained the mailing list from the local Big Brothers/Big Sisters

Organization and mailed out project information. Fourth, I recruited adolescents from a local dance and cheer company as well as several summer camps (e.g., cello camp, theater camp, math camp).

I attempted to target adolescents with meaningful histories of chronic peer victimization (i.e., adolescents at the extreme ends of the victimization continuum). For example, during the early phases of data collection, I invited adolescents who scored at least one standard deviation above the mean for victimization to participate in the study (i.e., based on their scores on the victimization measures from phase one). In addition, some parents with children who were victimized contacted me to be included in the study; other children were recommended for inclusion by persons who knew their social experiences well. Adolescents were paid $5.00 and entered into a raffle for an iPOD shuffle for participating in Phase I. Adolescents and parents were each paid a total of $40.00 for participating in two sessions in Phase II (i.e., $20 for each session).

2.2 Assessment of Victimization

2.2.1. Children's Self-Experiences Questionnaire – Self- and Parent-Report (CSEQ-SR, CSEQ-PR)

The CSEQ-SR/CSEQ-PR assessed peer-related instances of victimization (Crick & Grotpeter, 1995). This questionnaire consisted of three subscales including overt victimization (e.g., "I get hit and pushed by other kids"), relational victimization (e.g., "I am ignored by other classmates when someone is mad at me"), and being the recipient of prosocial behavior/general social support (e.g., ""How often does another kid give you help when you need it?"). Each subscale was comprised of five items which measured the frequency children experienced a particular event on a scale of 1 (never) to 5 (all the time). Both parents and adolescents completed this questionnaire. High reliability was found for both self and parental reports of overt (α = 0.81, 0.84), relational (α = 0.84, 0.86), and prosocial behavior/general support (α = 0.83, 0.79), respectively. See Table 2.1 for descriptive statistics of self-reported victimization, Table 2.2 for descriptive statistics of parent-reports of victimization, and Table 2.3

for descriptive statistics of z-scored reports of victimization (averaged across self- and parent-reports).

Table 2.1. Descriptive Statistics for Self-Reports of Victimization

Measure	Range	Min	Max	Mean	SD	Skewness (Std. Error)	Kurtosis (Std. Error)	α
Prosocial	18	7	25	17.83	4.03	-0.52 (0.23)	-0.16 (0.46)	0.83
Overt	14	5	19	8.32	3.49	1.10 (0.24)	0.58 (0.46)	0.81
Relational	16	5	21	9.90	4.00	0.56 (0.23)	-0.51 (0.46)	0.84
Physical	21	7	28	11.65	4.82	1.38 (0.23)	1.59 (0.46)	0.85
Verbal	18	5	23	11.39	4.88	0.54 (0.23)	-0.67 (0.46)	0.86
Indirect	40	12	52	24.35	8.94	0.80 (0.23)	0.22 (0.46)	0.89

2.2.2. Direct and Indirect Aggression Scales – Victim

The DIAS (Bjorkqvist, Lagerspetz, & Osterman, 1992) is a 24-item measure which measures how frequently aggression/victimization is experienced. Specifically, the DIAS consists of three subscales that assess 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 questions were answered along a Likert-type scale ranging from 1 (never) to 5 (very often). Both parents and adolescents completed this questionnaire. High reliability was found for both self and parental reports of physical (α = 0.85, 0.89), verbal (α = 0.86, 0.90), and indirect (α = 0.89, 0.91) aggression/victimization, respectively. See Table 2.1 for descriptive statistics of self-reported victimization, Table 2.2 for descriptive statistics of parent-reports of victimization, and Table 2.3 for descriptive statistics of z-scored reports of victimization (averaged across self- and parent-reports).

Table 2.2 Descriptive Statistics for Parent-Reports of Victimization

Measure	Range	Min	Max	Mean	SD	Skew	Kurtosis	α
Overt	10	5	15	7.47	2.59	1.14 (0.23)	0.70 (0.46)	0.84
Relate	16	5	21	10.04	3.31	0.37 (0.23)	-0.12 (0.46)	0.86
Physical	20.77	6.23	27	10.00	3.76	1.57 (0.23)	3.03 (0.46)	0.89
Verbal	15	5	21	10.14	3.57	0.85 (0.23)	0.41 (0.46)	0.90
Indirect	31	12	43	23.91	7.25	0.37 (0.23)	-0.68 (0.46)	0.91

Table 2.3 Descriptive Statistics for Measures of Victimization after being z-Transformed (Collapsed Across Parent and Child Reports)

Measure	Range	Min	Max	Mean	SD	Skew	Kurtosis
Overt	3.21	-0.95	2.26	0.00	0.86	1.00 (0.23)	0.21 (0.46)
Relational	3.66	-1.37	2.29	0.00	0.84	0.26 (0.23)	-0.58 (0.46)
Physical	3.80	-0.88	2.92	0.00	0.86	1.36 (0.23)	1.43 (0.46)
Verbal	3.74	-1.37	2.36	0.00	0.87	0.71 (0.23)	-0.01 (0.46)
Indirect	3.89	-1.51	2.38	0.00	0.88	0.43 (0.23)	-.0.51 (0.46)
Total	3.11	-1.18	1.94	0.00	0.75	0.62 (0.23)	-0.41 (0.46)

Note: Numbers in parentheses are the standard errors.

2.3 Assessment of Perceived Social Support

2.3.1. Network of Relationships Inventory (NRI)

The NRI consisted of 30 items which assess individuals' perceptions of various relationship qualities and characteristics (Furman & Buhrmester, 1985). Specifically, perceptions of companionship, disclosure/intimacy, pressure, satisfaction, conflict, support, criticism, approval, dominance, and exclusion were assessed. There are three Likert-type items (1 = never or hardly at all; 5 = always or extremely much) for each of the above relationship

dimensions. The NRI was used to assess the target adolescent's perceived relationship with their mother, father, best friend, and second best friend. This measure served as an index of perceived support and specifically examined relationship quality by looking at various positive and negative relationship dimensions. High reliability was found for self-reports of companionship (α = 0.79, 0.90, 0.81, 0.79), disclosure (α = 0.87, 0.93, 0.93, 0.89), satisfaction (α = 0.93, 0.93, 0.85, 0.86), support (0.85, 0.87, 0.88, 0.90), approval (0.85, 0.87, 0.78, 0.80), pressure (α = 0.85, 0.87, 0.81, 0.83), conflict (α = 0.87, 0.90, 0.86, 0.80), and criticism (α = 0.79, 0.81, 0.87, 0.82) for mothers, fathers, best friend, and second best friend, respectively. Moderate to low reliabilities were found for dominance (α = 0.53, 0.65, 0.53, 0.54) and exclusion (α = 0.47, 0.53, 0.50, 0.43) for mothers, fathers, best friend, and second best friend, respectively. See Table 2.4 for descriptive statistics; see Table 2.5 for correlations between dimensions of support.

Table 2.4 Descriptive Statistics for the Network of Relationships Inventory

Measure	Range	Min	Max	Mean	SD	Skewness (Std. Error)	Kurtosis (Std. Error)	α
Companion								
BF	11	4	15	11.92	2.65	-0.67 (0.23)	-0.28 (0.46)	0.81
BF2	12	3	15	10.40	3.01	-0.45 (0.23)	-0.20 (0.46)	0.79
Mom	12	3	15	11.81	2.76	-0.84 (0.23)	0.45 (0.46)	0.79
Dad	12	3	15	10.35	3.76	-0.62 (0.23)	-0.73 (0.46)	0.90
Disclosure								
BF	12	3	15	10.16	3.98	-0.45 (0.23)	-1.04 (0.46)	0.93
BF2	12	3	15	9.04	3.85	-0.11 (0.23)	-1.21 (0.46)	0.89
Mom	12	3	15	10.12	3.64	-0.23 (0.23)	-1.09 (0.46)	0.87
Dad	12	3	15	7.88	3.99	0.36 (0.23)	-1.17 (0.46)	0.93

Table 2.4. – continued

Satisfaction								
BF	10	5	15	13.00	2.29	-1.08 (0.23)	0.56 (0.46)	0.85
BF2	12	3	15	12.44	2.65	-1.00 (0.23)	0.62 (0.46)	0.86
Mom	12	3	15	12.82	2.97	-1.54 (0.23)	1.83 (0.46)	0.93
Dad	12	3	15	11.81	3.78	-1.16 (0.23)	0.23 (0.46)	0.93
Support								
BF	12	3	15	10.62	3.76	-0.46 (0.23)	-0.99 (0.46)	0.88
BF2	12	3	15	9.52	3.98	-0.24 (0.23)	-1.34 (0.46)	0.90
Mom	12	3	15	11.77	3.23	-0.90 (0.23)	-0.04 (0.46)	0.85
Dad	12	3	15	9.62	3.96	-0.34 (0.23)	-1.15 (0.46)	0.87
Approval								
BF	12	3	15	10.59	2.98	-0.30 (0.23)	-0.45 (0.46)	0.78
BF2	12	3	15	10.00	3.33	-0.23 (0.23)	-0.86 (0.46)	0.80
Mom	12	3	15	12.32	2.81	-1.34 (0.23)	1.73 (0.46)	0.85
Dad	12	3	15	11.39	3.47	-0.95 (0.23)	0.15 (0.46)	0.87
Pressure								
BF	9	3	12	5.28	2.48	1.00 (0.23)	0.30 (0.46)	0.81
BF2	12	3	15	5.28	2.70	1.31 (0.23)	1.32 (0.46)	0.83
Mom	11	3	14	6.14	3.29	0.69 (0.23)	-0.74 (0.46)	0.85
Dad	12	3	15	5.62	3.29	1.26 (0.23)	0.81 (0.46)	0.87
Conflict								
BF	11	3	14	6.43	2.83	0.75 (0.23)	-0.34 (0.46)	0.86
BF2	11	3	14	5.84	2.66	1.01 (0.23)	0.41 (0.46)	0.80
Mom	12	3	15	7.92	3.17	0.34 (0.23)	-0.39 (0.46)	0.87
Dad	12	3	15	6.84	3.23	0.77 (0.23)	0.01 (0.46)	0.90

Table 2.4. – continued

Criticism								
BF	12	3	15	5.11	2.70	1.65 (0.23)	2.46 (0.46)	0.87
BF2	11	3	14	4.98	2.64	1.53 (0.23)	1.82 (0.46)	0.82
Mom	12	3	15	5.35	2.87	1.41 (0.23)	1.75 (0.46)	0.79
Dad	9	3	12	6.00	2.28	0.63 (0.23)	0.07 (0.46)	0.81
Dominance								
BF	11	3	14	7.38	2.19	0.58 (0.23)	0.32 (0.46)	0.53
BF2	12	3	15	6.99	2.30	0.82 (0.23)	1.15 (0.46)	0.54
Mom	12	3	15	10.33	2.92	-0.36 (0.23)	-0.30 (0.46)	0.53
Dad	12	3	15	9.42	3.38	-0.28 (0.23)	-0.70 (0.46)	0.65
Exclusion								
BF	10	3	13	6.16	2.25	0.80 (0.23)	0.72 (0.46)	0.50
BF2	10	3	13	6.10	2.12	0.61 (0.23)	0.38 (0.46)	0.43
Mom	12	3	15	7.26	2.76	0.31 (0.23)	-0.37 (0.46)	0.47
Dad	11	3	14	7.14	2.98	0.32 (0.23)	-0.69 (0.46)	0.53
Overall Positive								
BF	48	27	75	56.29	13.04	-0.30 (0.23)	-0.90 (0.46)	0.88
BF2	55	20	75	51.40	14.42	-0.31 (0.23)	-1.01 (0.46)	0.90
Mom	57	18	75	58.83	13.26	-0.84 (0.23)	0.22 (0.46)	0.91
Dad	60	15	75	51.05	16.82	-0.59 (0.23)	-0.54 (0.46)	0.93
Overall Negative								
BF	52	15	67	30.35	9.45	1.08 (0.23)	1.67 (0.46)	0.81
BF2	45.81	16	61.81	29.18	9.62	1.33 (0.23)	1.68 (0.46)	0.83
Mom	57	15	72	37.00	11.51	0.68 (0.23)	0.26 (0.46)	0.82
Dad	52	15	67	35.02	11.06	0.72 (0.23)	0.61 (0.46)	0.77

Table 2.5 Correlations Between Dimensions of Perceived Social Support (Self-Report)

	BF	BF2	Mom	Dad
Companionship				
BF	1			
BF2	0.68**	1		
Mom	0.25**	0.18	1	
Dad	0.26**	0.22*	0.58**	1
Disclosure				
BF	1			
BF2	0.79**	1		
Mom	0.08	0.09	1	
Dad	-0.05	0.01	0.63**	1
Satisfaction				
BF	1			
BF2	0.57**	1		
Mom	0.22*	0.23*	1	
Dad	0.12	0.30**	0.33**	1
Support				
BF	1			
BF2	0.83**	1		
Mom	0.29**	0.28**	1	
Dad	0.23*	0.22*	0.53**	1
Approval				
BF	1			
BF2	0.79**	1		

Table 2.5. – continued

Mom	0.18	0.29**	1	
Dad	0.14	0.19*	0.48**	1
Pressure				
BF	1			
BF2	0.58**	1		
Mom	0.45**	0.32**	1	
Dad	0.26**	0.32**	0.71**	1
Conflict				
BF	1			
BF2	0.45**	1		
Mom	0.24*	0.09	1	
Dad	0.12	0.26**	0.47**	1
Criticism				
BF	1			
BF2	0.49**	1		
Mom	0.32**	0.23*	1	
Dad	0.21*	0.15	0.42**	1
Dominance				
BF	1			
BF2	0.58**	1		
Mom	0.39**	0.16	1	
Dad	0.29**	0.10	0.57**	1
Exclusion				
BF	1			
BF2	0.65**	1		

Table 2.5. – continued

Mom	0.22*	0.20*	1	
Dad	0.24*	0.26**	0.71**	1
Overall Positive				
BF	1			
BF2	0.79**	1		
Mom	0.20*	0.21*	1	
Dad	0.13	0.17	0.51**	1
Overall Negative				
BF	1			
BF2	0.49**	1		
Mom	0.40**	0.22*	1	
Dad	0.25**	0.24*	0.57**	1

Note: * $p < 0.05$; ** $p < 0.01$

2.4 Physical and Psychological Health Assessments

2.4.1. Health Survey

This assessment was used to assess how frequently an adolescent experienced health symptoms and health problems linked with stress. The survey consisted of 14 Likert-type questions ranging from 1 (not at all) to 4 (all the time) that assessed how frequently the symptoms occurred and 14 Likert-type questions ranging from 1 (does not hurt at all) to 4 (unbearable pain) that assessed the severity of the symptoms. Examples of symptoms assessed include stomach aches, flu, colds, mouth sores, sore throats, fatigue, fevers, and ear aches. In addition, this survey collected reports of how often adolescents went to the school nurse and the doctor. Adolescents completed this survey during both phase one and session one of phase two; parents completed the survey during session one of phase two. See Table 2.6 for descriptive statistics and reliabilities.

Table 2.6 Descriptive Statistics of Health Outcome Measures

Measure	Range	Min	Max	Mean	SD	Skewness (Std. Error)	Kurtosis (Std. Error)	α
Self Report								
Frequency	51	28	79	46.81	10.56	0.40 (0.23)	0.02 (0.46)	0.87
Severity	40	28	68	39.77	8.63	0.60 (0.23)	-0.16 (0.46)	0.94
Visits	4	2	6	3.23	0.86	0.55(.23)	0.08(.46)	0.72
Ab Pain	29	.00	29	10.71	8.22	0.37(.23)	-0.93(0.46)	0.86
Parent Report								
Frequency	37	28	65	43.99	7.39	0.37 (0.23)	0.06 (0.46)	0.86
Severity	30.10	28	58.10	38.42	7.11	0.66 (0.23)	-0.03 (0.46)	0.88
Visits	1.5	1	2.5	1.62	0.41	0.08(.23)	-0.67(0.46)	0.39
Combined Reports								
Frequency	34.33	28.33	62.67	42.83	6.90	0.17(.23)	-0.14(.46)	0.72
Severity	26.67	26.33	53.00	36.69	6.09	0.30(.23)	-0.53(.46)	0.67
Visits	3.33	2.00	5.33	3.23	0.74	0.39(.23)	-0.31(.46)	0.74

2.4.2. Abdominal Pain Index

In addition to assessing general health problems previously linked with stress, I also focused on abdominal pain. The Abdominal Pain Index consisted of five Likert-type questions. These questions assessed experiences of abdominal pain over the past two weeks. Questions examined the frequency of abdominal pain, length of the pain, and intensity of the pain. Only the adolescents completed this questionnaire. These items had high reliability, $\alpha = 0.86$.

2.4.3. Achenbach – CBCL and YSR

Achenbach – CBCL was used to assess adolescent's social competencies and problems such as aggression, hyperactivity, bullying, conduct problems, defiance, and violence (Achenbach & Rescorla, 2001). The questionnaire is comprised of two sections. Twenty Likert-type items assessed the adolescent's competency; 120 Likert-type items assessed the adolescent's behavior or emotional problems. The questionnaire is comprised of 12 subscales which assess a number of psychological and physical health outcomes, namely aggressive behavior, anxious/depressed, attention problems, delinquent rule-breaking behavior, social problems, somatic complaints, thoughts problems, withdrawn, externalizing problems, internalizing problems, total problems and DSM-oriented scales. For this dissertation, I examined internalizing and externalizing problems (i.e., adjustment problems), which was a composite of aggressive behavior and rule-breaking behavior. Both parents and adolescents completed versions of the Achenbach. Since parent- and self-reports were significantly correlated for internalizing ($r = 0.28$, $p < 0.01$) and externalizing ($r = 0.37$, $p < 0.01$) problems, I averaged parent and self responses. That is, I decided to collapse across the measures by creating *consensus trait measures* which are thought to be a way to remove biases associated with idiosyncratic reporting that may be present in child- versus parent-reports (see Bouchard & Loehlin, 2001, p. 258). See Table 2.7 for correlations between self- and parent-reports for internalizing and externalizing problems.

Table 2.7 Correlations Between Parent- and Self-Reports of Adjustment Problems

	Internal Problems SR	External Problems SR	Internal Problems	External Problems
Externalizing Problems SR	0.58**	1		
Internalizing Problems PR	0.28**	0.14	1	
Externalizing Problems PR	0.10	0.37**	0.31**	1

Note: ** $p < 0.05$

2.5 Experimental Questionnaires

2.5.1. Trier Social Stress Test (TSST) Post-Survey Questionnaire

A brief 18-item survey was administered after adolescents completed the TSST. This survey was comprised of Likert-type questions ranging from 1 (not at all) to 7 (very much) that assessed how adolescents felt during the speech delivery (e.g., perceived acceptance, rejection, evaluated, confident, in control). In addition, this post-survey assessed the adolescents' opinions about giving the speech (e.g., the task was challenging, stressful). To assess self-reported stress experienced during the TSST, I created a composite score for how accepted adolescents felt while giving their speech. The composite consisted of the following items: (1) did you feel liked during the task?; (2) did you feel accepted during the task?; (3) did you feel disliked during the task? (reverse-scored); and (4) did you feel rejected during the task? (reverse-scored) with a reliability of 0.85. In addition, I combined how stressful and how challenging adolescents viewed the task ($r = 0.62$, $p < 0.001$; $\alpha = 0.75$). I also specifically looked at the individual items which assessed whether adolescents felt like they wanted to give up during their speech, in control, and evaluated.

2.6 Procedure

A two-tiered consent process was used in which a parent/legal guardian gave consent for the adolescent to participate and adolescents assented to participate. Parents and their adolescent child were recruited to participate in a study on friendship, peer relationships, and health. During recruitment, parents were informed about the nature of the study. This project consisted of two phases in which phase one had one session and phase two had two sessions.

In phase one, adolescents completed a series of surveys either at their school in small groups or online individually. These questionnaires were completed prior to the laboratory session in order to rule out the plausible explanation that responding to the questionnaires may influence reactions to the experimental manipulation. Questionnaires completed prior to arrival at the laboratory included assessments of victimization (DIAS – Victim Version; Bjorkqvist,

Lagerspetz, & Osterman, 1992; CSEQ-SR; Crick & Grotepher, 1995), social support (NRI-D; Furman & Buhrmester, 1985; Buhrmester, Camparo, & Christensen, 1991; Social Support Scale; Harter, 1985), and health outcomes. Weight, height, and waist circumference were also collected (but are not discussed in the current dissertation).

Adolescents and their parent then came into the laboratory at the University of Texas at Arlington for a two-part second phase of research. Upon arrival, the adolescent and parent were welcomed to the study and lead into a room for the consent process. As in phase one, parents provided written consent and adolescents provided written assent. During session one of phase two, adolescents completed a second assessment of health outcomes as well as a number of questionnaires that assessed individual differences in personality, loneliness, and depression. Again, the measures of personality, loneliness, and depression were part of a larger study and were not analyzed as part of this dissertation. Parents completed parental-reports of their adolescent's experiences of victimization, social support, and health outcomes (i.e., modified versions of the questionnaires the adolescent completed in phase one). Adolescents and their parent were then taught how to collect samples of the adolescent's saliva (see section below on cortisol collection and analysis). Adolescents were instructed to collect four samples of their saliva over each of two non-sport school days. Samples were collected immediately when the adolescent awoke, 30 minutes after waking, immediately when the adolescent returned home from school, and 30 minutes before going to bed. The saliva samples were collected to measure salivary basal cortisol levels in order to determine each individual adolescent's diurnal cortisol pattern. After learning how to collect their saliva samples, adolescents were then taught how to use a Palm Pilot. The Palm Pilots were used to ensure correct saliva sampling by prompting adolescents to completely soak the cotton swab, to make sure the cap to the sample tube was on tightly, and to place the tube in the freezer. Adolescents logged onto the palm pilots during each saliva sampling; they were told that the Palm Pilots would record the date and time that the questions were answered. All adolescents were given an instruction sheet to post

on their refrigerator at home as well as my contact information in case they had problems or questions while collecting the saliva samples.

Adolescents and their parent then returned to the laboratory for the second session of phase two. Typically, participants returned within three to nine days. This second session was held between 4:00pm and 7:00pm to control for diurnal cycles with the majority of participants beginning the session at either 4:00pm or 5:30pm. Parents completed several questionnaires about their child's personality, levels of loneliness and depression, and the Achenbach-CBCL. Again, measures of personality, loneliness, and depression were part of a larger study and were not analyzed for this dissertation. Meanwhile, adolescents were brought into a separate room where they began a ten minute rest period. The rest period was required to minimize potential activation of the HPA axis which would lead to an increase in cortisol levels; this potential increase in cortisol could confound later activation during the TSST. After the 10 minute rest period, the adolescent was asked to provide the first saliva sample of the evening.

The adolescent was then led to another room in which the experimental manipulation occurred. This room was set up such that when the adolescent walked into the room he/she was facing a table where two undergraduate research assistants (RAs) sat in white lab coats. A videocamera was placed on the table. In addition, the two RAs each had a clipboard in front of them and appeared serious (i.e., kept a neutral face and did not smile or nod their heads). At this point, I explained to the adolescent that he/she would deliver a speech explaining to the committee why he/she would make an ideal class president. The adolescent was told that he/she would have 10 minutes to prepare for the speech. They were also told that the committee members would take notes regarding the content and delivery of the speech. I pointed out that each committee member had been trained in behavioral observation and would be recording behaviors accordingly. Moreover, I told the adolescents that the speech would be videotaped so it could be shown to several of their peers who would also evaluate whether or not the adolescent would be a good class president. I then summarized the task by telling the

adolescents that they should imagine they were invited to introduce themselves to a group of teachers who would be evaluating their candidacy for class president and that their speech needed to last for five minutes. The adolescents were then given a chance to ask questions before being led back into the preparation room.

Once back in the preparation room, I told participants that they would now have the chance to make notes for the speech. I also pointed out that these notes could not be used during the speech delivery. Each adolescent was told to try very hard to make a "believable impression" since the committee members would ask additional questions in case there was any disagreement about whether or not they thought the participant would be a good class president or in case they had any follow-up questions. I then reminded the participants that they needed to make their speech last for five minutes and gave them another opportunity to ask questions. Participants were then left alone for 10 minutes to prepare; halfway through the preparation time, I gave them a 5-minutes-left-warning. After the 10 minute preparation period, I collected a second saliva sample.

Adolescents were then brought back into the experimental room. The committee chair turned on the video camera while I told the participant that they would be asked to state their ID number (and reminded them what their number was). Once the camera was on, I checked with the committee chair that the participant could be seen in the camera and then left the room. Adolescents were asked by the committee chair to state their participant ID and then began their speech. During the speech delivery, the two committee members remained quiet while the adolescent was speaking fluently. If the adolescent paused for longer than 20 seconds, the committee chair (who always sat on the right) said "You still have time, please continue…." If another 10 seconds elapses, the committee members began asking questions (e.g., what qualifies you in particular for this position?) until the five minutes ended. Two additional questions were then asked (e.g., "If you were elected class president, what would your first order of business be?"). If the participant talked for the full five minutes, then two follow-up

questions were asked (see Appendix for the full set of questions). The committee chair then instructed the adolescent to wait while they completed their evaluation (roughly 15-20 seconds). The participants were then thanked for giving their speech and left the room.

I then brought the adolescent back to the resting room. At this time, a third saliva sample was collected. Adolescents then completed the post-TSST survey and the Achenbach-YSR. A fourth and final saliva sample was collected 30 minutes after the third sample. Parents and adolescents were then carefully debriefed together and paid for their participation.

CHAPTER 3

OVERVIEW OF DATA ANALYSIS

3.1 *Cortisol Data Collection and Analysis*

Cortisol samples were collected in salivettes (Starstedt) nested tubes and tested with salivary cortisol kits from Salimetrics (State College, PA). The tubes contain a small cotton swab similar to swabs used at the dentist office. Participants placed the cotton swab in their mouth between their cheek and gum for between 45 – 60 seconds until it was thoroughly moisten. Participants were told they could roll the cotton swab over and under their tongue and gently "gum" the swab by squeezing their cheek against their gum. They were instructed *never* to bite on the cotton swab since doing so would remove some of the collected saliva and hormones. Once the cotton swab was saturated with saliva, it is placed in the tube. Adolescents recorded the exact time and date of the saliva sample on the tube. Samples were then placed the freezer until participants returned to the lab. I then stored the samples in a refrigerator until they could be spun and frozen at -20℃ for future assay. The Enzyme-Linked Immunoabsorbence Assay (ELISA) was used to measure the quantity of cortisol in each sample. The assays were analyzed in Dr. Baum's cortisol laboratory at UT Arlington.

3.2 Handling of Missing Data

In order to determine how detrimental missing data is, the pattern that the missing values take must be examined. It is the pattern, rather than the quantity, of missing data that dictates how to handle missing data points (Tabachnick & Fidell, 2007). I ran the missing value analysis (MVA) in SPSS to assess the pattern of my missing data points. The Little MCAR tests were *not* significant, thereby indicating that the missing data points were missing completely at random (MCAR; Little, 1988) (χ^2 = 35.30, 172.34, 0.00, 99.81, 503.85, *df*s = 42, 257, 18598,

6240, 3739, *p*s = 0.76, 1.00, 1.00, 1.00, 1.00, for Achenbach, cortisol, child phase 1, child phase 2, and parent reports respectively) . In other words, MCAR means that the missing data is *not* related to other variables; it is the ideal pattern when missing data is present. Since my missing data points were MCAR, I used expectation maximization (EM) methods which substituted estimated expectations of missing data for the missing data points and then ran maximum likelihood estimation with the included substitutions. Once convergence was reached, the final data set (with the imputed values) was saved. I used this final complete data set with no missing values for my analyses.

3.3 *Overview of Data Analytic Strategy*

The purpose of this dissertation was to examine the effects of peer victimization on (1) physical health outcomes; (2) cortisol production (both diurnal patterns and reactivity to social stress); and (3) psychological outcomes. Moreover, I sought to determine whether perceived social support buffered against the negative influences of being victimized in early adolescence.

Peer victimization was treated as both a continuous variable as well as a dichotomized variable (i.e., victims versus nonvictims). Treating peer victimization as both continuous and dichotomized allowed me to examine the effects of victimization using a variable-centered approach (i.e., as a continuous variable) and a person-centered approach (i.e., as a dichotomized variable). To create the continuous variable, I first examined my zero order correlations to determine whether the individual subscales of victimization were correlated. Since all the subscales were highly correlated across both parent- and self-reports (see Table 3.1), I created a composite score comprised of all the parent- and self-reports for all the victimization subscales to again remove any biases associated with child- versus parent-raters. I also examined whether boys and girls significantly differed in their experiences of victimization. I found that boys reported significantly more overt victimization ($t(105) = 3.54$, $p < 0.01$, $M_{boys} = 0.32$, $SD_{boys} = 0.98$, $M_{girls} = -0.25$, $SD_{girls} = 0.65$), physical victimization ($t(105) = 4.13$, $p < 0.001$, $M_{boys} = 0.36$, $SD_{boys} = 1.02$, $M_{girls} = -0.28$, $SD_{girls} = 0.59$), and more verbal victimization ($t(105) =$

2.22, $p < 0.05$, $M_{boys} = 0.21$, $SD_{boys} = 0.99$, $M_{girls} = -0.16$, $SD_{girls} = 0.73$) than did girls. No significant differences in experiences of victimization between girls and boys were found for total victimization (an overall composite of all subscales of victimization), relational victimization, or indirect victimization. Future work will examine the impact of specific subscales of victimization (e.g., relational, overt) on biological functioning; the current dissertation examined the impact of overall victimization.

Table 3.1 Correlations Between Parent and Child Reports of Victimization

	Overt PR	Relational PR	Physical PR	Verbal PR	Indirect PR
Overt SR	0.47**	0.33**	0.45**	0.44**	0.38**
Relational SR	0.30**	0.40**	0.25*	0.36**	0.44**
Physical SR	0.40**	0.27**	0.49**	0.31**	0.34**
Verbal SR	0.48**	0.49**	0.44**	0.50**	0.54**
Indirect SR	0.32**	0.45**	0.26**	0.36**	0.54**

Note: * $p < 0.05$; ** $p < 0.01$

To create groups of victimized versus nonvictimized persons I used a two-step classification process that is thought to lead to a more valid and robust pattern. I began by conducting agglomerative hierarchical cluster analyses which begins by treating each person as a cluster and then combining individuals into clusters (based on proximity to one another) until all persons are in one large cluster. The goal is determine the appropriate number of clusters by examining the dendogram and agglomeration coefficients. Researchers look for the percentage of change from each stage of clustering. A large change in the agglomeration cluster, which reflects the distance between the cluster centers, indicates that two heterogeneous clusters were combined (i.e., two clusters that have large distances between them). As such, the researcher wants to choose the solution prior to heterogeneous clusters being combined. The dendogram provides a visual representation of this clustering process, which further aids the

researcher in this process. Ward's method was chosen because it is most recommended (Steele & Aylward, 2007). The five dimensions of victimization (collapsed across rater) were used to establish the initial cluster solution. Based on the agglomeration coefficient changes and the dendogram, I chose a two –cluster solution[1].

On the second step of my analyses, I used k-cluster means analysis (with normalized Euclidean distance as the distance metric) to confirm my two cluster group solution. K-mean clustering partitions the participants into clusters by minimizing the SS_{within} within each cluster (or their distance to the cluster center). The specific cluster centers for the five victimization dimensions from the hierarchical cluster analysis were used as the initial cluster centers for the k-means clustering. The cluster solution converged in two iterations (which is very good). In addition, 99.98% of the participants were classified in the same groups for both methods. That is, only two participants were classified differently using these two methods. Using McNemar's test, which assesses the changes in participant's scores on nominal data from one assessment to another assessment, I found that there were no significant changes in group membership, $p = 0.50$. In addition, the victimized group (n = 31) were significantly more victimized ($M = 0.99$, $SD = 0.43$) than the nonvictimized group (n = 76; $M = 0.04$, $SD = 0.39$) on the overall composite of victimization, $F(1, 105) = 234.85$, $p < .01$, $\eta^2 = 0.69$.

To assess physical health outcomes, several composites of parent- and self-reports were created to serve as dependent measures (1) frequency of health problems; (2) severity of health problems; (3) frequency of adolescent's visits to the school nurse/doctor; and (4) abdominal pain. (See Tables 3.2 and 3.3 for correlations). The subscales of internalizing and externalizing problems from the Achenbach were collapsed across raters and used to evaluate psychological outcomes.

[1] 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.

Table 3.2 Correlations Between Parent and Adolescent Reports of Health Outcomes

	Freq sym T1	Sev sym T1	Freq visits T1	Sev visits T1	Freq sym T2	Sev sym T2	Freq visits T2	Sev visit T2	Freq sym PR	Sev sym PR	Freq visit PR
Freq sym T1	1										
Sev sym T1	0.83**	1									
Freq visits T1	0.66**	0.59**	1								
Sev visits T1	0.42**	0.47**	0.48**	1							
Freq sym T2	0.69**	0.66**	0.41**	0.23*	1						
Sev sym T2	0.58**	0.72**	0.37**	0.27**	0.80	1					
Freq visits T2	0.54**	0.42**	0.63**	0.28**	0.55**	0.41**	1				
Sev visits T2	0.32**	0.33**	0.34**	0.51**	0.42**	0.42**	0.43**	1			
Freq sym PR	0.28**	0.19	0.15	0.09	0.38**	0.21*	0.27**	.13	1		
Sev sym PR	0.28**	0.19*	0.17	0.11	0.26**	0.21*	0.25**	.03	0.79**	1	
Freq visits PR	0.36**	0.23*	0.40**	0.19*	0.35**	0.25**	0.43**	.16	0.52**	0.43**	1
Sev visits PR	0.33**	0.22*	0.21*	0.29**	0.26**	0.20*	0.34**	.23*	0.53**	0.47**	.58**

Note: * $p < 0.05$; ** $p < 0.01$

Table 3.3 Correlations Between Final Health Outcomes Composites (Average of Parent- and Self-Reports)

	Frequency of symptoms	Severity of symptoms	Frequency of visits	Abdominal Pain
Frequency of symptoms	1			
Severity of symptoms	0.84**	1		
Frequency of visits	0.67**	0.56**	1	
Abdominal Pain	0.45**	0.42**	0.34**	1

Note: * $p < 0.05$; ** $p < 0.01$

CHAPTER 4

RESULTS

4.1 *Does Peer Victimization Predict Poorer Health Outcomes?*

I expected peer victimization would predict self-reported health outcomes. Specifically, I expected that peer victimization would predict (1) frequency of health symptoms; (2) severity of health symptoms; (3) frequency of visits to the nurse and/or doctor; and (4) self-reports of abdominal pain. High correlations were found between victimization and health outcomes (see Table 4.1). To examine this hypothesis, I first conducted a series of regression analyses in which peer victimization was treated as a continuous variable. As expected, I found that victimization predicted how frequently adolescents experienced health problems ($t(105) = 7.31$, $p < 0.01$, $sr^2 = 0.34$) and how severe health symptoms were experienced $t(105) = 6.76$, $p < 0.01$, $sr^2 = 0.30$; see Figures 4.1 and 4.2). Being highly victimized also predicted increased reports of abdominal pain ($t(105) = 4.98$, $p < 0.01$, $sr^2 = 0.19$; see Figure 4.3). In addition, victimization predicted how frequently adolescents visited the school nurse or doctor $t(105) = 3.86$, $p < 0.01$, $sr^2 = 0.12$ see Figures 4.4).

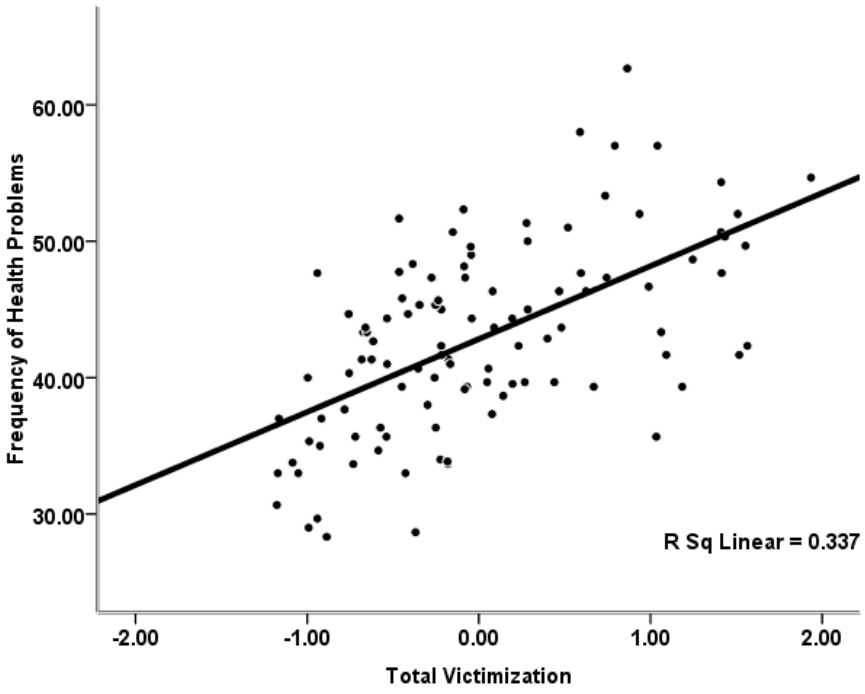


Figure 4.1 Peer Victimization Predicts Frequency of Health Problems

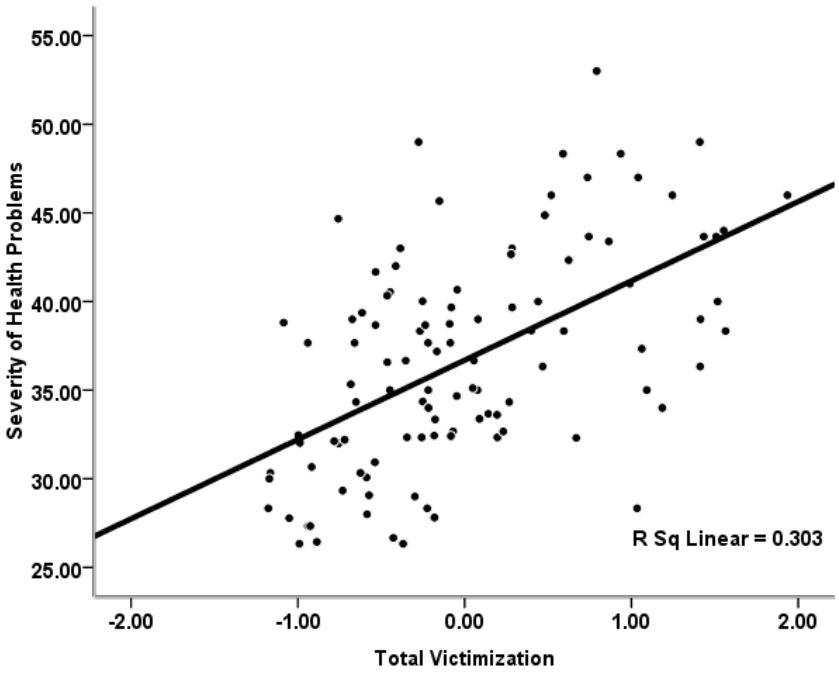


Figure 4.2 Peer Victimization Predicts Severity of Health Problems

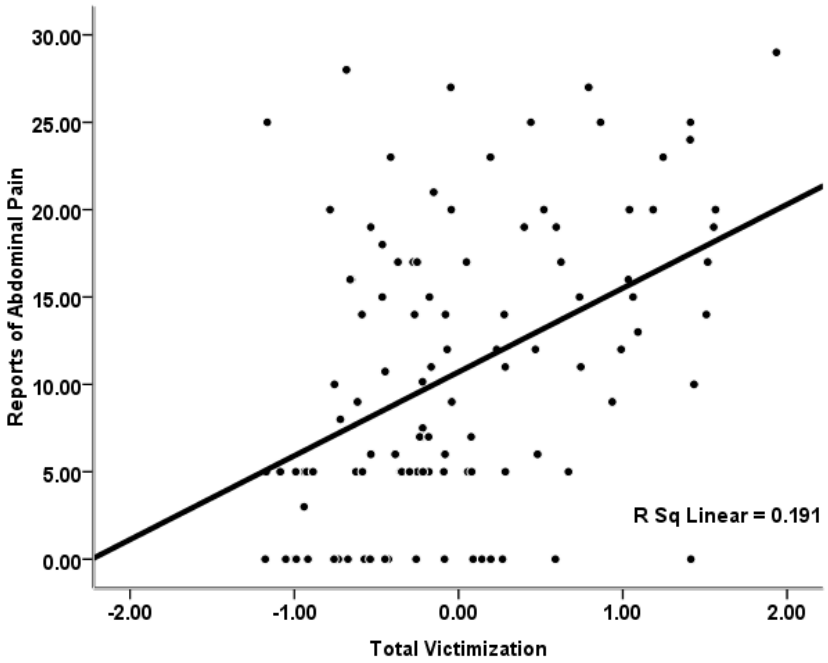


Figure 4.3 Peer Victimization Predicts Self-Reports of Abdominal Pain

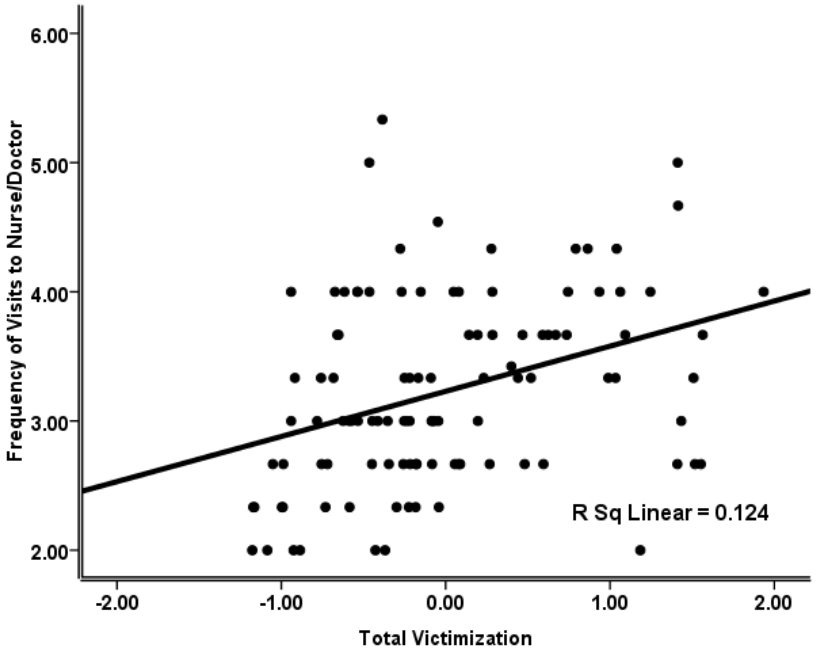


Figure 4.4 Peer Victimization Predicts Frequency of Visits to the School Nurse/Doctor

Second, I looked at victimization from a person-centered approach using the two groups from my cluster analysis to examine whether there were differences in health outcomes between victimized versus nonvictimized adolescents. As expected, I found significant differences between victimized and nonvictimized adolescents. Specifically, I found that victimized adolescents had higher reports of frequency and severity of health problems (*t*s(105) = -5.61, -6.25, *p*s < 0.01, respectively), abdominal pain (*t*(105) = -4.50, *p* < 0.01), and visits to the school nurse/doctor (*t*(105) = -3.07, *p* < 0.01). See Figures 4.5, 4.6, 4.7, and 4.8 for visual displays of these significant differences.

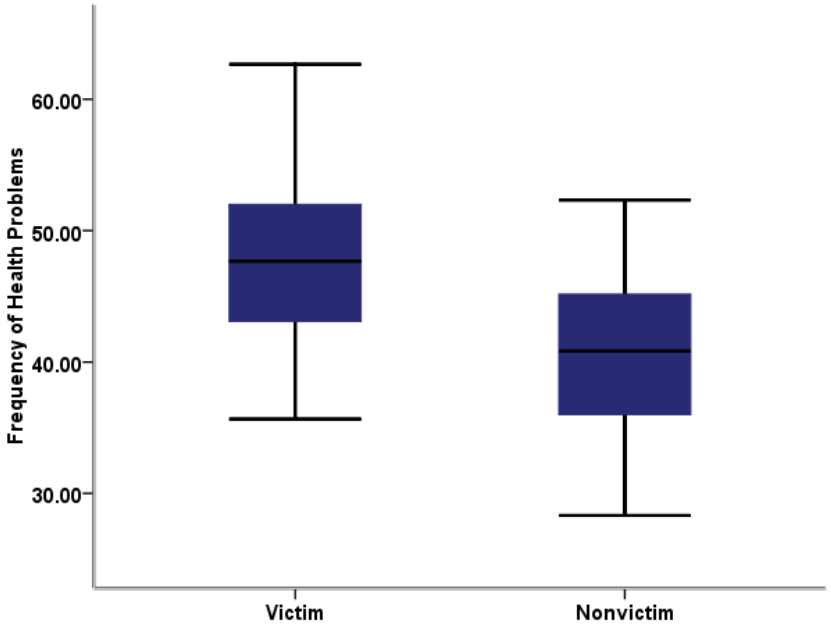


Figure 4.5 Frequency of Health Problems for Victimized versus Nonvictimized Adolescents

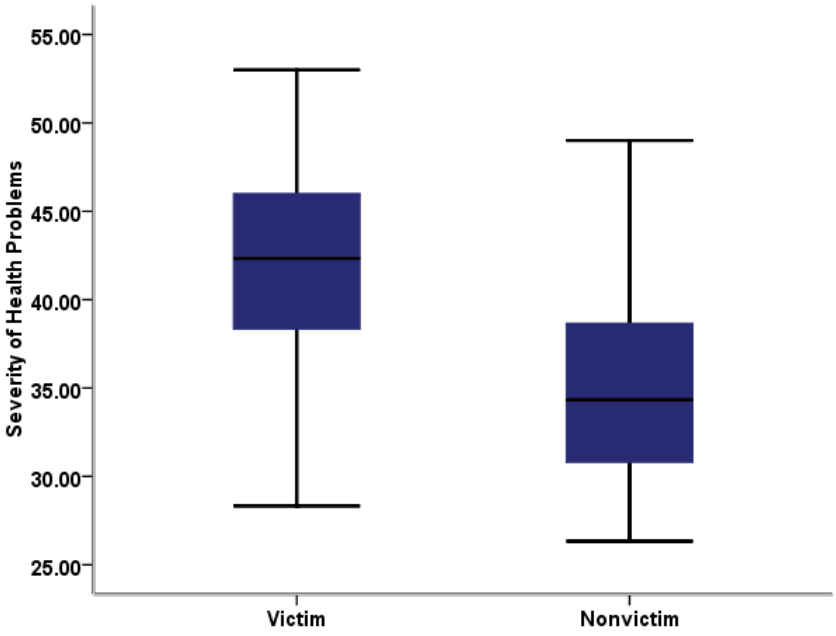


Figure 4.6 Severity of Health Problems for Victimized versus Nonvictimized Adolescents

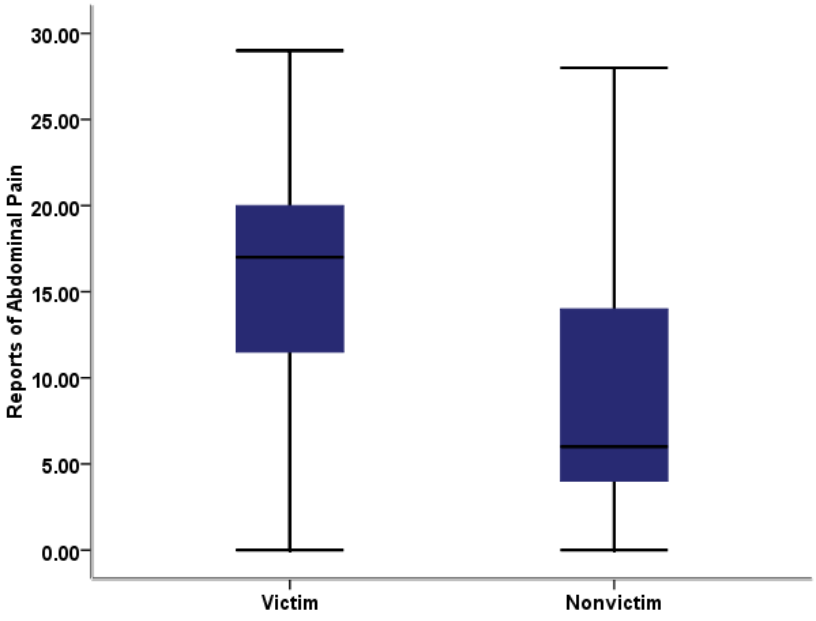


Figure 4.7 Abdominal Pain for Victimized versus Nonvictimized Adolescents

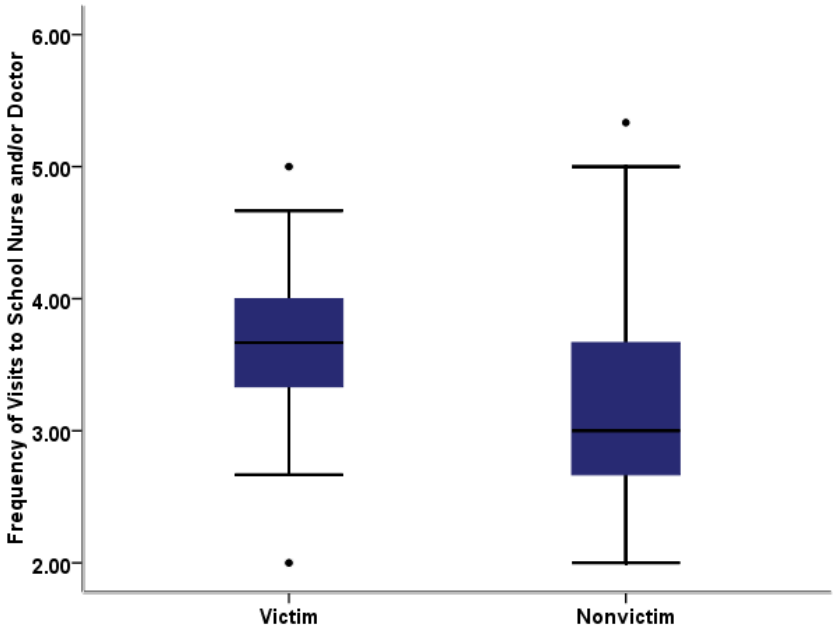


Figure 4.8 Frequency of Visits to the School Nurse and/or Doctor for Victimized versus Nonvictimized Adolescents

4.2 *Does Sex of Participant Moderate the Link Between Victimization and Health?*

Next, I conducted a series of moderated multiple regression analyses to examine whether sex of the participant moderated the link between victimization and health outcomes. I entered centered victimization and sex on step one; on step two I entered the cross product. I found that sex marginally moderated the link between victimization and reports of abdominal pain, $t(103) = -1.93$, $sr^2 = 0.03$, $p = 0.057$. A significant effect was found for boys ($t(45) = 6.16$, $p < 0.01$, $sr^2 = 0.45$ indicating that victimization significantly predicted more abdominal pain. A marginal effect was found for girls ($t(58) = 1.83$, $p = 0.07$, $sr^2 = 0.05$. Both victimization and sex uniquely contributed to reports of abdominal pain ($ts(103) = 4.97, 2.00$, $sr^2s = 0.18, 0.03$, ps, $< 0.01, 0.05$, respectively). See Figure 4.9. Sex did not moderate the link between victimization and frequency of symptoms, severity of symptoms nor frequency of visits to the doctor and/or nurse ($ts(103) = 0.84, 0.18, 0.30$, $ps = 0.40, 0.86, 0.79$, $sr^2s = 0.00$.

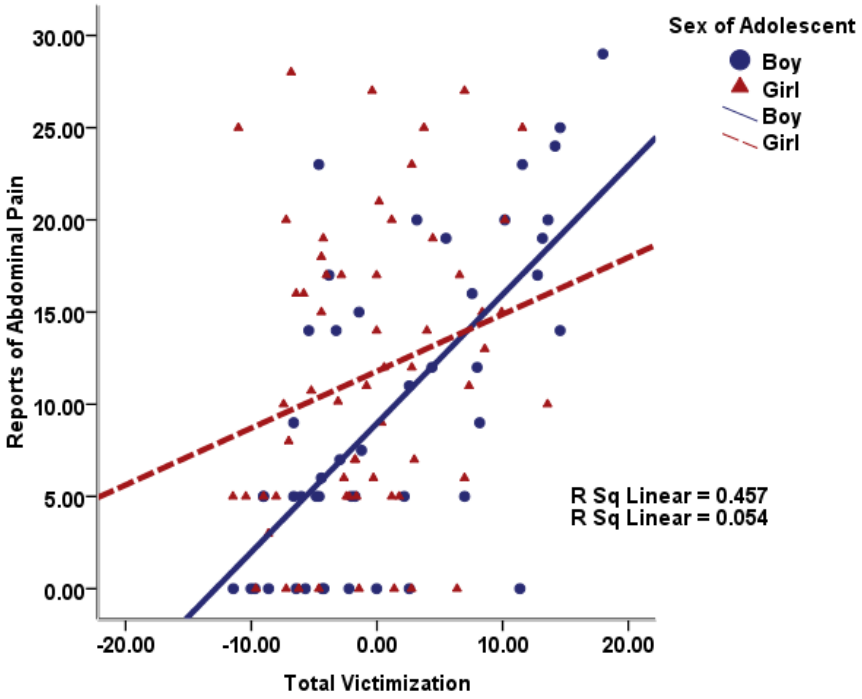


Figure 4.9 Victimization Predicts Abdominal Pain for Boys and Girls

Given the low power to detect effects (i.e., only 31 victimized adolescents, of which only 13 were girls) and the recommendation of Aiken and West (1991), I decided to split my data set by sex and re-run the series of regression analyses. These analyses allowed me to examine the pattern between victimization and health outcomes for girls and boys. Indeed, I found that victimization significantly predicted frequency of symptoms, severity of symptoms and frequency of visits to the doctor/nurse both boys (ts(45) = 5.93, 5.08, 3.28, ps < 0.05, sr^2 = 0.44, 0.36, 0.19, respectively) and girls (ts(58) = 5.39, 5.01, 2.85, ps < 0.01, 0.01, 0.05, sr^2 = 0.33, 0.30, 0.12, respectively). See Figures 4.10, 4.11, and 4.12.

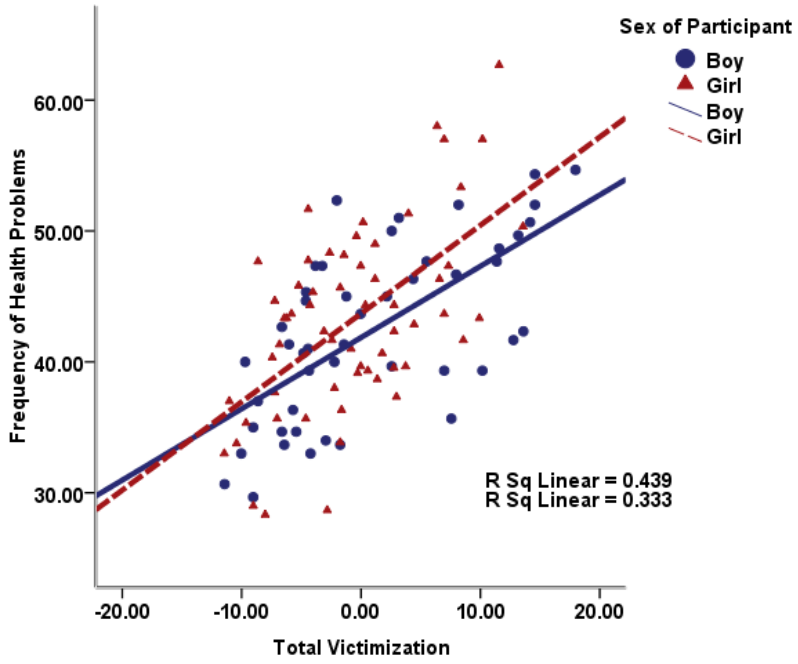


Figure 4.10 Victimization Predicts Frequency of Health Problems for Boys and Girls

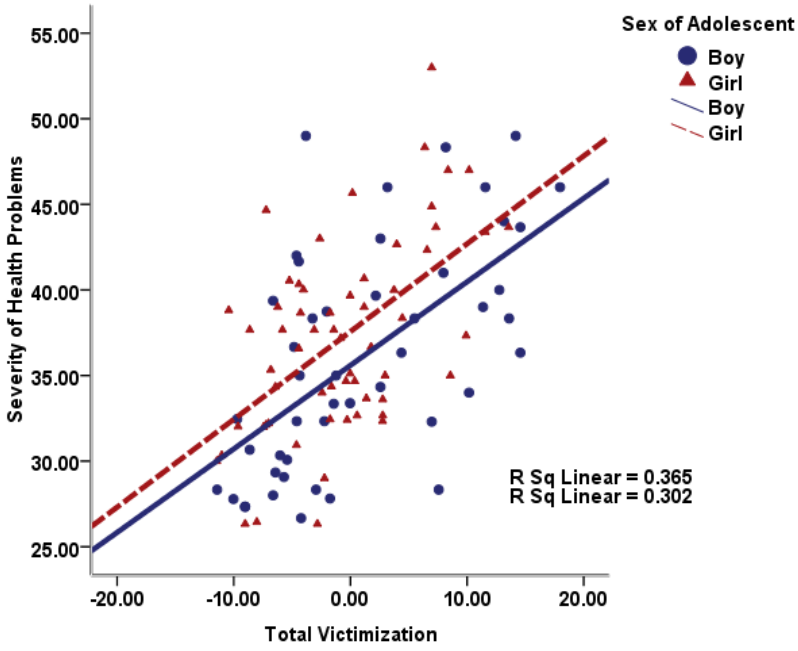


Figure 4.11 Victimization Predicts Severity of Health Problems for Boys and Girls

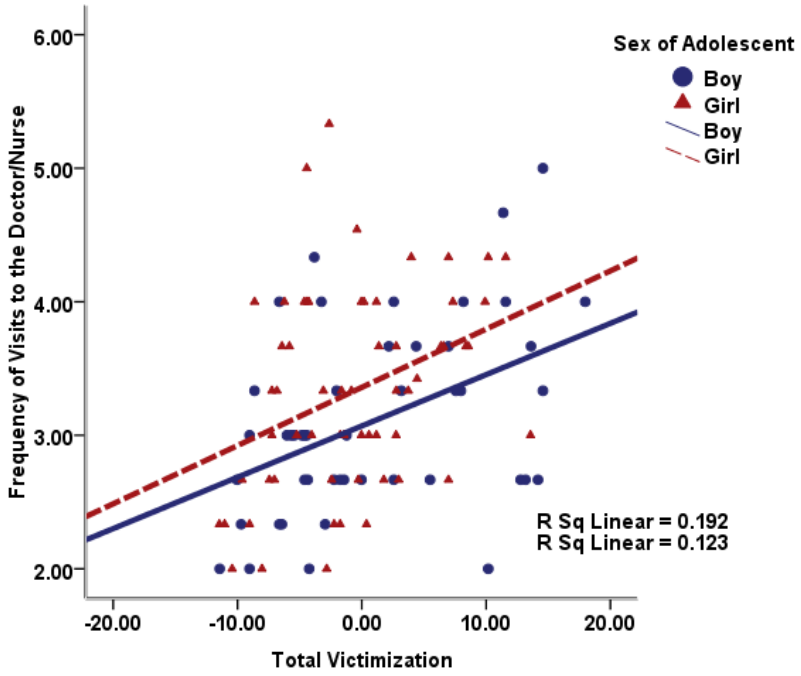


Figure 4.12 Victimization Predicts Frequency of Visits to the Doctor/Nurse for Boys and Girls

4.3 *Does Perceived Social Support Moderate the Victimization-Health Link?*

I expected that perceiving social support from close others would moderate the link between peer victimization and health outcomes. Specifically, I expected that high levels of positive dimensions of social support would buffer adolescents against health problems whereas high levels of negative dimensions would exacerbate health problems. I ran a series of moderated multiple regression (MMR) analyses to test these hypotheses. For each MMR model, I entered the centered victimization score, centered perceived support from friends, and the centered perceived support from parents onto the first step. I then created cross products by multiplying the centered perceived score by the centered victimization score. In other words, I created two cross products for each dimension of social support (i.e., one for perceived friend support, one for perceived parental support). In addition, I created a friend X parent cross product for each dimension. These cross products were entered onto the second step of the model. Finally, I created the three-way interaction (i.e., centered victimization X perceived friend

support X perceived parental support) and entered it onto the third step of the model. I examined each dimension of support separately.

Initially, there were four dependent measures: frequency of health symptoms, severity of health symptoms, frequency of health visits, and abdominal pain. In addition, there were 10 dimensions of support, which equated to 40 regression models. As such, I dropped abdominal pain and severity of symptoms from these analyses to limit the number of models to 20 regression models. Abdominal pain was dropped because it was redundant with frequency of health problems, and there was only one assessment of abdominal pain (i.e., one self-report assessment). Severity was dropped because it also redundant with the frequency of health outcomes (r = 0.84). To control further for Type I error rates, only results for the two remaining dependents measures that were significant at $p < 0.05$ will be discussed here[2]. Additional marginal findings for these two measures are reported in Tables 4.2 – 4.5.

Table 4.1 Correlations Between Victimization Measures and Health Outcomes

	Frequency of symptoms	Severity of symptoms	Frequency of visits	Abdominal Pain
Total Overt	0.46**	0.45**	0.23**	0.39**
Total Relational	0.60**	0.58**	0.42**	0.36**
Total Physical	0.39**	0.39**	0.16	0.39**
Total Verbal	0.52**	0.48**	0.36**	0.39**
Total Indirect	0.56**	0.51**	0.36**	0.38**
Total Victim	0.58**	0.55**	0.35**	0.44**

Note: * $p < 0.05$; ** $p < 0.01$

[2] Clearly, this is not "stringent" by many researchers' standards. However, I wanted to provide a complete picture of my findings.

Table 4.2 Main Effects When Examining Whether Social Support Moderates the Link Between Victimization and Frequency of Symptoms

Relationship Dimension/Effect	t-statistic	sr^2	b-weight
Overall Positive Support			
Victimization	7.47**	0.33	0.62
Overall Negative Support			
Victimization	5.77**	0.20	0.41
Companionship			
Victimization	7.28**	0.32	0.58
Disclosure			
Victimization	8.26**	0.40	0.67
Friend	1.92+	0.02	0.14
Satisfaction			
Victimization	7.20**	0.29	0.58
Parent	-1.67+	0.02	-0.17
Support			
Victimization	7.90**	0.37	0.64
Approval			
Victimization	6.86**	0.27	0.57
Parent	-1.84+	0.02	-0.20
Pressure			
Victimization	6.77**	0.28	0.55
Friend	1.82+	0.02	0.25
Conflict			

Table 4.2. – continued

Victimization	6.22**	0.24	0.57
Criticism			
Victimization	5.62**	0.193	0.50
Dominance			
Victimization	6.73**	0.28	0.56
Exclusion			
Victimization	6.87**	0.27	0.54

Note: + $p \leq 0.10$; * $p < 0.05$; ** $p < 0.01$

Table 4.3 Main Effects When Examining Whether Social Support Moderates the Link Between Victimization and Frequency of Visits to the Doctor/Nurse

Main Effect	t-statistic	sr^2	b-weights
Overall Positive Support			
Victimization	4.46**	0.16	0.05
Friend	2.24*	0.04	0.01
Overall Negative Support			
Victimization	2.99**	0.07	0.03
Companionship			
Victimization	3.94**	0.13	0.04
Disclosure			
Victimization	4.41**	0.16	0.05
Friend	2.18*	0.04	0.02
Satisfaction			

Table 4.3. – continued

Victimization	6.29**	0.26	
Support			
Victimization	4.61**	0.17	0.05
Friend	2.53*	0.05	0.02
Approval			
Victimization	4.29**	0.14	0.04
Friend	1.90+	0.03	0.02
Pressure			
Victimization	3.84**	0.12	0.04
Conflict			
Victimization	3.43**	0.09	0.04
Criticism			
Victimization	3.45**	0.10	0.04
Dominance			
Victimization	2.91**	0.07	0.03
Friend	2.01*	0.03	0.04
Exclusion			
Victimization	3.56**	0.10	0.04

Note: + $p \leq 0.10$; * $p < 0.05$; ** $p < 0.01$

Table 4.4 Perceived Social Support Moderates the Link Between Victimization and the Frequency of Health Outcomes

Dimension	t-statistic	sr^2	B-Weights		
Victimization X Relationship					
Friend			-1 SD	0 SD	+1 SD
Disclosure	2.59**	0.04	0.47**	0.67**	0.87**
Support	1.98*	0.02	0.48**	0.65**	0.80**
Parent					
Support	-1.86+	0.02	0.78**	0.65**	0.49**
Exclusion	2.61*	0.04	0.32**	0.54**	0.77**
Parent X Friend					
Satisfaction	2.27*	0.03	0.47**	0.60**	0.69**

Note: + $p \leq 0.10$; * $p < 0.05$; ** $p < 0.01$

Table 4.5 Perceived Social Support Moderates the Link Between Victimization and Frequency of Health Visits

Victimization X Dimension of Support	t-statistic	sr^2	B-Weights by Levels of Relationship Dimension		
Friend			-1 SD	0 SD	+1 SD
Approval	1.92*	0.03	0.02	0.04**	0.07**
Pressure	-1.71+	0.02	0.06**	0.04**	0.02
Conflict	-1.90 *	0.03	0.06**	0.04**	0.02
Parent					
Dominance	2.32*	0.04	0.01	0.03**	0.05**

Note: + $p \leq 0.10$; * $p < 0.05$; ** $p < 0.01$.

4.3.1 Main Effects

As discussed above, victimization uniquely predicted negative health outcomes. However, since I have directly examined the link between victimization and health outcomes above, I will not re-report the significant main effects for victimization here (but see Tables 4.2 and 4.3).

Unexpectedly, adolescents who reported higher levels of disclosing intimate information to their friends also reported more frequent visits to the doctor/nurse than adolescents who reported lower levels of disclosing intimate information to friends ($t(103) = 4.41$, $p < 0.01$, $sr^2 = 0.16$). In addition, adolescents who perceived higher levels of support from their friends reported more frequent visits to the doctor/nurse ($ts(103) = 2.53$, $p < 0.01$, $sr^2 = 0.05$). In sum, the data suggest that adolescents report more health problems when they perceive more positive

support from their close friends. This finding is opposite of what I hypothesized. Unexpectedly, there were no consistent patterns of main effects for parent support.

4.3.2 Two-way Interactions

I found several dimensions of social support moderated the link between victimization and health outcomes. Contrary to what I predicted, friend support did not buffer against negative health outcomes. Rather, support seemed to exacerbate the link between victimization and poor health outcomes. For example, the relationship between victimization and frequency of negative health outcomes was strongest for adolescents who reported disclosing more to their friends, $t(101) = 2.59$, $p < 0.01$, $sr^2 = 0.04$ (see Figure 4.13). Similarly, the victimization-health link was strongest for adolescents who perceived more social support from their friends, $t(103) = 1.98$, $p < 0.05$, $sr^2 = 0.02$ (see Figure 4.14). In summary, the data suggest that, contrary to expectations, victimized adolescents who perceive high levels of positive support from two close friends have more frequent health problems than victimized adolescents who perceive lower levels of positive support.

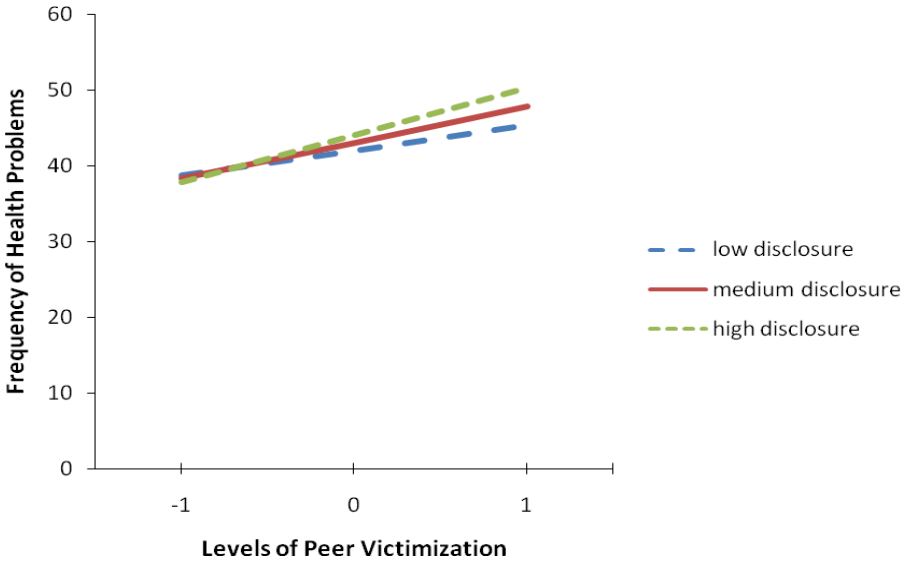


Figure 4.13 Perceived Disclosure from Close Friends Moderates Link Between Victimization and Frequency of Health Problems

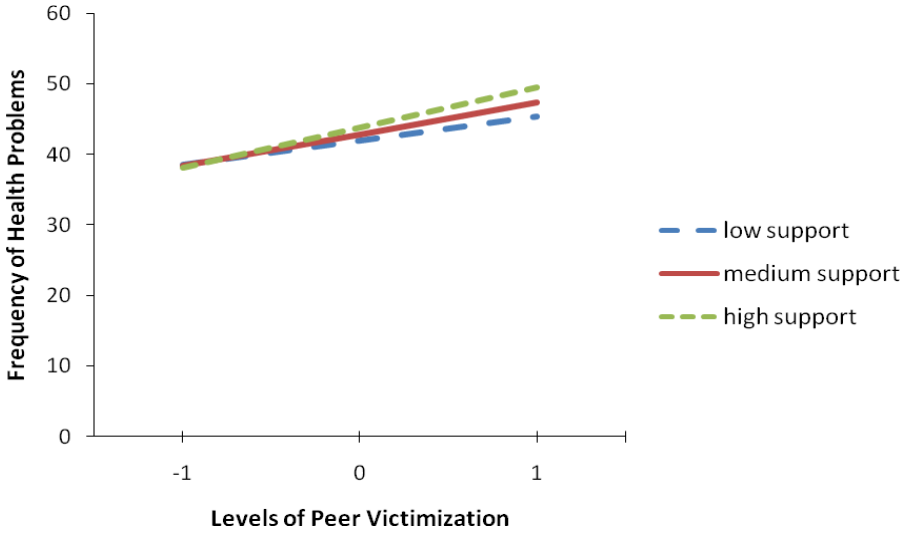


Figure 4.14 Perceived Support from Close Friends Moderates Link Between Victimization and Frequency of Health Problems

A similar pattern emerged for the association between victimization and the frequency of health visits. Approval from close friends moderated the link between victimization and frequency of visits to the doctor/nurse ($t(103) = 1.92$, $p = 0.05$, $sr^2 = 0.03$). Contrary to expectations, high levels of approval were associated with a stronger relationship between more frequent visits to the doctor/nurse and victimization (see Figure 4.15). Perceived conflict from two close friends also moderated the link between victimization and frequency of visits to the doctor/nurse in an unexpected pattern, $t(103) = -1.90$, $p = 0.05$, $sr^2 = 0.03$. For low levels of conflict, the relationship between victimization and frequency of health visits was the strongest (see Figure 4.16). These data suggest high levels of positive support and low levels of negative support from close friends predict more frequent visits to the doctor/nurse for victimized children.

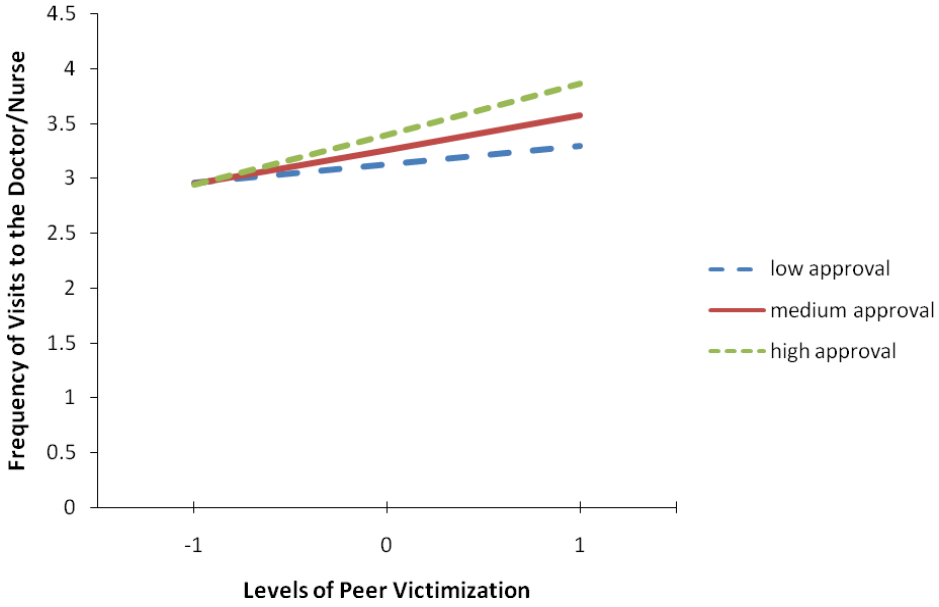


Figure 4.15 Perceived Approval from Close Friends Moderates Link Between Victimization and Frequency of Visits to the Doctor/Nurse

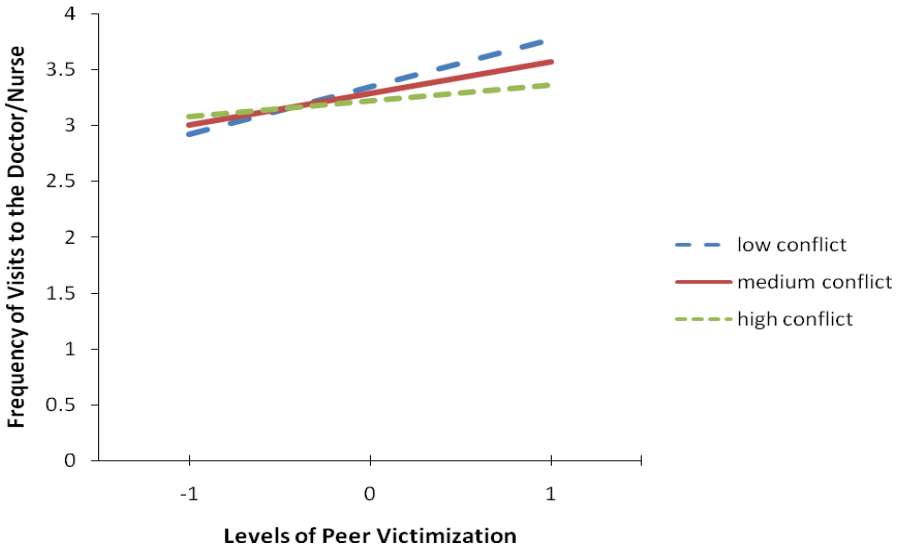


Figure 4.16 Perceived Conflict from Close Friends Moderates Link Between Victimization and Frequency of Visits to the Doctor/Nurse

For parents, perceived exclusion moderated the link between victimization and the frequency of health problems, $t(103) = 2.61$, $p < 0.05$, $sr^2 = 0.04$. As anticipated, high levels of exclusion from parents exacerbated the link between victimization and health problems (see

Figure 4.17). This finding mirrors previous research that has found that a coercive home environment increases problems for victimized children (e.g., Taylor, Lerner, Sage, Lehman, & Seeman, 2004; Loeber & Dishion, 1984). In addition, I found a parent X friend effect for satisfaction ($t(103) = 2.27$, $p < 0.05$, $sr^2 = 0.03$) such that the link between victimization and frequency of health problems was stronger when adolescents perceived low parental satisfaction but high friend satisfaction (i.e., these adolescents reported the most frequent health problems). See Figure 4.18.

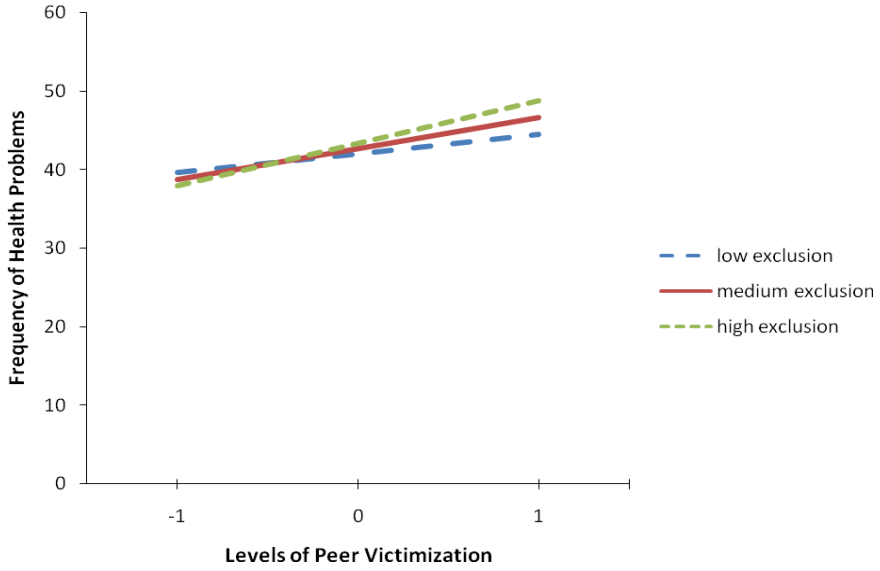


Figure 4.17 Perceived Exclusion from Parents Moderates the Link Between Victimization and Frequency of Health Problems

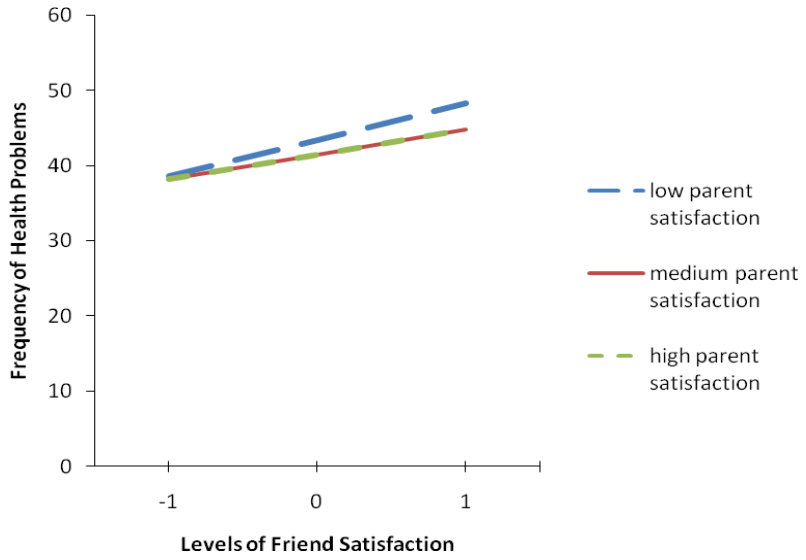


Figure 4.18 Link Between Peer Victimization and Frequency of Health Problems at High and Low Levels of Parent and Friend Satisfaction

Given that the moderating results for friend support were in the opposite direction than I anticipated, I next examined whether the moderating effect held for the positive dimensions of friend social support after controlling for negative dimensions of support. I first ran a MANOVA in which I entered the negative dimensions of friend social support to determine whether victims and nonvictims were different on these dimensions. It is possible that victims are reporting more support from their friends while also being bullied by them (e.g., excluded, dominance). I found an overall victim main effect for the negative dimensions of relationship components with victims reporting more friend dominance, conflict, criticism, and exclusion, $F(7, 99) = 7.33$, $p < 0.01$. Based on the MANOVA results, I controlled for perceived friend dominance, conflict, criticism, and exclusion and then re-examined whether perceived friend disclosure and support moderated the link between victimization and frequency of health problems. Perceived disclosure ($t(7, 99) = 2.74$, $b = 0.03$, $sr^2 = 0.04$, $p < 0.01$) still moderated the link between victimization and frequency of health problems even after controlling for negative friendship dimensions. (The same moderating pattern held even after controlling for the negative friendship dimensions such that higher levels of disclosure exacerbated the link between

disclosure and frequency of health problems.) Perceived friend support marginally moderated the link between victimization and frequency of health problems even after controlling for negative friendship components ($t(7, 99) = 1.76$, $sr^2 = 0.02$, $p = 0.08$). Finally, perceived friend approval marginally moderated the link between victimization and frequency of office visits after controlling for dominance, conflict, criticism, and exclusions ($t(7, 99) = 1.96$, $sr^2 = 0.03$, $p = 0.05$). As such, it appears that this exacerbating influence of positive friend support on the victimization-health link was not solely due to the victims being bullied by their friends.

4.3.3 Person Centered Approach to Examining Social Support to Health Outcomes.

To further examine whether there were differential relationships between support and health outcomes for victims and non-victims, I conducted correlation analyses separately for my victim and non-victim groups (see Table 4.6). I then conducted t-tests for independent correlations to examine if the relationship was different for victims than non-victims (Preacher, 2002; Cohen & Cohen, 1983). There was no consistent evidence that social support acted as a buffer against health outcomes when the adolescent was being bullied by his or her peers (see Table 4.6).

Table 4.6 Correlations Between Dimensions of Social Support and Health Outcomes

Support Dimension	Symptoms	Visits	Symptoms	Visits
	Victims (N = 31)		Nonvictims (N = 76)	
Overall Positive				
Friend	0.28	0.30	-0.04	-0.16
Parent	-0.28	-0.18	-0.13	-0.06
Overall Negative				
Friend	0.03	-0.14	0.25	0.13
Parent	0.23	0.32+[a]	0.12	0.12[a]
Companionship				

Table 4.6. – continued

Friend	0.19	0.19	-0.10	0.09
Parent	-0.30	-0.12	-0.12	-0.06
Disclosure				
Friend	0.39* [a]	0.23	0.03 [b]	0.170
Parent	-0.02	-0.01	-0.02	0.02
Satisfaction				
Friend	-0.04	0.20	-0.14	0.03
Parent	-0.36* [a]	-0.31+ [a]	-0.19+ [a]	-0.13 [a]
Support				
Friend	0.23	0.22	0.09	0.25**
Parent	-0.18	-0.14	-0.02	0.07
Approval				
Friend	0.25	0.39*	-0.14	0.06
Parent	-0.41* [a]	-0.23	-0.25* [a]	-0.18
Pressure				
Friend	0.16 [a]	-0.01	0.23* [a]	0.11
Parent	0.12	0.22	0.06	-0.04
Conflict				
Friend	-0.11	-0.26	0.11	0.07
Parent	0.12	0.19	0.18	0.19
Criticism				

Table 4.6. – continued

Friend	0.07 [a]	-0.16	0.31** [a]	0.10
Parent	0.27	0.32+	0.18	0.10
Dominance				
Friend	0.20	0.20 [a]	0.16	0.21+ [a]
Parent	0.02	0.41*	-0.07	0.03
Exclusion				
Friend	-0.30	-0.27	0.14	0.04
Parent	0.39*	0.09	0.11	0.19

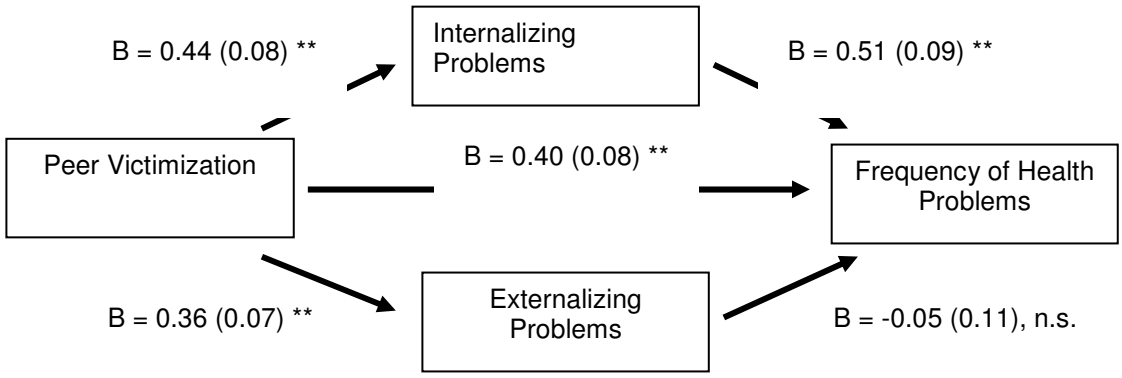
Note: * $p < 0.05$; ** $p < 0.01$

4.4 *Do Adjustment Problems Mediate the Link Between Victimization and Health Outcomes?*

I next examined whether adjustment problems (i.e., internalizing and externalizing problems) mediated the link between victimization and health outcomes. I followed procedures outlined by MacKinnon et al. (2002) and Preacher and Hayes (2008) by using bootstrapping procedures to test for indirect effects with 1000 bootstrap samples. I entered both internalizing and externalizing problems into the mediation model. Internalizing problems mediated the link between victimization and frequency of symptoms (Sobel test: z = 3.34, effect = 0.21, $p < 0.001$, bootstrapping: 95% LL = 0134, 95% UL = 0.37), severity of symptoms (z = 3.34, effect = 0.16, $p < 0.001$, 95% LL = 0.08, 95% UL = 0.26), and frequency of visits to the doctor/nurse (Sobel test: z = 2.76, effect = 0.18, $p < 0.001$, bootstrapping: 95% LL = 0.07, 95% UL = 0.29).

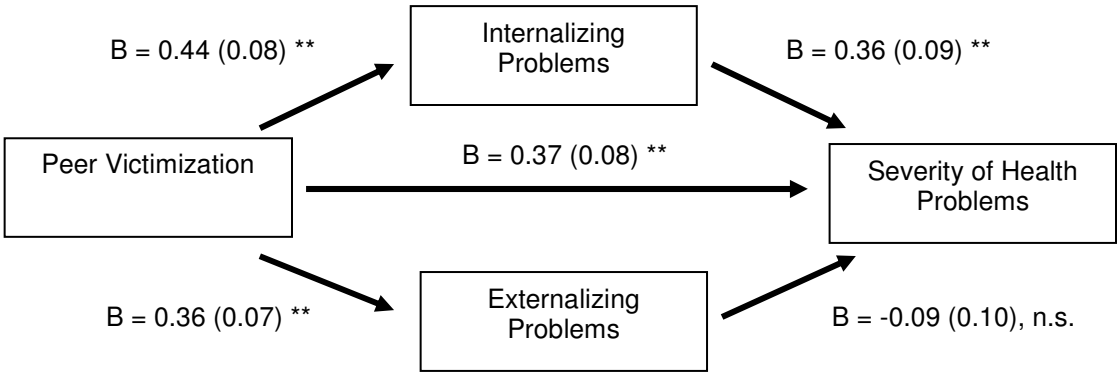
Externalizing problems did not mediate the link between victimization and frequency of symptoms (Sobel test: z = -0.48, effect = -0.02, $p > 0.05$, bootstrapping: 95% LL = -0.12, 95%

UL = 0.07), severity of symptoms (Sobel test: z = -0.86, effect = -0.03, $p > 0.05$, bootstrapping: 95% LL = -0.10, 95% UL = 0.04), frequency of visits to the doctor/nurse (Sobel test: z = 0.17, effect = 0.001, $p > 0.05$, bootstrapping: 95% LL = -0.01, 95% UL = 0.01). In addition, the path meditational path for internalizing problems to health problems was significantly different than the mediation path of externalizing to health for frequency of symptoms (Sobel test: z = 2.38, effect = 0.025, $p < .02$). There were no differences between internalizing and externalizing paths for severity of symptoms (Sobel test: z = 1.38, effect = 0.02, $p = 0.17$) and frequency of doctor/nurse visits (Sobel test: z = 1.26, effect = 0.02, $p = 0.21$). See Figures 4.19, 4.20, 4.21, and 4.22 for mediation models for internalizing problems. Neither internalizing problems nor externalizing problems mediated the link between victimization and abdominal pain (Sobel test: z = -0.36, 0.82, effect = -0.02, 0.05, $p > 0.05$, bootstrapping: 95% LL = -0.16, -0.10, 95% UL = 0.10, 0.20). In sum, I found that internalizing problems partially mediated the link between victimization and health problems (with the exception of abdominal pain reports), but externalizing problems did not mediate the victimization-health link.



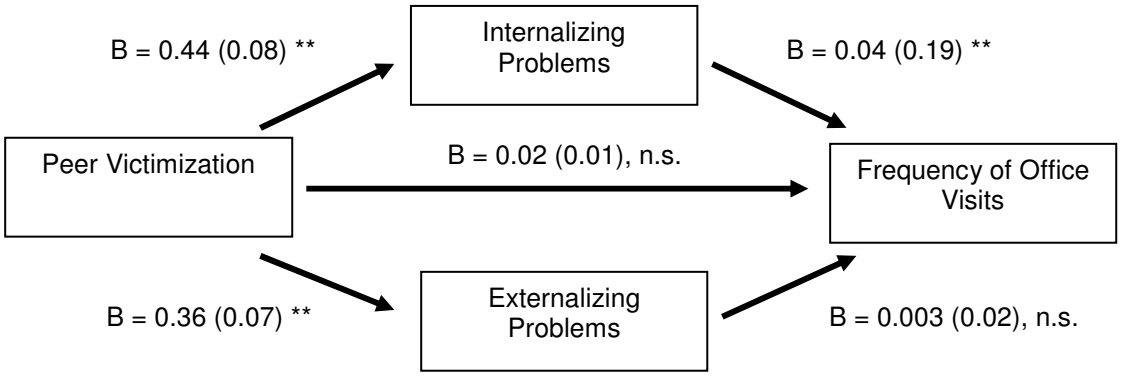
Note: ** $p < 0.01$; numbers in parentheses are standard errors

Figure 4.19 Internalizing Problems Partially Mediate the Link Between Victimization and Frequency of Health Problems



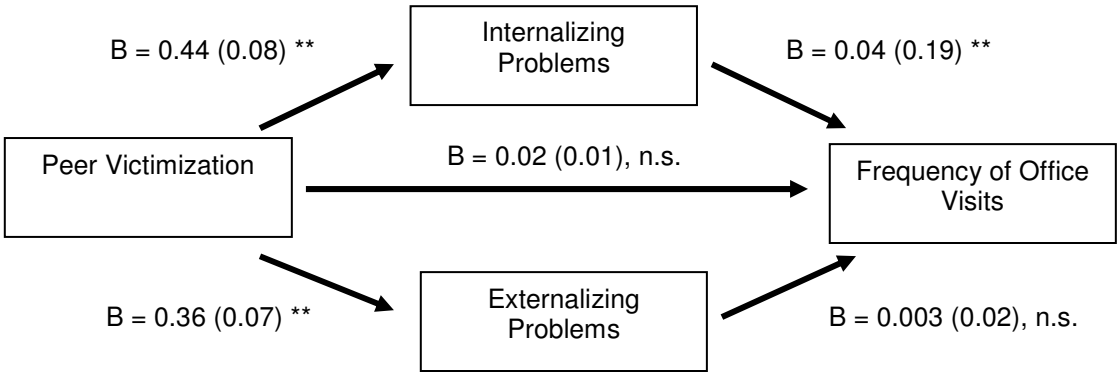
Note: ** $p < 0.01$; numbers in parentheses are standard errors

Figure 4.20 Internalizing Problems Partially Mediates the Link Between Peer Victimization and Severity of Health Problems



Note: ** $p < 0.01$; numbers in parentheses are standard errors

Figure 4.21 Internalizing Problems Partially Mediate the Link Between Victimization and Frequency of Office Visits to the Doctor and/or Nurse



Note: ** $p < 0.01$; numbers in parentheses are standard errors

Figure 4.22 Adjustment Problems Do Not Mediate the Link Between Victimization and Reports of Abdominal Pain

4.5 *Does Being Victimized Influence the Daily Diurnal Pattern of Cortisol Production?*

I anticipated that being peer victimized would influence the diurnal pattern of cortisol. More specifically, it was expected that victimized adolescents would show hypocortisolism or lower daily levels of cortisol than their non-victimized peers, especially in the morning (i.e., a flattening of the diurnal pattern in the cortisol awakening response). Cortisol was collected immediately upon waking, 30 minutes after waking, immediately after school, and 30 minutes before bedtime during two school days. Time points were averaged across the two days. The data were run as mg per deciliter, which is equal to 100 milliliters. The data were first converted to mg/ml by multiplying each data point by 100 and were then log-transformed to normalize the assessments (see Table 4.7 for descriptive statistics).

Table 4.7 Descriptive Statistics for Cortisol Data Before and After Natural Log Transformation

Measure	Range	Min	Max	Mean	SD	Skewness (Std. Error)	Kurtosis (Std. Error)
Before Transforming							
Lab T1	0.71	0.04	0.74	0.12	0.10	3.50 (0.23)	16.36 (0.46)
Lab T2	0.63	0.03	0.67	0.12	0.09	3.24 (0.23)	14.72 (0.46
Lab T3	0.57	0.02	0.60	0.13	0.10	2.42 (0.23)	7.12 (0.46)
Lab T4	0.66	0.03	0.68	0.14	0.11	2.69 (0.23)	8.64 (0.46)
Daily T1	1.49	0.09	1.59	0.35	0.19	2.90 (0.23)	14.87 (0.46)
Daily T2	2.11	0.13	2.24	0.53	0.31	3.02 (0.23)	12.74 (0.46
Daily T3	0.38	0.03	0.41	0.10	0.06	2.52 (0.23)	9.00 (0.46)
Daily T4	0.60	0.01	0.61	0.07	0.08	4.35 (0.23)	22.88 (0.46)
After Transforming							
Lab T1	3.00	3.61	6.61	4.63	0.58	0.80 (0.23)	1.12 (0.46)
Lab T2	3.12	3.37	6.49	4.64	0.54	0.51 (0.23)	1.02 (0.46)
Lab T3	3.21	3.18	6.39	4.70	0.60	0.30 (0.23)	0.44 (0.46)
Lab T4	3.19	3.33	6.53	4.70	0.61	0.54 (0.23)	0.59 (0.46)
Daily T1	2.82	4.55	7.37	5.75	0.47	0.23 (0.23)	0.78 (0.46)
Daily T2	2.83	4.89	7.71	6.16	0.47	0.44 (0.23)	1.50 (0.46)
Daily T3	2.55	3.47	6.01	4.53	0.44	0.67 (0.23)	0.95 (0.46)
Daily T4	3.95	2.47	6.42	3.91	0.72	0.91 (0.23)	1.66 (0.46)
Average Daily	2.28	4.22	6.50	5.09	0.38	0.74 (0.23)	1.81 (0.46)
Average Lab	2.45	3.58	6.03	4.67	0.49	0.30 (0.23)	0.22 (0.46)

Using the person-centered approach, I conducted a repeated measures ANOVA with my victim groups as my independent factor and cortisol levels at four time points as my repeated measures factors. There was a significant cubic effect for the victimization X diurnal

pattern interaction, $F(1, 105) = 5.05$, $p < 0.05$, $\eta^2 = 0.05$, which suggests that the shape of the diurnal pattern differed significantly by victim type. Given the cubic effect, I then ran paired sample t-tests by victimization to examine which sequential assessments were statistically different. For both victimized and nonvictimized adolescents, time 1 (taken immediately upon waking) differed from time 2 (taken 30 minutes after waking; *t*s = -2.94, -8.90, dfs = 30, 74, *p*s < 0.001); time 2 differed from time 3 (taken immediately upon returning home from school; *t*s = 12.63, 28.04, dfs = 30, 75, *p*s < 0.001); and time 3 differed from time 4 (taken 30 minutes before bed; *t*s = 6.18, 7.26, dfs = 30, 75, *p*s < 0.001). I next conducted independent sample t-tests for correlations between the assessments to determine at which points during the day victimized and nonvictimized adolescents evidenced different patterns of cortisol change (Preacher, 2002). No significant differences were found between victimized and nonvictimized adolescents for changes in cortisol between time 1 and time 2 (z = 0.76, $p = 0.22$), time 2 and time 3 (z = 0.09, $p = 0.46$), nor time 3 to time 4 (z = 0.45, $p = 0.33$). See Figure 4.23. Next, I conducted independent sample t-test to determine at which specific points during the day victimized and nonvictimized adolescents evidenced different cortisol levels. I found that victimized adolescents had significantly lower cortisol levels at time 2 (i.e., 30 minutes after waking; $t(105) = 2.71$, $p < 0.01$) and at time 4 (i.e., 30 minutes before bed; $t(105) = 2.03$, $p = 0.045$).

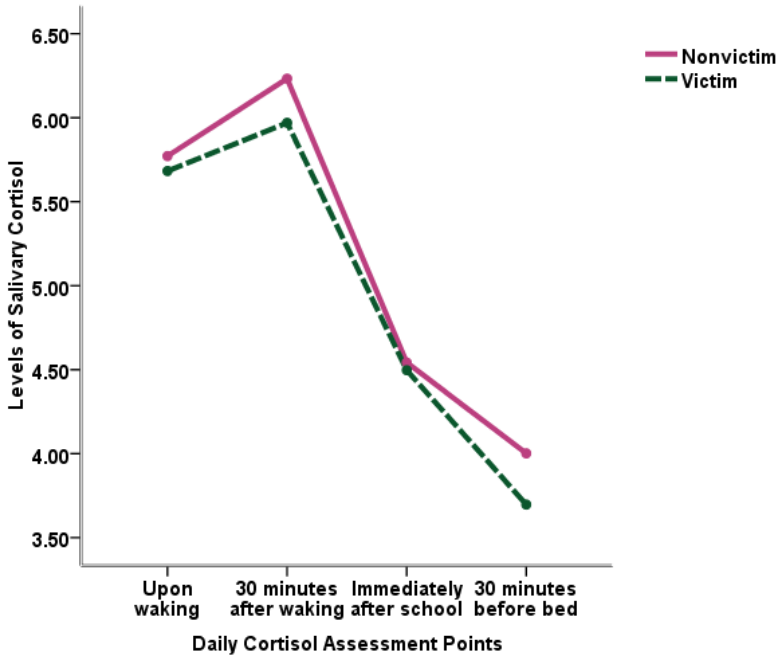


Figure 4.23 Daily Diurnal Pattern of Salivary Cortisol Production for Victimized and Nonvictimized Adolescents

I then split the data file by sex of the participant and reran the above analyses to determine whether the link between victimization and daily cortisol production differed for girls and boys. I found evidence of a cubic effect for victimization over the daily assessments for both boys ($F(1, 45) = 2.86$, $p = 0.10$, $\eta^2 = 0.06$) and girls ($F(1, 58) = 2.29$, $p = 0.14$, $\eta^2 = 0.04$). See Figure 4.24. To dissect this trend, I ran paired sample t-tests. I found that both nonvictimized and victimized boys evidenced significantly different levels of cortisol from time 1 to time 2 (ts = -6.69, -3.98, dfs = 28, 17, ps < 0.01, respectively), time 2 to time 3 (ts = 20.11, 10.48, dfs = 28, 17, ps < 0.001, respectively), and time 3 to time 4 (ts = 5.63, 6.34, dfs = 28, 17, ps < 0.01, respectively). For girls, nonvictimized adolescents evidenced different levels of cortisol from time 1 to time 2 ($t(46) = -6.15$, $p < 0.01$), from time 2 to time 3 ($t(46) = 20.36$, $p < 0.01$), and from time 3 to time 4 ($t(46) = 4.95$, $p < 0.01$). Victimized girls did not evidence differences in cortisol levels between time 1 and time 2 ($t(12) = -1.00$, p =0.34) which indicates a dampened awakening response (i.e., the typical spike in morning cortisol 30 minutes after waking).

Victimized girls did evidence different levels of cortisol between times two and three ($t(12) = 7.12$, $p < 0.01$) and between times three and four ($t(12) = 2.84$, $p < 0.05$). See Figure 4.25.

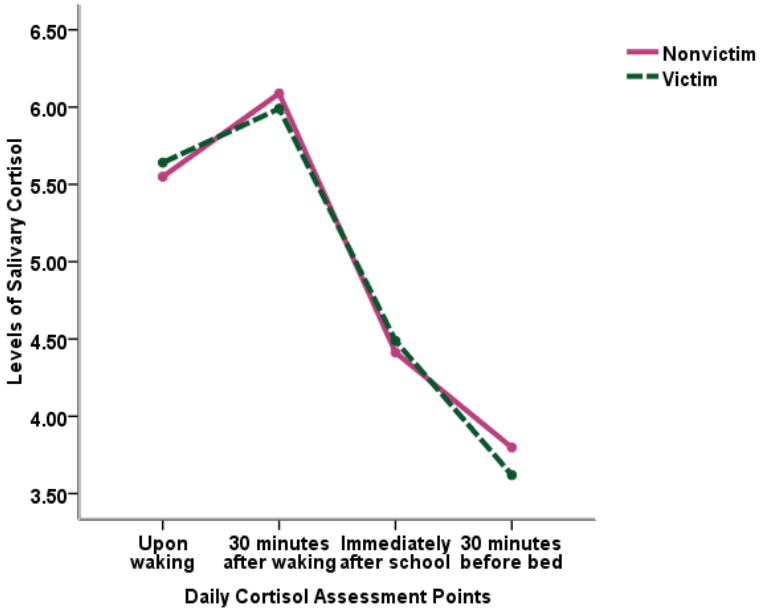


Figure 4.24 Daily Diurnal Pattern of Salivary Cortisol Production for Victimized and Nonvictimized Boys

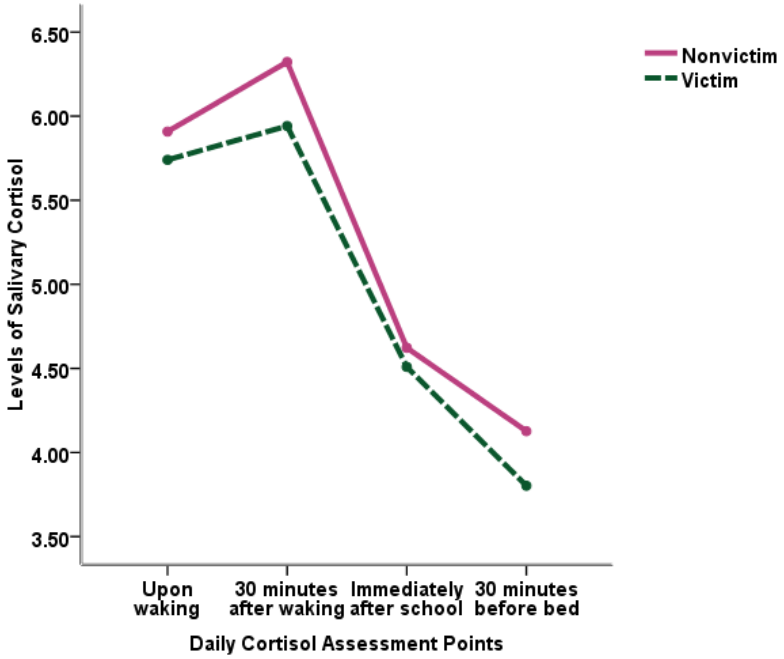


Figure 4.25 Daily Diurnal Pattern of Salivary Cortisol Production for Victimized and Nonvictimized Girls

I then examined whether the link between victimization and change in cortisol production between each assessment point differed for boys and girls. I found that victimized and nonvictimized girls evidenced a significant difference in change in cortisol levels from time 1 (i.e., immediately when wake) to time 2 (i.e., 30 minutes after waking; $z = 1.76$, $p < 0.05$) and a marginally significantly change from time 3 (i.e., immediately after school) to time 4 (i.e., 30 minutes before bed; $z = 1.32$, $p = 0.09$). A similar pattern was found for boys such that victimized and nonvictimized boys evidenced marginally different patterns of cortisol change from time 1 to time 2 ($z = -1.68$, $p = 0.05$) and from time 3 to time 4 ($z = -1.33$, $p = 0.09$). The change in cortisol from time 2 (i.e., 30 minutes after waking) to time 3 (i.e., immediately after school) for victimized and nonvictimized adolescents was not significantly different for girls ($z = 0.23$, $p > 0.05$) or boys ($z = -0.33$, $p > 0.05$). Finally, I ran independent t-tests to determine at which points victimized versus nonvictimized boys and girls differed in cortisol production. No significant differences were found between victimized and nonvictimized boys. However, victimized girls evidenced significantly lower cortisol levels at time 2 (i.e., 30 minutes after waking) than nonvictimized girls ($t(58) = 2.56$, $p < 0.05$). In addition, I found a trend such that victimized girls had lower cortisol levels at time 4 than nonvictimized girls ($t(105) = 1.38$, $p = 0.17$).

4.6 *Do Daily Cortisol Levels Predict Self-Reported Health Outcomes?*

I expected that daily cortisol would predict self-reported health outcomes. I first sought to determine whether average daily cortisol levels (i.e., area under the curve) predicted health outcomes. I did not find any significant links between average daily cortisol and frequency of health problems ($t(105) = 0.74$, $p = 0.46$), severity of health symptoms ($t(105) = 0.57$, $p = 0.57$), frequency of visits to the doctor/nurse ($t(105) = 0.057$, $p = 0.57$), nor abdominal pain ($t(105) = 0.60$, $p = 0.55$).

Although the expected link between average daily cortisol and health problems was not found, research has recently suggested the importance of looking at patterns of cortisol

production during the day (i.e., looking at particular assessments of cortisol throughout the day in relation to the other assessment points). For example, Hansen et al. (2006) found that bullied workers had lower morning cortisol levels; indeed, hypocortisolism has been linked with maltreatment and post traumatic stress disorder (e.g., Heim et al., 2000). As such, I examined whether specific assessments of daily cortisol predicted health outcomes. I expected that lower levels of cortisol at time 2 (i.e., 30 minutes after waking) would predict more health problems since lower levels would suggest a dampened cortisol awakening response. Researchers have suggested that a flattening of the diurnal pattern (i.e., hypocortisolism) is associated with poor health outcomes. Therefore, it may be that lower levels in the morning accompanied by higher levels in the evening would predict poorer health outcomes.

To examine this hypothesis, I ran a regression model in which I controlled for age and sex of the participant on step 1. These controls were entered because previous research has found that cortisol is influenced by sex and age (e.g., Vaillancourt et al., 2008). I then entered the four daily cortisol assessments on step 2. As predicted, I found that lower levels of cortisol at time 2 (i.e., 30 minutes after waking) predicted more health problems in general, ($t(100) = -1.67$, $p = 0.099$, $sr^2 = 0.03$) and more frequent office visits ($t(100) = -1.83$, $p = 0.07$, $sr^2 = 0.03$). I also found that higher levels of cortisol at time 4 (i.e., 30 minutes before bed) predicted more frequent health problems ($t(100) = 1.96$, $p = 0.05$, $sr^2 = 0.04$). See Figures 4.26, 4.27, and 4.28. Although the findings were marginal, the pattern of results suggests that lower morning cortisol as well as higher evening cortisol may be associated with poorer health outcomes. No significant differences were seen for boys versus girls.

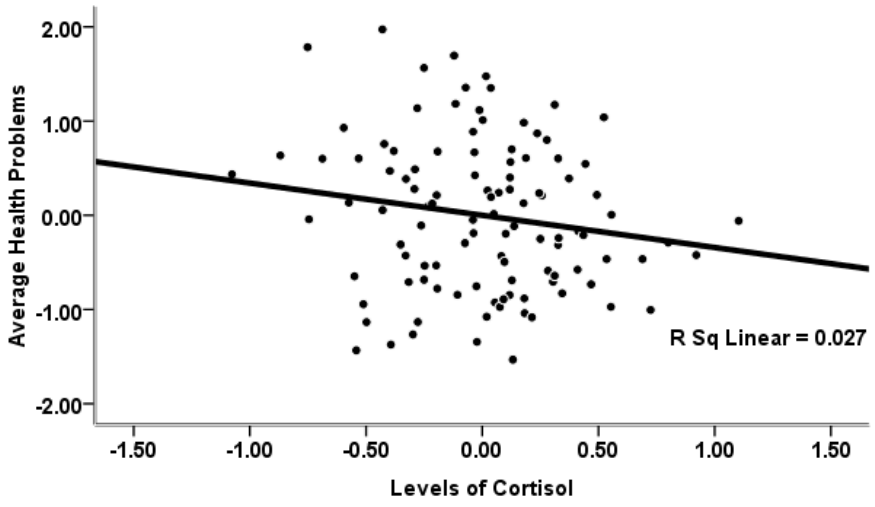


Figure 4.26 Daily Cortisol Levels 30 Minutes After Waking Predict Average Health Problems

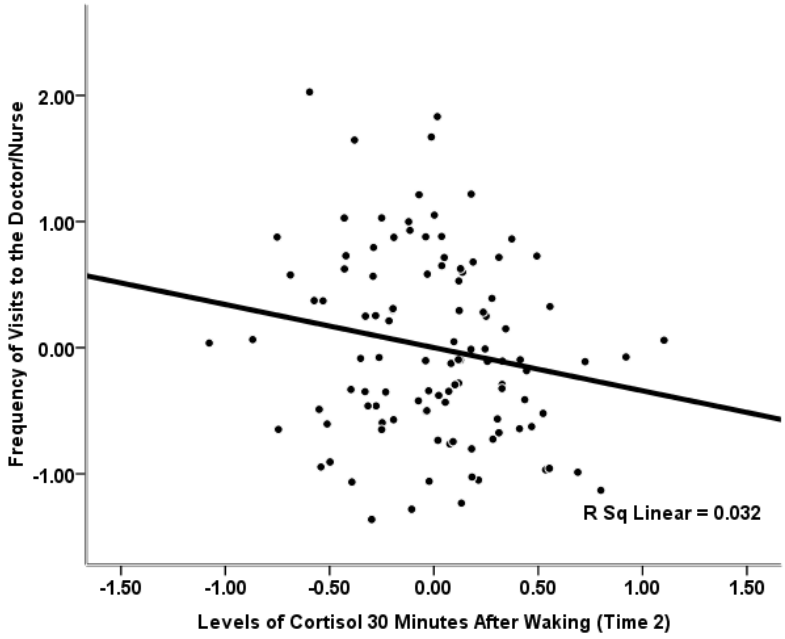


Figure 4.27 Daily Cortisol Levels 30 Minutes After Waking Predict Frequency of Visits to the Doctor/Nurse

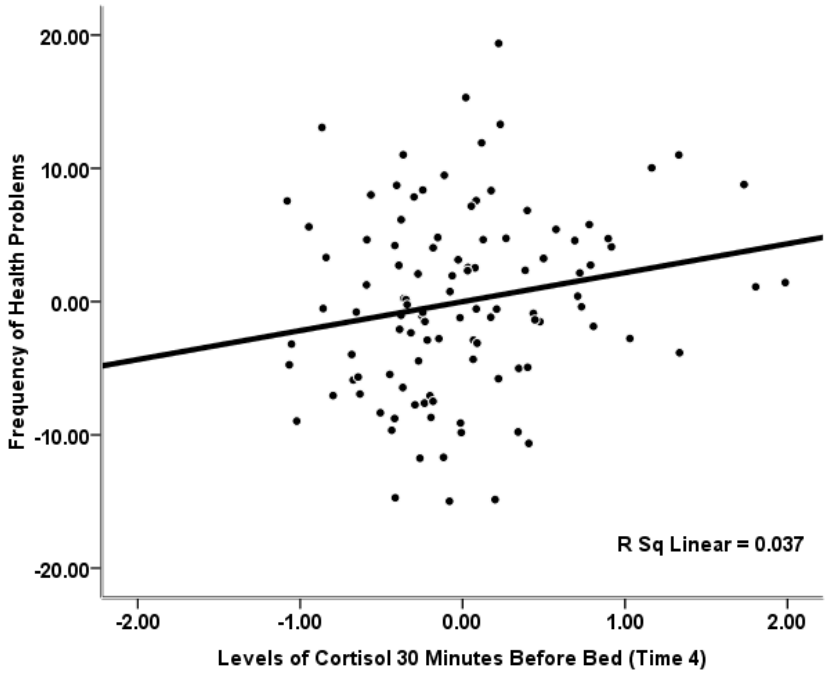


Figure 4.28 Levels of Daily Cortisol 30 Minutes Before Bed Predict Frequency of Health Problems

4.7 *Do Daily Cortisol Levels Mediate the Victimization-Health Link?*

I also predicted that overall daily cortisol levels would mediate the link between victimization and health. To test this prediction, I conducted Sobel tests and utilized bootstrapping procedures as outlined by MacKinnon et al. (2002). I did not find evidence that average daily cortisol levels mediated the link between victimization and frequency of health problems (Sobel test: z = -0.81, p = 0.42; bootstrapping: LL 95% = -0.53, UL 95% = 0.10), severity of health problems (Sobel test: z = -0.74, p = 0.46; bootstrapping: LL 95% = -0.53, UL 95% = 0.10), frequency of visits to the doctor/nurse (Sobel test: z = -0.61, p = 0.54; bootstrapping: LL 95% = -0.05, UL 95% = 0.02), nor abdominal pain (Sobel test: z = -0.68, p = 0.50; bootstrapping: LL 95% = -0.65, UL 95% = 0.18).

Next, I examined whether cortisol at different time points throughout the day carried the influence of victimization to health problems using procedures outlined in Preacher and Hayes (2008). Sex of participant, age of participant, and internalizing problems were entered as

controls in the model. There was no evidence of mediation for frequency of health visits (e.g., total indirect effect: point estimate = 0.03, SE = 0.04, z = 0.83, n.s) or frequency of health symptoms (e.g., total indirect effect: point estimate = -0.01, SE = 0.04, z = -0.25, n.s.).

4.8 *Does Being Victimized Influence Self-Reported Reactions to the TSST?*

I first examined the correlations between victimization and self-reported reactions to the TSST. I found significant correlations between victimization and viewing the speech as challenging ($r = 0.28$, $p < 0.01$) and wanting to give up/quit ($r = 0.34$, $p < 0.01$). That is, more victimized adolescents viewed the speech as more challenged and reporting wanting to give up/quit more than less victimized adolescents. I also found a negative correlation between victimization and reports of feeling accepted ($r = -0.20$, $p < 0.05$) such that the more victimized an adolescent was, the less accepted he/she felt. Furthermore, I did not find a significant correlation between victimization and feeling out of control ($r = -0.18$, $p = 0.07$) or feeling evaluated during the delivery of the speech ($r = 0.09$, $p = 0.37$). In other words, both victimized and nonvictimized adolescents felt similarly evaluated during their speech delivery and perceived similar degrees of control. In addition, I examined whether boys and girls significantly differed in their self-reported reactions to the TSST. Indeed, I found significant differences such that girls reported feeling less accepted, rated more poorly, found the task more threatening, and more rejected. (See Table 4.8 for correlations between victimization and the post-TSST questions.)

Table 4.8 Correlations Between Victimization and Self-Reported Reactions to the TSST

Item/Composite from Post-TSST Survey	Overall Correlation	For Girls (n = 60)	For Boys (n = 47)
Accepted (composite)	-0.20*	-0.37**[a]	-0.05[b]
Stressful (composite)	0.28**	0.35**[a]	0.22[a]
Evaluated during task	0.09	0.07	0.10
Wanted to give up/quit	0.34**	0.44**[a]	0.23[a]
Stressful (item)	0.28**	0.31*[a]	0.26+[a]
Effectiveness	-0.18+	-0.14	-0.20
Performance rated	-0.10	-0.27*[a]	0.08[b]
Difficult	0.10	0.03	0.15
Effort/tried hard	0.12	0.19	0.4
Felt confident	-0.20*	-0.18	-0.21
Control	-0.18+	-0.28*[a]	-0.10[a]
Performance	-0.15	-0.27*[a]	-0.05[a]
Threatening	0.07	0.35**[a]	-0.16[b]
Challenging	0.22*	0.34**[a]	0.10[a]
Gave up/quit trying	0.25*	0.36**[a]	0.13[a]
Evaluated at later time	0.02	-0.31	0.05
Felt liked	-0.16	-0.19	-0.13
Accepted	-0.21*	-0.40**[a]	-0.01[b]
Disliked	0.17	0.31*[a]	0.07[a]
Rejected	0.14	-0.38**[a]	-0.04[b]

Note: * $p < 0.05$; ** $p < 0.01$

Second, using a MANOVA with victimization and sex entered into the equation, I examined whether self-reported reactions to the TSST were predicted by peer victimization. Indeed, there was an overall effect for victimization ($F(5, 99) = 2.33$, $p < 0.05$, Pillai's Trace = 0.11). Given the significant effect of the model, I then conducted Roy-Bargmann's step-down ANOVA procedure to further examine which of my dependent variables differed between victimized and nonvictimized adolescents. I entered the dependent items in order of theoretical significant: stress, acceptance, evaluated, wanting to give up, and feeling in control. I found that both reports of feeling stressed and wanting to give up/quit were predicted by victimization (Fs(1, 103/100) = 5.16, 6.10, ps < 0.05, 0.05, η^2s = 0.05, 0.06).

4.9 *Does Being Victimized Influence Cortisol Reactivity During the TSST?*

Next I examined whether victimization predicted levels of cortisol during the Tier Social Stress Test (TSST). Salivary cortisol was assessed at four time points during the TSST, namely 10 minutes after arrival (i.e., after resting 10 minutes), after a 10 minute speech preparation period, immediately after delivering the speech, and 30 minutes after giving the speech. The first assessment served as a baseline measure of participants' salivary cortisol. Using a person-centered approach, I conducted a repeated measures analysis to determine whether victimization predicted cortisol levels across the TSST time points. There was a significant quadratic victimization by assessment interaction ($F(1, 105) = 5.28$, $p < 0.05$, $\eta^2 = 0.05$) indicating that the pattern of cortisol production throughout the TSST differed for victimized and nonvictimized adolescents (see Figure 4.29). To determine where the significant change occurred, I next conducted paired sample t-tests in which I examined whether levels of sequential cortisol samples differed (i.e., time 1 versus time 2, time 2 versus time 3, time 3 versus time 4). For victimized adolescents, time point four was significantly lower than time point three ($t(30) = 2.71$, $p < 0.05$). No significant differences were observed for nonvictimized adolescents. I then ran t-tests for independent correlations to determine whether the relationship between time 3 and time 4 differed for victimized versus nonvictimized adolescents

(Preacher, 2002; Cohen & Cohen, 1983). Although not significant, the trend suggested that victimized adolescents had a steeper drop in cortisol from time 3 to time 4 than did nonvictimized adolescents (z = -0.87, p = 0.19).

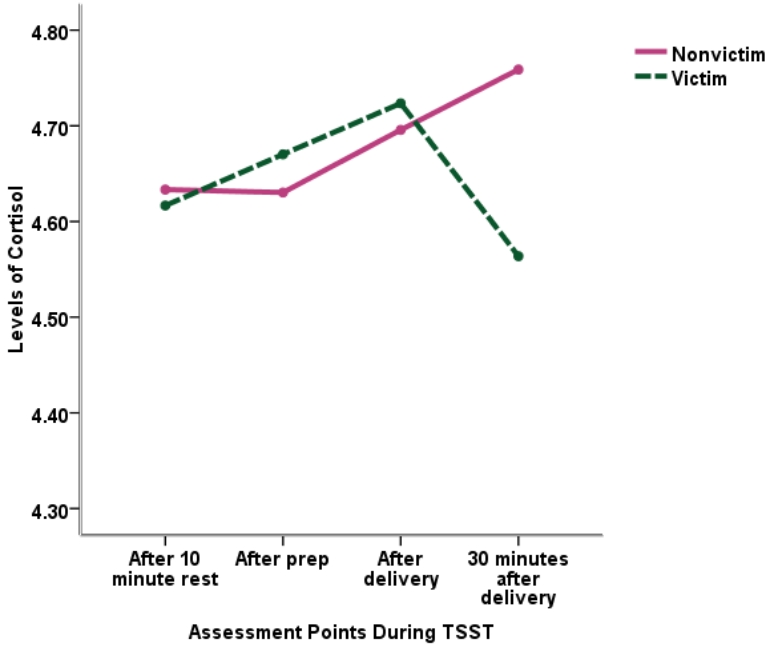


Figure 4.29 Cortisol Reactivity for Victimized and Nonvictimized Adolescents During the TSST

Since the initial in-lab assessment served as a baseline measure of cortisol, I regressed time 1 onto time 2, time 3, and time 4 in order to control for individual differences in initial levels of salivary cortisol. (Note: victimization did *not* predict levels of cortisol at time one, $t(105)$ = 0.23, p = 0.82.) When I ran my repeated measures ANOVA controlling for levels of cortisol at time 1, I found a significant interaction between victimization and assessment points ($F(2, 104)$ = 3.54, $p < 0.05$, η^2 = 0.06, Pillai's Trace = 0.06). More specifically, I found a significant linear trend ($F(1, 105)$ = 4.80, $p < 0.05$, η^2 = 0.04) and a marginal quadratic trend ($F(1, 105)$ = 3.09, p = 0.08, η^2 = 0.03). I then ran paired sample t-tests again to determine where the differences were located. As above, I found that the last two assessments differed significantly for victimized adolescents ($t(30)$ = 2.79, $p < 0.01$) but not for nonvictimized adolescents ($t(75)$ = -1.32, p = 0.19). Finally, I ran t-tests for independent correlations to examine whether the change

between the 3rd and 4th assessments differed for victims versus non-victims. As found above, the relationships were not significantly different between victimized versus nonvictimized adolescents (z = -0.88, $p = 0.38$).

I next examined whether the link between victimization and cortisol reactivity differed for boys versus girls. For boys, there is a trend (although not significant) for a quadratic effect indicating that cortisol reactivity may differ for victimized versus nonvictimized boys ($F(1, 45) = 1.37$, $p = 0.25$, $\eta^2 = 0.03$; see Figure 4.30). For girls, I found a evidence of a quadratic trend suggesting that cortisol reactivity differs for victimized and nonvictimized girls ($F(1, 58) = 3.97$, $p = 0.05$, $\eta^2 = 0.06$; see Figure 4.31). I then ran paired sample t-tests to determine where the significant differences were in the trends. Both boy victims ($t(17) = 1.64$, $p = 0.12$) and girl victims ($t(12) = 2.34$, $p < 0.05$) evidenced a change in cortisol from time 3 (i.e., immediately after giving their speech) to time 4 (30 minutes after giving their speech).

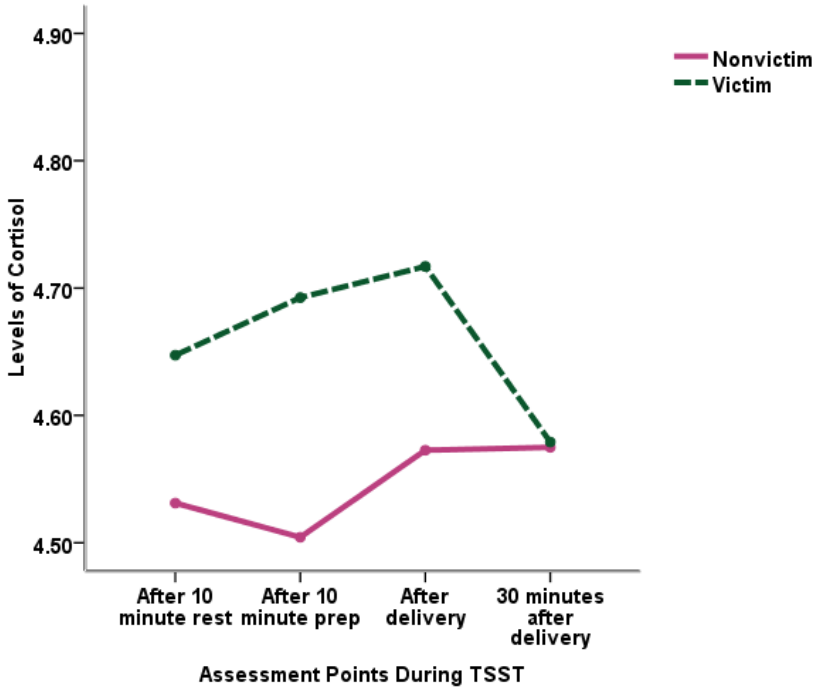


Figure 4.30 Cortisol Reactivity for Victimized and Nonvictimized Boys During TSST

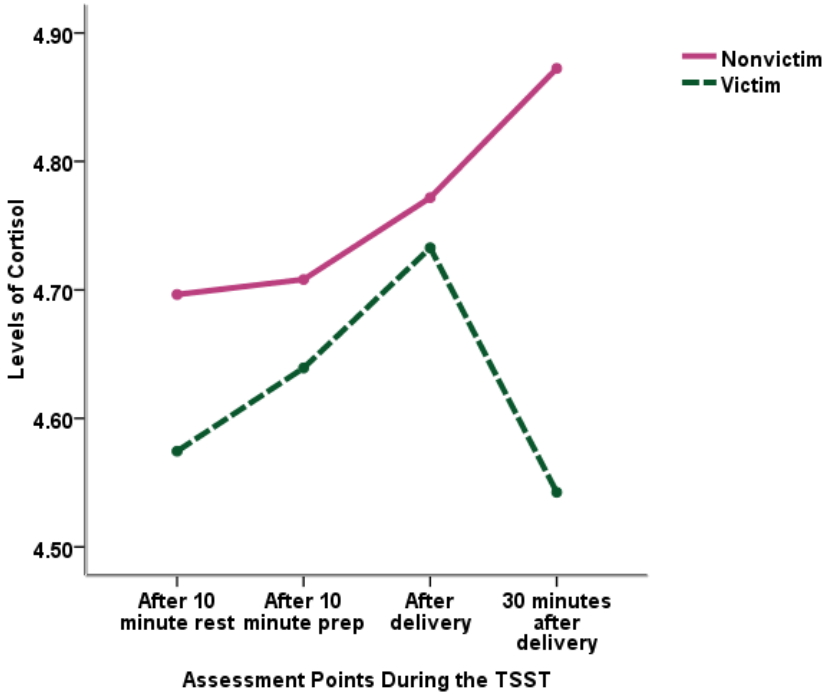


Figure 4.31 Cortisol Reactivity for Victimized and Nonvictimized Girls During the TSST

I then used a variable centered approach to examine whether victimization predicted average cortisol production during the TSST (i.e., area under the curve). Surprisingly, victimization did not predict average cortisol production even after controlling for sex of the participant, ($t(2, 104) = -0.15$, $p = 0.89$, $sr^2 < 0.01$). Since I was expecting that victimization would differentially predict average cortisol production during the TSST for boys and girls, I split my data set by sex and reran the regression analyses. Although not significant, I found trends suggesting that during an acute social stressor (i.e., the TSST) victimization predicted lower average cortisol for girls ($t(58) = -1.47$, $p = 0.15$, $sr^2 = 0.04$) and higher average cortisol for boys ($t(45) = 1.17$, $p = 0.25$, $sr^2 = 0.03$). See Figure 4.32.

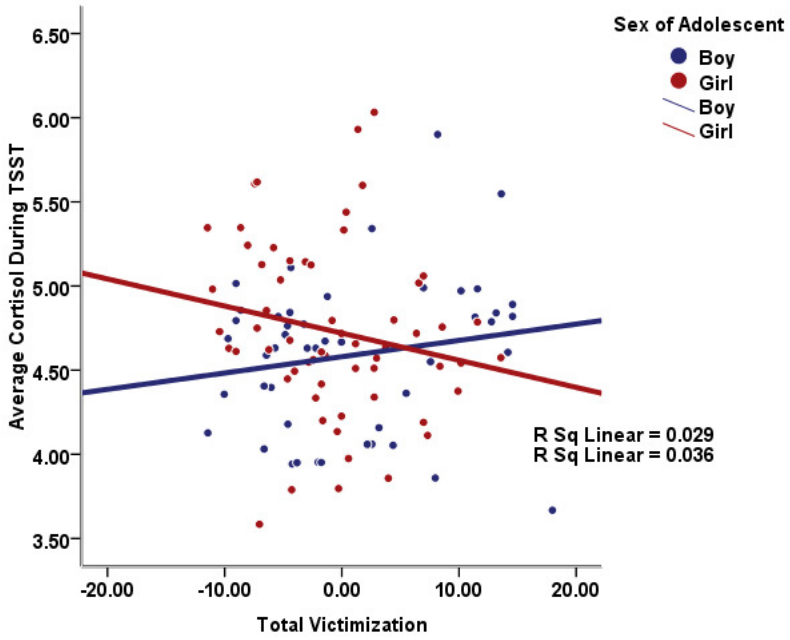


Figure 4.32 Victimization Predicts Average Cortisol Levels During an Acute Social Stressor for Boys and Girls

4.10 *Does Cortisol Reactivity During an Acute Social Stressor Predict Self-Reported Health Outcomes?*

I then examined whether adolescents who were more reactive to an acute social stressor (i.e., TSST) would exhibit more health problems. In other words, these children may be less able to cope with daily social stressors, which would in turn lead to poorer health outcomes. To examine this hypothesis, I ran a second regression model where I entered age, sex, and average daily cortisol on step 1 and entered the four lab assessment points on step 2. First, I found that cortisol levels at time 3 (immediately after delivering their speech) predicted more health problems ($t(99) = 1.68$, $p = 0.097$, $sr^2 = 0.03$) whereas cortisol levels at time 4 (30 minutes after delivering their speech) negatively predicted health problems ($t(99) = -2.21$, $p = 0.03$, $sr^2 = 0.05$). Second, I found that higher cortisol levels at time 3 predicted more frequency health problems ($t(99) = 2.28$, $p < 0.05$, $sr^2 = 0.05$) whereas lower cortisol levels at time 4 predicted more frequency health problems ($t(99) = -2.16$, $p < 0.05$, $sr^2 = 0.04$). Third, I found that higher cortisol levels at time 3 predicted more frequent visits to the doctor/nurse ($t(99) =$

2.54, $p < 0.01$, $sr^2 = 0.06$); lower levels of cortisol at time 4 predicted more frequent office visits ($t(99) = -2.89$, $p < 0.01$, $sr^2 = 0.07$).

4.11 *Does Cortisol Reactivity Mediate the Link between Victimization and Health?*

Because victimization was related to cortisol reactivity and cortisol reactivity in turn was related to health outcomes, it is possible that cortisol reactivity mediates the link between victimization and health (Baron & Kenny, 1986; Kenny, Kashy, & Bolger, 1998). In other words, victimization could affect health indirectly via cortisol reactivity to acute social stressors. I again used procedures outlined in Preacher and Hayes (2008) to examine whether cortisol at different points during the TSST mediated the link between victimization and health problems. In this analysis, victimization was the predictor, the four lab cortisol assessments were entered as mediators, and frequency of health visits was entered as the outcome measure. In addition, age, sex, daily cortisol levels, and internalizing problems were entered as control variables.

Although there was no overall total indirect effect (across the four mediators), recovery cortisol levels (i.e., 30 minutes after the speech) marginally mediated the victimization-health effect when controlling for the other three lab assessments of cortisol (See Table 4.9). Preacher and Hayes (2008) note that it is not only possible but acceptable to find specific indirect effects even when the total indirect effect is nonsignificant. This finding suggests that victimization was related to lower recovery cortisol levels which, in turn, led to more visits to health professionals (See Figure 4.33). There was no evidence of a mediation effect of cortisol reactivity for frequency or severity of symptoms.

Table 4.9 Cortisol Reactivity Mediates the Link Between Peer Victimization and Frequency of Health Visits

			Bootstrapping		
			Product of Coefficients	Percentile 95% CI	
	Point Estimate	SE	Z	Lower	Upper
Total	0.0046	0.0045	1.02	-0.0035	0.0142
Lab Arrival	-0.0012	0.0022	-0.55	-0.0082	0.0012
After Prep	-0.0005	0.0025	0.20	-0.0100	0.0026
After Speech	-0.0004	0.0042	0.09	-0.0104	0.0073
30 min after Speech	0.0068	0.0051	1.33+	-0.0002	0.0202
Arrival vs. Prep	-0.0007	0.0034	0.20	-0.0081	0.0068
Prep vs. after Speech	-0.0001	0.0058	0.02	-0.0152	0.0094
After Speech vs. 30 min after Speech	-0.0072	0.0080	0.90	-0.0282	0.0038

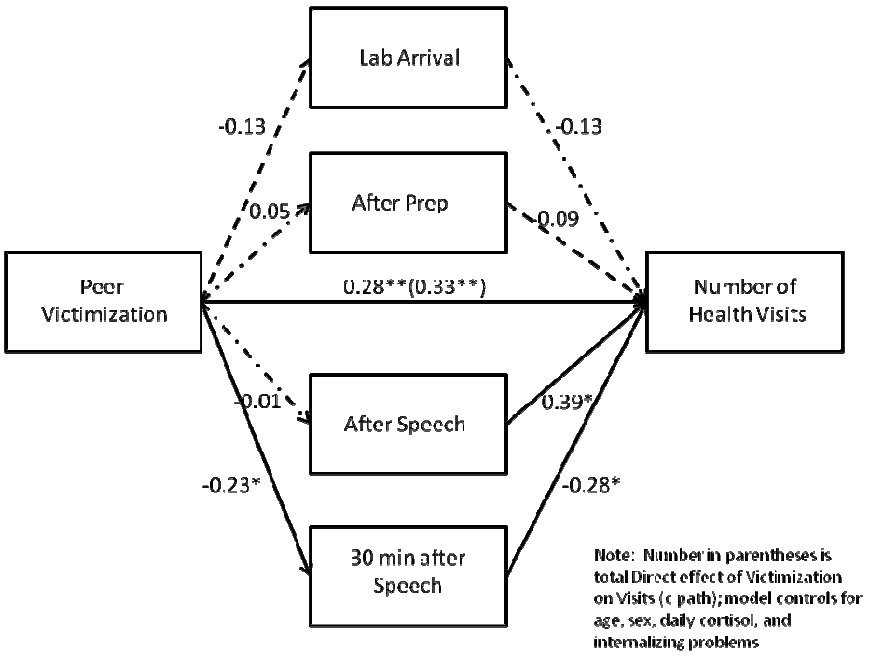


Figure 4.33 Do Cortisol Levels During an Acute Social Stressor Mediate the Link Between Victimization and Visits to the Doctor/Nurse?

4.12 *Supplementary Analyses*

Finally, I explored the possibility that the diurnal pattern may be different for victimized adolescents with and without health problems. To group individuals into health groups, I again conducted a hierarchical cluster analysis and found that there were two health groups. Next, I examined the clustering of participants by victimization and health. There were 51 adolescents who reported having good health and 56 adolescents who reported stress-related health problems. Of the 51 adolescents who reported having good health, only 3 were classified as victims (compared to the 28 victims who reported stress-related health problems). That is, over 90% of victims reported having stress-related health problems. Conversely, only 36% nonvictimized children reported have stress-related health problems. This differential pattern of health problems for victims and nonvictims was statistically significant, $\chi^2(1) = 25.25$, $p < 0.001$.

Next, I examined whether healthy victims had different diurnal patterns than victims who reported stress-related health problems. Indeed, there was an overall victim X time quadratic effect, $F(1, 29) = 4.80$, $p < 0.04$, $\eta^2 = 0.14$. This finding should be interpreted with extreme

caution given the very small N for the health victims (N = 3). However, these differences further suggest that victimized children with health problems may have a flattening of their diurnal pattern (see Figure 4.34).

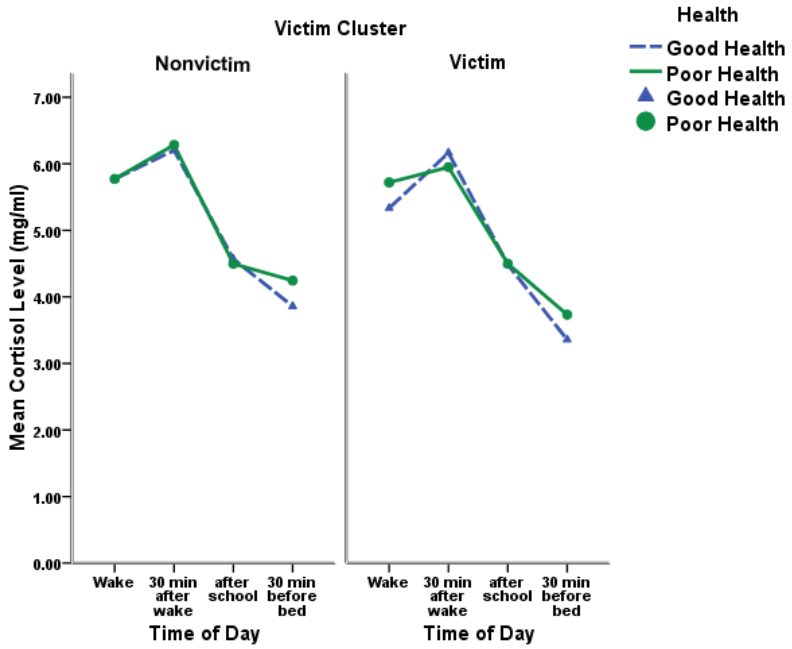


Figure 4.34 Does the Daily Diurnal Pattern Differ Among Victimized and Nonvictimized Adolescents Depending on Overall Health?

CHAPTER 5

DISCUSSION

The current dissertation was a first step in trying to more fully understand the link between peer victimization and poor health outcomes. First, I examined whether peer victimization predicted poor health outcomes (i.e., frequency of health problems, severity of health problems, frequency of visits to the doctor/nurse, and reports of abdominal pain) in adolescents. Second, I tested whether sex of the adolescent and social support from close others moderated the link between victimization and poor health. Third, I examined whether adjustment problems mediated the link between victimization and health problems. Fourth, I examined whether peer victimization predicted daily diurnal patterns of cortisol and whether these patterns predicted self-reports of health outcomes. I then directly examined whether daily cortisol levels mediated the link between victimization and self-reported health outcomes. Fifth, I examined whether victimization influenced self-reported reactions and cortisol reactivity during an acute social stressor. Sixth, I examined whether cortisol reactivity during an acute social stressor predicted self-reported health outcomes. Finally, I examined whether cortisol reactivity mediated the link between victimization and health. The overarching goal of this dissertation was to examine potential reasons *why* there is a link between peer victimization and physical health problems.

5.1 *The Link Between Peer Victimization and Physical Health Outcomes*

Previous research has estimated that 10 – 30% of American children repeatedly experience peer victimization (Grills & Ollendick, 2002; Haynie et al., 2001; Limber & Small, 2003; Nansel et al., 2001; Perry et al., 1998). In my sample, 29% of adolescents (n = 31) were classified as being victimized. That is, 29% of my participants reported being a frequent target

of their peers' aggression across multiple measures of victimization (i.e., overt, relational, indirect, verbal, physical). This finding is alarming given the negative outcomes associated with being bullied. A wealth of research has examined how victimization negatively impacts psychological and mental health problems such as loneliness, depression, and anxiety (e.g., Olweus, 1993; Bjorkqvist et al., 1982; Kochenderfer-Ladd & Ladd, 2001). However, only recently has research begun to find a link between victimization and physical health problems (Rigby, 1998; Williams, Chambers, Logan, & Robinson, 1996; Wolke, Woods, Bloomfield, & Karstadt, 2001). This dissertation attempted to replicate and extend these findings between victimization and poor physical health.

As expected, increased peer victimization was positively associated with greater frequency and severity of health problems, more visits to health professionals, and more reports of abdominal pain. Notably, peer victimization accounted for large amounts of the variance in health problems. Indeed, peer victimization accounted for 34% of the variance in frequency of health problems, 30% of the variance in severity of health problems, 12% of the variance in frequency of visits to health professionals, and 21% of the variance in reports of abdominal pain. Moreover, this study found that both boys and girls who are victimized are equally at risk for experiencing increased health problems. The lack of a moderating effect for sex on the link between victimization and health should not be taken as evidence that such moderation does not exist given the small sample of victimized boys (n = 18) and victimized girls (n = 13). However, the data provide strong evidence of a robust link between victimization and poor health.

This victimization-physical health link is important. In addition to being more unhappy at school, wishing to avoid school more, and having a higher absentee rate than nonvictimized children (e.g., Boulton & Underwood, 1992; Olweus, 1993; Kochenderfer & Ladd, 1996), victimized adolescents report more health problems and more visits to their doctor/nurse than their nonvictimized peers. This finding suggests that victimized children may not be avoiding

school simply because they want to avoid interacting with their peers. Rather, their absences from school may be due in part to actual illnesses.

5.2 *Is the Link Between Victimization and Poor Health Outcomes Moderated by Perceived Social Support?*

Not all victimized children are equally at risk for developing health problems. Indeed, the literature on social support and health in adults suggests that social support buffers against negative health outcomes (DeLongis & Holtzman, 2005; Knack, Waldrip, & Jensen-Campbell, 2007). Positive social support (especially from friends) has also previously been found to buffer against future psychological and mental health outcomes whereas the absence of social support has previously been found to exacerbate future psychological and mental health outcomes. For example, Hodges et al. (1999) found that more adjustment problems were seen in victimized adolescents who did not have a best friend compared to victimized adolescents with a best friend. Davidson and Demarary (2007) found that social support from close friends moderated the link between victimization and internalizing problems such that victimized students with high support evidenced lower internalizing problems compared to those with lower levels of support. Taken together with the adult literature on support and physical health, I anticipated that positive dimensions of social support would buffer adolescents against health problems whereas negative dimensions of social support would exacerbate the link between victimization and health problems.

While I did find moderating effects for support from two best friends, the results were in the opposite direction of what I expected. Specifically, I found that support from close friends exacerbated the link between victimization and health outcomes. I found that the amount of intimate information adolescents disclosed to their two best friends mattered most for victimized adolescents. Disclosing more intimate information when victimized was linked with a higher frequency of health problems whereas less intimate disclosure was linked with less frequent health problems. Amount of disclosure did not matter for nonvictimized adolescents (i.e., nonvictimized adolescents reported similar levels of health problems regardless of how much

they disclosed to their friends). In addition, I found that higher levels of approval from close friends predicted more visits to the doctor/nurse for victimized adolescents than lower levels of approval. Again, perceived friend approval did not matter for nonvictimized adolescents. Finally, the less conflict victimized adolescents perceived in their relationship predicted more visits to health professional than higher levels of conflict. Level of conflict did not significantly influence the link between victimization and health at low levels of victimization. On the other hand, the degree to which parents excluded their child buffered against the number of health problems the child experienced. That is, victimized adolescents who perceived that their parents exclude them a lot reported more frequent health problems than victimized adolescents whose parents did not exclude them.

Given the body of literature suggesting beneficial effects from peer support, these results that peer support exacerbates health problems were surprising. However, victimized adolescents may (1) have lower perceptions of support from their friends; (2) have friends who cannot effectively offer support; (3) cannot effectively utilize support from friends; and (4) may have friends who are also victimized or who are bullies. Another possibility is that the positive effects from peer support may not have manifested yet. Demaray and Malecki (2003) have found that victimized adolescents perceived less social support than both non-victims and bullies but rated support from classmates as being more important than non-victims and bullies rated it. Moreover, Gini (2008) found that victimized adolescents are less satisfied by their friendships than nonvictimized adolescents. Although my social support questionnaire assessed how often adolescents disclose intimate information to their close friends, I did not assess the outcome of the disclosure (i.e., friends' reaction, how supportive the friend is after disclosing information). It is possible that even though victimized adolescents are sharing a lot of intimate information with their friends, their friends are not responding to the information in helpful ways. Perhaps worse, if their friends are also bullies, the disclosed information may be used to further victimize the adolescent.

It is also feasible that victimized adolescents who disclose a lot of information engage in co-rumination with their close friends. Co-rumination is frequently defined as over-talking problems with friends. Rose, Carlson, and Waller (2007) suggested that girls engage in co-rumination as a way of seeking and offering support. However, they found that while co-rumination led to increased feelings of closeness in relationships, it also led to heighten depression and anxiety for girls (but not boys). My finding that self-disclosure negatively moderates the link between victimization and visits to the doctor suggests that high levels of self-disclosure have negative health effects on victimized adolescents.

Malecki and Demaray (2003) examined whether the form of social support (i.e., emotional, appraisal, instrumental, informational) predicted outcome measures such as social skills, problems behaviors, emotional symptoms, and personal and school adjustment in students in 5th through 8th grades. They found that close friend support did not predict any of the outcome variables. Malecki and Demaray also found that adolescents placed the least value on appraisal from close friends. Moreover, they found that adolescents typically perceive support in the form of appraisal less often than other types of support from close friends. Interestingly, I found that approval from close friends moderated the link between victimization and the number of visits to the doctor/nurse. Specifically, victimized adolescents who perceived higher levels of approval from close friends reported more frequent visits to health care professionals compared to victimized adolescents who perceived lower levels of approval. My findings are interesting in light of the findings of Malecki and Demaray. According to their findings, appraisal from close friends did not predict outcome measures commonly linked with peer victimization (e.g., internalizing and externalizing problems, school adjustment). However, my research is suggesting that high levels of approval from close friends predict the number of visits to the doctor/nurse for victimized adolescents. It is possible that, in light of their experiences of victimization, approval from their close friends is especially salient. This saliency, however, does not appear to be protective but is instead associated with more negative health outcomes.

Another potential reason for the unexpected moderating effects of social support may be due to who is providing the social support. Pellegrini, Bartini, and Brooks (1999) found that victimized adolescents tend to be friends with a mix of students including those who are bullies, victims, and neither victims or bullies. Hodges, Malone, and Perry (1997) also found that victimized adolescents are more likely to be friends with other victims. Moreover, the more victimized the friend was, the stronger the link was between the friend's internalizing and externalizing problems and the target-child's levels of victimization. Thus, the unexpected moderating effects of social support may be explained if victimized adolescents with the worst health outcomes are turning to other victimized peers or bullies (who bully them) for support. For example, if a victimized child discloses information to a bully, the bully can use it to further relationally victimize the child.

Perhaps more shocking, Crick and Nelson (2002) found that victimization occurs within dyadic friendships. That is, peer victimization does not solely occur in the larger peer group context but also occurs within friendships. Moreover, Grotpeter and Crick (1996) found that the friendships of children who engage in relational victimization are often very intimate and involve high levels of jealousy. In addition, these friendships have high levels of relational victimization. Although some of my victimized adolescents may have perceived positive support from their friends, they may also be bullied by their friend. In addition, it is possible that some of my participants were not part of mutual friendships. That is, although the participant named their two best friends, those friends may not reciprocate the friendship. Perceived support may not be enough to buffer a victimized adolescent against poor physical health outcomes. Rather, perceived support from friends may only act as a buffer in a mutual friendship. Unfortunately, I did not collect information about whether the participants' friends were victims or bullies or whether the friendship was mutual. Future studies should examine the possibility that victimized adolescents who rely on other victimized students may be at a higher risk for negative physical

health outcomes than victimized adolescents who turn to nonvictimized adolescents or even bullies for support.

Swann and his colleagues (1992) postulated in the self-verification theory that persons with low self-esteem seek out feedback from others that matches their negative self-image. This theory suggests that persons prefer to receive feedback that matches (rather than mismatches) their self-imagine, even if the feedback is negative. Joiner, Katz, and Lew (1997) found that negative feedback about oneself was linked with increased depression and peer rejection. Interestingly, Weinstock and Whisman (2004) found a positive link between depression and negative feedback seeking (i.e., the more depressed one is, the more negative feedback is sought). Moreover, they found that negative feedback seeking was also associated with more perceived partner rejection (within heterosexual dating couples). The self-verification theory suggests that the unexpected moderating role of peer social support may partially be explained by victimized adolescents seeking out negative feedback from their friends that matches their experiences of being victimized. This negative feedback could perhaps (a) reinforce reasons for why they are being targeted by bullies (e.g., personality traits, physical weaknesses); (b) be focused around the bullying experience (e.g., expressing dislike for the bullies); or (c) be perceived as supportive even though the support is negative in nature. Receiving mismatching information from friends may be a stressful experience for victimized adolescents rather than a buffering, helpful experience.

Adolescence is a period when many changes are taking place. One such change important to the current discussion is the transition from relying on parents for support and guidance to relying on peers (Furman & Buhrmester, 1985; Harris, 1995). In addition to seeking out their parents, adolescents increasingly start depending on their friends for support (Sullivan, 1953; Hartup, 1983; Spear, 2000). By the 7th grade, Furman and Buhrmester (1992) found that adolescents were rating their peers as being equally supportive as their parents. Since my sample consisted of adolescents in 5th to 8th grade it is possible that my participants have not

fully learned how to effectively utilize support from friends. In addition, given that support from friends is just beginning to be an important avenue of support, the buffering effects of positive peer support may not be evident yet. That is, younger adolescents may not know how to effectively use social support from friends whereas older adolescents can take advantage of positive support from close friends. Thus, there may be a development effect where social support from friends buffers against negative health outcomes as adolescents grow older.

As expected, the degree that parents excluded their adolescent moderated the link between victimization and frequency of health problems. The link between victimization and frequency of health problems was strongest when levels of parental exclusion were high whereas low levels of exclusion buffered victimized adolescents against frequent health problems. This finding mirrors meta-analytic results indicating that higher levels of parental rejection predict higher depression during childhood (McLeod, Weisz, & Wood, 2007). Rigby, Slee, and Martin (2006) also found that that poor peer relations coupled with poor parenting (i.e., low parent care, high parent control) was significantly linked with poor mental health. The current findings provide preliminary evidence that being excluded by one's parents moderates the link between peer victimization and frequency of health problems such that a stronger link is seen when exclusion by parents is high.

While researchers are continuing to find that some aspects of relationships have negative outcomes on adjustment (e.g., Rose, Carlson, & Waller, 2007), my current findings contribute to the literature by suggesting that relationship components can also negatively affect physical health. Additional research is needed to first replicate these new findings and second to more fully understand *why* the negative link exists. Longitudinal research should investigate whether the current pattern of results continue throughout adolescence.

5.3 *Do Adjustment Problems Mediate the Link Between Victimization and Health Outcomes?*

Given the well-established link between victimization and adjustment problems, I examined whether adjustment problems mediated the victimization-health link. Indeed,

internalizing problems partially mediated the link between victimization and health problems (i.e., frequency and severity of health problems and trips to the doctor/nurse). Externalizing problems, however, was not a significant mediator. While internalizing problems was a significant mediator, the direct links between victimization and health outcomes were still significant even with internalizing problems in the model. Thus, as expected, internalizing problems did not fully explain the link between victimization and negative health outcomes.

5.4 *Exploring the Links Between Victimization, Daily Cortisol Patterns, and Health Outcomes*

Although research is beginning to see a link between peer victimization and health problems, this link has been largely based on findings from self- and parent-reports. Indeed, most studies on the victimization-health link have looked at either health outcomes or cortisol patterns. No study to date has examined both health outcomes and cortisol patterns simultaneously when examining victimization's influence of health. Research needs to start exploring potential underlying mechanisms in this victimization-poor health link. In the current study, I included both measures of health and measures of salivary cortisol levels in an effort to determine whether cortisol levels could help examine the link between victimization and poor health outcomes. Although I relied on self- and parent-reports of victimization and health outcomes, I gathered cortisol samples from adolescents over two school days (as well as in the laboratory; see below). Samples were collected at four time points each day in order to assess the diurnal cortisol pattern across the day. I expected that peer victimization would predict daily patterns of cortisol which in turn would predict health outcomes.

This dissertation was one of the first studies to examine how victimization, diurnal patterns of cortisol, and health outcomes are related. Although Vaillancourt et al. (2008) examined the link between daily cortisol and victimization, they solely looked at two assessments per day and did not include measures of health outcomes. This dissertation allowed for the examination of the diurnal pattern by including four assessment points per day (for two days). I found the expected overall diurnal pattern where cortisol levels peaked 30

minutes after waking and then decreased over the course of the day. Moreover, there was a cubic effect for assessments by victimization indicating that the diurnal pattern differed for victimized and nonvictimized adolescents. Victimized adolescents had lower levels of cortisol 30 minutes after waking and 30 minutes before going to bed than nonvictimized adolescents. This finding is consistent with other findings that lower levels of cortisol are linked with peer victimization (Vaillancourt et al.; Hansen et al., 2006). In addition, Cicchetti and Rogosch (2001) found that children who were physically maltreated evidenced lower levels of morning cortisol than children who were not maltreated or those children who were sexually maltreated. Lower cortisol levels, specifically hypocortisolism, are also linked with PTSD (Heim et al., 2000). My research suggests that victimization is not merely typical school-yard behavior and a normal part of growing up. Indeed, peer victims have similar diurnal patterns to persons who have experienced stressful events (e.g., Kliewer, 2006; Vaillancourt et al., 2008; Hansen et al., 2006).

Research from other areas has also focused on this cortisol awakening response. The awakening response is defined by the typical increase in cortisol roughly 30 minutes after waking. This awakening response is usually the peak in the diurnal pattern; cortisol levels gradually drop throughout the remainder of the day. Some researchers conceptualize the awakening response as a "cortisol mobilization response" during which the body mobilizes its resources for the day (e.g., Tops et al., 2008, p. 553). This morning response has been linked with the occurrence and prevalence of stressors. For example, Tops et al. found that women with a high fear of negative social evaluation (i.e., higher levels of social stress) evidenced a lower cortisol awakening response than women with a lower fear of negative social evaluation. I also found evidence for a blunted cortisol awakening response in victimized adolescents. Victimized adolescents evidenced significantly lower cortisol levels 30 minutes after waking compared to nonvictimized adolescents. Perhaps more interesting, victimized girls did *not* show a significant *change* in cortisol within the first 30 minutes of waking (i.e., no change between waking and 30 minutes later) whereas nonvictimized girls *did* show a significant increase 30

minutes after waking. It is worth noting that this change in cortisol during the first 30 minutes is significantly different for victimized and nonvictimized girls. In other words, victimized girls show a dampened cortisol awakening response whereas nonvictimized girls do not. No significant differences were seen between victimized and nonvictimized boys.

Gender differences in cortisol patterns are increasingly being seen in the literature. Weekes et al. (2008) found that women evidenced higher levels of cortisol on stressful days (i.e., examination day) than on nonstressful days; men did not show elevated cortisol levels on stressful day. Barnett, Steptoe, and Gareis (2005) showed that adults who are dealing with more marital conflict reported higher levels of stress (with women reporting feeling more stressed). Moreover, they also exhibited a dampened cortisol awakening pattern and a flatter slope throughout the day (likely due to the dampened awakening response. Given the evidence of gender differences in the literature, future studies should continue to examine the link between victimization and daily cortisol patterns differ for girls and boys.

An interesting link emerged between daily cortisol levels and health problems. I found that lower cortisol levels 30 minutes after waking and higher cortisol levels 30 minutes prior to going to bed both predicted more health problems. Although marginal, this finding is interesting given that hypocortisolism (i.e., lower morning levels and higher afternoon/evening levels) has previously been linked with increased mental health problems such as loneliness and anxiety (e.g., Steptoe, Owen, Kunz-Ebrecht, & Brydon, 2004). Moreover, victimized girls evidenced lower levels of cortisol 30 minutes after waking. Taken together, the data suggest links between victimization, lower morning cortisol, and increased health problems for girls. Additional research is needed to more fully understand how daily patterns and levels of salivary cortisol impact health outcomes for victimized boys and girls. Studies with a larger sample should examine whether daily cortisol plays a mediating role between victimization and health outcomes.

5.5 *Exploring the Links Between Victimization, Cortisol Reactivity, and Health Outcomes*

When discussing cortisol reactivity, a linear relationship between stress and cortisol typically comes to mind. That is, the more stress a person perceives/experiences, the higher levels of cortisol found in his/her body. However, the relationship between acute stressors and cortisol reactivity is not as clear-cut as researchers once thought. For example, Weekes et al. (2008) examined whether real-world stressors (i.e., examination days among college students) and linked with elevated cortisol levels. They found that women evidenced higher levels of cortisol on stressful days (i.e., examination days) than on nonstressful days whereas men did not show elevated cortisol levels on stressful day. The type of stressor also appears to play a role in cortisol reactivity. Stroud, Salovey, and Epel (2002) found that higher cortisol responses were seen during achievement-based tasks for men and during social rejection-based tasks for women.

In my data, I saw the expected increase in cortisol reactivity for nonvictimized adolescents. That is, cortisol levels gradually increased over the course of the TSST and peak 30 minutes after delivery the speech. This pattern of cortisol reactivity in my nonvictimized adolescents seems to match the typical stress response. For my victimized adolescents, however, there is a sharper (although nonsignificant) increase immediately after being introduced to the social stress task, indicating that victimized adolescents are more reactive to the acute social stressor. Moreover, victimized adolescents evidence a significant drop in cortisol during the 30 minutes following their speech delivery. This drop in cortisol was surprising since cortisol usually takes approximately 20 to 30 minutes to enter saliva after the onset of a stressor.

These findings, however, mirror those of Roy (2004). Roy found that in firemen, lower mean cortisol reactivity was negatively associated with stress. That is, lower mean cortisol responses during a stressful lab task were linked with a higher incidence of real-life stressors prior to participating in the lab task. Moreover, he suggests that higher levels of cortisol

reactivity during an acute stressor are linked with more adaptive coping styles, less negative affect, and less recent stress experiences. Interestingly, Davies and colleagues (2007) found that actual levels of parental conflict negatively predicted cortisol reactivity in children when they listened to their parents engage in a mock conflict. In other words, lower cortisol reactivity was linked with greater amounts of parental conflict. Taken together, my findings converge with previous research to suggest that victimized adolescents are indeed experiencing more daily experiences of social stress than nonvictimized adolescents and that this exposure to heightened stressors is impacting their HPA axis functioning.

Although not reaching significance, I found trends suggesting that cortisol reactivity differs for boys and girls. First, the trend indicates that both nonvictimized boys and girls exhibited the expected increase in cortisol reactivity throughout the speech. Although not significant, nonvictimized girls seem to have higher cortisol levels throughout the speech than nonvictimized boys. Both victimized boys and girls appear to have similar levels and patterns of cortisol reactivity; both boys and girls evidence a significant drop in cortisol after delivering their speech although the drop seems magnified for girls. In my sample, girls reported feeling less accepted and more rejected than boys. Girls perceived the speech as more threatening and felt they were rated more poorly than boys perceived the task. These increased negative reactions to the speech could explain the trends for differences between boys and girls. My self-report findings match the work of Sally Dickerson who has found that women are more impacted than men by social evaluative threat (Dickerson, in press). However, Dickerson has repeatedly found that high degrees of social-evaluative threat are linked with elevated cortisol reactivity during the TSST. My research has added to her findings by further examining a normal population of adolescents. Indeed, examining individual differences in victimization gives us additional information about how adolescents' HPA axis is functioning. Future research with a larger sample size should further examine these trends in cortisol reactivity for girls and boys.

Research suggests that consistent high reactivity is linked with negative mental and physical health outcomes. Lovallo and Gerin (2003) proposed that high reactivity can lead to negative health problems in several ways including cognitive-emotional reactions and increased activation in the hypothalamus. The current dissertation provided early evidence that acute social stressors elicit emotional reactions (especially in girls) and impacts cortisol production (which is the end product of the HPA axis activation). Thus, I expected that cortisol reactivity would mediate the link between peer victimization and negative health outcomes. I found that cortisol levels 30 minutes after the speech delivery partially mediated the link between victimization and how frequency adolescents went to the doctor. Specifically, I found that peer victimization predicted lower cortisol levels 30 minutes after delivering the speech which predicted more frequent visits to the doctor. This finding is slightly different to what I expected; I anticipated that initial cortisol reactivity to the speech would also mediate the victimization-health link.

5.6 *Uncertainty of Cortisol's Role in the Human Body*

In spite of the growing number of studies examining cortisol, little is known about the exact role cortisol plays in the human body. Recent research has challenged the notion of a straightforward linear relationship between stress and cortisol. That is, higher levels of perceived stress do not always positively correlate with higher levels of cortisol. Moreover, dsyregulation of the HPA axis could result in higher *or* lower levels of cortisol production; it is not yet clear whether both higher and lower levels have equally detrimental health effects. Previous research has demonstrated that higher levels of cortisol are linked with outcomes such as degeneration of cells in the hippocampus and altered immune functioning. Additional evidence suggests that low levels of cortisol may be linked with difficulty in coping with physically painful experiences. For example, (Garofalo, Robinson, Gatchel, & Wang, 2007) examined patients experiencing acute lower back pain and found that persons with lower cortisol levels reported higher pain ratings compared with persons with higher cortisol levels.

More research examining how the interplay between stress and the HPA axis influences health outcomes is needed.

5.7 Concluding Comments

This dissertation was an initial step in trying to understand *why* the victimization-poor health link is so robust. This study was the first to include measures of victimization, cortisol, and health in a single study. Indeed, the major contribution of this dissertation was the ability to examine the interplay between victimization, cortisol, and self-reported health outcomes. I began by demonstrating a strong positive association between victimization and negative health outcomes. I then examined the robustness of the victimization-health link by testing whether the link could be moderated by either sex of the participant or perceived social support. No moderating effects were found for sex of the participant which suggests that the link between victimization and health outcomes are equally detrimental for both boys and girls. Unexpected results were seen for the moderating effect of perceived social support in that perceived support from friends actually exacerbated the link between victimization and health problems. Low levels of parental support buffered victimized adolescents against negative health problems. I then examined the central hypotheses of this dissertation and found preliminary evidence that peer victimization influenced both diurnal patterns of cortisol as well as reactivity during an acute social stressor. Furthermore, the data suggested a link between cortisol levels and negative health outcomes.

In spite of the exciting findings here, caution must be exercised in generalizing the results of this study. Due to the small sample size (i.e., only 31 victims), several of my interesting trends may be due to chance and should thus be replicated in a larger sample. While it is possible that the effects could be washed away with a larger sample, the current trends are in the predicted direction. I expect the inclusion of more victimized adolescents would strengthen the associations found. Moreover, this study was correlational in nature. Even though my statistical models and theoretical framework imply a clear causal relationship, care

must be taken not to interpret the findings as causal. Indeed, causal statements cannot be deduced from correlational studies.

Furthermore, I expect to see a developmental trend where the link between victimization, cortisol, and health outcomes becomes more prominent as children progress through adolescence. Given that peer victimization peaks during early adolescence, it is possible that the negative effects of victimization have not yet fully impacted the HPA axis. In addition, adolescence is a period of increased vulnerability to stressful events (Walker et al., 2001). Early adolescents perceive fairly stable levels of stress whereas perceived stress decreases in later adolescence (Seiffe-Krenke, Aunola, & Nurmi, 2009). Indeed, all of my participants were in early adolescence (i.e., 5th to 8th grade). Perhaps more importantly, alterations to the HPA axis do not occur overnight. For example, Putnam (2007) found that children who were sexually abused initially evidenced hypercortisolism. Around 15 years of age, their pattern of cortisol had shifted to hypocortisolism; however, it was not until between the ages of 18 to 20 that this pattern of hypocortisolism became stable. This shift is somewhat expected given that elevations in cortisol are beneficial in the short-term (i.e., help mobilize resources); however, chronically high levels of cortisol have detrimental effects on health (e.g., suppressed immune functioning, deterioration of neural synapses). Thus, it is likely that the trends I found in the current sample mark the beginning of a shift to hypocortisolism for victimized adolescents. Longitudinal research is needed to examine this hypothesis.

The current dissertation advances the field, in part, by demonstrating that the negative effects of victimization are not solely confined to psychological and mental health outcomes. Contrary to popular belief, for approximately 10 to 30% of American children, peer victimization is not merely part of normal everyday “kid” behavior. Rather, the current findings provide early evidence that peer victimization in adolescence significantly alters biological functioning, specifically that activity of the HPA axis. Moreover, there appear to be links between victimization, cortisol, and health problems that call for further exploration. The results from this

dissertation are very exciting in that it moves the field another step closer to understanding *why* the link between peer victimization and negative health outcomes exists.

APPENDIX A

THINGS THAT HAPPEN TO ME AT SCHOOL

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

Scale

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

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

APPENDIX B

DIRECT AND INDIRECT AGGRESSION SCALE (DIAS)

How do classmates act toward you when they have problems with or get angry with you?

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

1. How often are you hit by others 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 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?.

APPENDIX C

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

APPENDIX D

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?

APPENDIX E

NETWORK OF RELATIONSHIPS INVENTORY – D (NRI-D)

Instructions: The questions below ask about your relationships with the seven types of people listed on the right. On each blank line write one number from 1 to 5. Look at the bottom of the page to see what each number means. Rate the "father figure" or "mother figure" who lives in your home if you live with someone who is not your natural parent.

Scale:

1 = Never or hardly at all
2 = Seldom or not too much
3 = Sometimes or somewhat
4 = Often or very much
5 = ALWAYS or EXTREMELY much

Companionship (COM)

1. How often do you spend fun time with this person?
11. How often do you and this person go places and do things together?
21. How often do you play around and have fun with this person?

Intimate Disclosure (DIS)

2. How often do you tell this person things that you don't want others to know?
12. How often do you tell this person everything that you are going through?
22. How often do you share secrets and private feelings with this person?

Pressure (PRE)

3. How often does this person push you to do things that you don't want to do?
13. How often does this person try to get you to do things that you don't like?
23. How often does this person pressure you to do the things that he or she wants?

Satisfaction (SAT)

4. How happy are you with your relationship with this person?
14. How much do you like the way things are between you and this person?
24. How satisfied are you with your relationship with this person?

Conflict (CON)

5. How often do you and this person disagree and quarrel with each other?
15. How often do you and this person get mad at or get in fights with each other?
25. How often do you and this person argue with each other?

Support (SUP)

6. How often do you turn to this person for support with personal problems?
16. How often do you depend on this person for help, advice, or sympathy?
26. When you are feeling down or upset, how often do you depend on this person to cheer things up?

Criticism (CRI)

7. How often does this person point out your faults or put you down?
17. How often does this person criticize you?
27. How often does this person say mean or harsh things to you?

Approval (APP)

8. How often does this person praise you for the kind of person you are?
18. How often does this person seem really proud of you?
28. How much does this person like or approve of the things you do?

Dominance (DOM)

9. How often does this person get their way when you two do not agree about what to do?
19. How often does this person end up being the one who makes the decisions for both of you?
29. How often does this person get you to do things their way?

Exclusion (EXC)

10. How often does this person ***not*** include you in activities?
20. How often does it seem like this person ignores you?
30. How often does it seem like this person ***does not*** give you the amount of attention that you want?

APPENDIX F

YOUR OPINIONS ABOUT GIVING YOUR SPEECH (POST TSST QUESTIONNAIRE)

1. Did you feel liked during the task?
2. Did you feel accepted during the task?
3. Did you feel disliked during the task?
4. Did you feel rejected during the task?
5. How effective do you think you were in convincing those around you that you should get the job you were applying for in your speech?
6. How do you think those around you would have rated your performance overall?
7. This session was: 1 = not very difficult 7 = very difficult
8. During the session, I: 1 = tried very hard 7 = did not try
9. During the session, I felt: 1 = not at all confident 7 = very confident
10. During the session, I felt: 1 out of control 7 in control
11. Overall, I thought I performed: 1 = very poorly 7 very well
12. Overall, I thought the tasks were threatening
13. Overall, I thought the tasks were challenging
14. Overall, I thought the tasks were stressful
15. I just "gave up or quit trying
16. I wanted to give up or quit trying
17. My performance on the speech was being evaluated by others during the task.
18. My performance on the speech could be evaluated by others at a later time

APPENDIX G

QUESTIONS TO ASK DURING THE TSST

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

APPENDIX H

FORM TO MARK ON WHILE PARTICIPANT GIVES SPEECH

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

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

Is the participant: MALE FEMALE **(30 seconds)**

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

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

_____

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

ASK TWO FOLLOW-UP QUESTIONS FROM LIST (IF RAN FULL TIME) WHEN FINISHED; IF SHORT, ASK QUESTIONS UNTIL TIME IS UP, THEN ASK TWO FOLLOW-UP QUESTIONS

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

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

Did participant take the entire 5 minutes? YES NO

Rate the overall quality of the speech

1	2	3	4	5
Very poor		Average		Excellent

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

1	2	3	4	5
Not at all		Average		Extremely

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

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

REFERENCES

Achenbach, T. M., & Rescorla, L. A. (2001). Manual for the ASEBA school-age forms and profiles. Burlington, VT: University of Vermont, Research Centre for Children, Youth and Families.Aiken, L. S., & West, S. G. (1991). Multiple Regression: Testing and interpreting interactions. Thousand Oaks: Sage.

Adams, R.E., Bukowski, W.M., & Bagwell, C. (2005). Stability of aggression in early adolescence as moderated by reciprocated friendship status and friend's aggression. *International Journal of Behavioral Development, 29,* 139-145.

Aiken, L. S., & West, S. G. (1991). *Multiple Regression: Testing and interpreting interactions*. Thousand Oaks: Sage.

Amoroso, D.M., & Walters, R.H. (1969). Effects of anxiety and socially mediated anxiety reduction on paired associated learning. *Journal of Personality and Social Psychology, 11,* 388-396.

Bagwell, C. L., Newcomb, A. F., & Bukowski, W. M. (1998).Preadolescent friendship and peer rejection as predictors ofadolescent adjustment. Child Development, 69, 140-153.

Barnett, R.C., Steptoe, A., & Gareis, K.C. (2005). Marital-role quality and stress-related psychobiological indicators. *Annals of Behavioral Medicine, 30*, 36-43.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173-1182.

Baumeister, R. F., & Leary, M. R. (1995). The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychological Bulletin*, *117*, 497-529.

Baumeister, R. F., & Twenge, J. M. (2003). The social self. In T. Millon & M. J. Lerner (Eds.), Handbook of psychology: Personality and social psychology, Vol. 5 (pp. 327-352). New York: John Wiley & Sons.

Berndt, T. J. (2002). Friendship quality and social development. *Current Directions in Psychological Science, 11*, 7-10.

Blair, C., Granger, D., Willoughby, M., Kivlighan, K., & the family life project investigators (2006). Maternal sensitivity is related to hypothalamic-pituitary-adrenal axis stress reactivitiy and regulation in response to emotion challenge in 6-month-old infants. *Annals of the New York Academy of Sciences, 1094*, 263-267.

Bouchard, T.J., & Loehlin, J.C. (2001). Genes, evolution, and personality. *Behavior Genetics*, *31*, 243-273.

Boulton, M.J. & Underwood, K (1992). Bully/victim problems among middle school children. *British Journal of Educational Psychology, 62*, 73-87.

Bowlby, J. (1973). *Attachment and loss. Vol 2: Separation: Anxiety and anger.* New York: Basic Books.

Bjorkqvist, K., Lagerspetz, K.M.J., Osterman, K. (1992). The Direct and Indirect Aggression Scales. Finland: Abo Akademi University.

Buck, R.W., & Parke, R.D. (1972). Behavioral and physiological to the presence of a friendly or neutral person in two types of stressful situations. *Journal of Personality and Social Psychology, 24,* 143-153.

Bugental, D.B., Martorell, G.A., & Barraza, V. (2003). The hormonal costs of subtle forms of infant maltreatment. *Hormones and Behavior, 43,* 237-244.

Bukowski, W. M., Hoza, B., & Boivin, M. (1994). Measuring friendship quality during pre- and early adolescence. *Journal of Social and Personal Relationships, 11,* 471-484.

Cannon, W.B. (1929). Bodily changes in pain, hunger, fear, and rage (2nd Edition). D. Apppleton & Co., New York.

Carels, R.A., Sherwood, A., & Blumenthal, J.A. (1998). Psychosocial influences on blood pressure during daily life. *International Journal of Psychophysiology, 28,* 117-129.

Carver, C.S. (2007). Stress, coping, and health. In H.Friedman & R. Silver (Eds.) Foundations of health psychology. (pp 117 – 144). New York: Oxford University Press.

Cicchetti, D. & Rogosch, F.A. (2001a). Diverse patterns of neuroendocrine activity in maltreated children. *Development and Psychopathology, 13,* 677-693.

Cicchetti, D. & Rogosch, F.A. (2001b). The impact of child maltreatment and psychopathology on neuroendocrine functioning. *Development and Psychopathology, 13*, 783-804.

Chen, Zhansheng, Williams, K.D., Fitness, J., & Newton, N.C. (2008). When hurt will not heal: Exploring the capacity to relive social and physical pain. *Psychological Science, 19*, 789-795.

Craig, K.D. (1999). Emotions and psychobiology. In P.D. Wall & R. Melzack (Eds.), *Textbook of pain,* 4th Edition. pp. 331-344. Edinburgh: Churchill Livingstone.

Crick, N. R., & Grotpeter, J. K. (1995). Relational aggression, gender, and socialpsychological adjustment. *Child Development,* 66, 710-722.

Crick, N.R. & Nelson, D.A. (2002). Relational and physical victimization within friendships: Nobody told me there'd be friends like these. *Journal of Abnormal Child Psychology, 30*, 599-607.

Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences*. Hillsdale, NJ: Erlbaum.

Cohen, J.L., Lakey, B., Tiell, K., & Neely, L.C. (2005). Recipient-provider agreement on enacted support, perceived support, and provider personality. *Psychological Assessment, 17,* 375-378.

Cohen, S. & Wills, T.A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin, 98*, 310-357.

Davidson, L.M. & Demaray, M.K. (2007). Social support as a moderator between victimization and internalizing-externalizing distress from bullying. *Social Psychology Review, 36*, 383-405.

Davies, P.T., Sturge-Apple, M.L., Cicchetti, D., & Cummings, E.M. (2007). The role of child adrenocortical functioning in pathways between interparental conflict and child maladjustment. *Developmental Psychology, 43,* 918-930.

DeLongis, A. & Holtzman, S. (2005). Coping in context: The role of stress, social support, and personality in coping. *Journal of Personality (special issue on Personality and Daily Experience), 73,* 1633-1656.

Demarary, M.K., & Malecki, C.K. (2003). Perceptions of the frequency and importance of social support by students classified as victims, bullies, and bully/victims in an urban middle school. *School Psychology Review,* 32, 471 -489.

Dembo, R. & Schmeidler, J. (2003). Family empowerment intervention: An innovative service for high-risk youths and their families. Binghamton, NY: Haworth.

DeVries, A.C., Glasper, E.R., & Detillion, C.E. (2003). Social modulation of stress responses. *Physiology and Behavior, 79*, 399-407.

Dickerson, S.S. (in press). Physiological responses to experience of social pain. To appear in G. MacDonald and L. Jensen-Campbell (Eds.), *Social Pain: A Neuroscientific, Social, and Health Psychology Analysis.* Washington DC: American Psychological Association.

Dickerson, S.S. & Kemeny, M.E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130,* 355-391.

Dougall, A.L. & Baum, A. (2001). Stress, health, and illness. In A. Baum, T.A., Revenson, & J.E. Singer (Eds.) *Handbook of Health Psychology.* (pp. 321-337). Manwah, NJ: Lawrence.

Egan, S. K. & Perry, D. G. (1998). Does low self-regard invite victimization? *Developmental Psychology,* 34, 299-309.

Eisenberger, N. I., & Lieberman, M. D. (2004). Why rejection hurts: A common neural alarm system for physical and social pain. *Trends in Cognitive Science, 8(7),* 294-300.

Eisenberger, N.I. & Lieberman, M.D. (2005). Why it hurts to be left out: The neurocognitive overlap between physical and social pain. In K.D. Williams, J.P. Forgas, & W.V. Hippel. The Social Outcast: Ostracism, Social Exclusion, Rejection, and Bullying. (pp. 109-123). Psychological Press: New York.

Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science, 302*, 290-292.

Eisenberger, N.I., Jarcho, J.M., Lieberman, M.D., & Naliboff, B.D. (2006). An experimental study of shared sensitivity to physical pain and social rejection. *Pain.*

Eisenberger, N.I., Taylor, S.E., Gable, S.L., Hilmert, C.J., & Lieberman, M.D. (2007). Neural pathways link social support to attenuated neuroendocrine stress responses. *NeuroImage, 35,* 1601-1612.

Feldman, S.I., Downey, G. & Schaffer-Neitz, R. (1999). Pain, negative mood, and perceived support in chronic pain patients: A daily diary study of people with reflex sympathetic dystrophy syndrome. *Journal of Consulting and Clinical Psychology, 67,* 776-785.

Furman, W. & Buhrmester, D. (1985). Children's perceptions of the personal relationships in their social networks. *Child Development, 21,* 1016-1024.

Furman, W. & Buhrmester, D. (1992). Age and sex differences in perceptions of networks of personal relationships. *Child Development, 63,* 103-115.

Gillespie, C.F. & Nemeroff, C.B. (2007). Corticotropin-releasing factor and the psychobiology of early-life stress. *Current Directions in Psychological Science, 16*, 85-89.

Gini, G. (2008). Associations among overt and relational victimization and adolescents' satisfaction with friends: The moderating role of the need for affective relationships with friends. *Journal of Youth and Adolescence, 37*, 812-820.

Glaser, R., Sheridan, J., Malarkey, W.B., MacCallum, R.C., & Kiecolt-Glaswer, J.K. (2000). Chronic stress modulates the immune response to a pneumococcal pneumonia vaccine. *Psychosomatic Medicine, 62,* 804-807.

Grills, A. E., & Ollendick, T. H. (2002). Peer victimization, global self-worth, and anxiety in middle school children. *Journal of Clinical Child and Adolescent Psychology, 31*, 59-68.

Grotpeter, J.K. & Crick, N.R. (1996). Relational aggression, overt aggression, and friendship. *Child Development, 67*, 2328-2338.

Gunnar, M.R., Morison, S.J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages. *Development and Psychopathology, 13,* 611-628.

Hansen, A.M., Hogh, A., Persson, R., Karlson, B, Garde, A.H., Orbaek, P. (2006). Bullying at work, health outcomes, and physiological stress response. *Journal of Psychosomatic Research, 60*, 63-72.

Harmon-Jones, E., & Sigelman, J. (2001). State anger and prefrontal brain activity: Evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology, 80(5),* 797-803.

Harris, J.R. (1995). Where is the child's environment? A group socialization theory of development. *Psychological Review, 102,* 458-489.

Hart, J., Gunnar, M., & Cicchetti, D. (1996). Altered neuroendocrine activity in maltreated children related to depression. *Development and Psychopathology, 8*, 201-214.

Harter, S. (1985). Competence as a dimension of self-evaluation: Toward a comprehensive model of self-worth. In R.E. Leahy (Ed.) The development of the self (pp 55-121). Orlando, FL: Academic Press.

Hartup W.W. (1983). Peer Relations. In E.M. Hetherington (Ed.) *Handbook of Child Psychology,* Vol. IV (pp 103-196). New York: Wiley.

Hartup, W.W. (1996). The company they keep: Friendships and their developmental significance. *Child Development, 67,* 1-13.

Haynie, D.A., Berg, S., Johansson, b., Gatz, M., & Zarit, S.H. (2001). Symptoms of depression in the oldest old: A longitudinal study. *Journal of Gerontology B. Psychological Sciences and Social Sciences, 56,* 111-118.

Heim C, Newport DJ, Heit S, Graham YP, Wilcox M, Bonsall R, Miller AH, Nemeroff CB (2000). Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. JAMA ; 284:592–597

Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. *Biological Psychiatry, 54*, 1389 - 1398.

Hodges, E. V. E., Boivin, M., Vitaro, F., & Bukowski, W. M. (1999). The power of friendship: Protection against an escalating cycle of peer victimization. *Developmental Psychology, 75,* 94-101.

Hodges, E. V. E., Malone, M. J., & Perry, D. G. (1997). Individual risk and social risk as interacting determinants of victimization in the peer group. *Developmental Psychology,* 33, 1032-1039.

Hodges, E.V.E. & Perry, D.G. (1996). Victimization is never just child's play. *National School Safety Center News Journal,* 4-7.

Huey, S.J. & Weisz, J.R. (1997). Ego control, ego resiliency, and the five-factor model as predictors of behavioral and emotional problems in clinic-referred children and adolescents. *Journal of Abnormal Psychology, 106,* 404 – 415.

Inslicht, S. Marmar, C., Meylan, T., Metzler, T., Hart, S., Otte, C., McCaslin, S.E., Larkin, G.L., Hyman, K.B., & Baum, A. (2006). Increased cortisol in women with intimate partner violence-related posttraumatic stress disorder. *Annals of the New York Academy of Sciences, 1071,* 428-429.

Jackson, T., Weiss, K.E., Lunquist, J.J., & Soderlind, A. (2005). Sociotropy and perceptions of interpersonal relationships as predictors of eating disturbances among college women: Two prospective studies. *Journal of Genetic Psychology, 166*, 346-359.

Jensen-Campbell, L.A., Knack, J., Allen, G., Sarkar, S., Rex-Lear, M., & Gomez, M. (2007). *Does Need to Belong Sensitize Neurological Reactions to Social Pain?* Manuscript in Preparation.

Johnson, D.M., Delahanty, D.L., & Pinna (2008). The cortisol awakening response as a function of PTSD severity and abuse chronicity in sheltered battered women. *Journal of Anxiety Disorders, 22*, 793-800.

Kenny, D. A., Kashy, D. A., & Bolger, N. (1998). Data analysis in social psychology. In D. Gilbert, S. Fiske, & G. Lindzey (Eds.), *The handbook of social psychology* (Vol. 1, 4th ed., pp. 233-265). Boston, MA: McGraw-Hill.

Kiecolt-Glaswer, J.K., Newton, T., Cacioppo, J.T., MacCallum, R.C., Glaser, R., & Malarkey, W.B. (1996). Marital conflict and endocrine function: Are men really more physiologically affected than women? *Journal of Consulting and Clinical Psychology, 64,* 324-332.

Kirschbaum, C. & Hellhammer, D.H. (1999). Noise and stress: Salivary cortisol as a non-invasive measure of allostatic load. *Noise and Health, 4,* 57-65.

Kirschbaum, C., Klauer, T., Filipp, S.H., & Hellhammer, D.H. (1995). Sex-specific effects of social support on cortisol and subjective responses to acute psychological stress. *Psychosomatic Medicine, 57*, 23-31.

Kirschbaum, C., Pirke, K.M., & Hellhammer, D.H. (1993). The "Trier Social Stress Test:"A tool for investigating psychobiological stress responses in a laboratory setting.*Neuropsychobiology, 28*, 76-81.

Kliewer, W. (2006). Violence exposure and cortisol responses in urban youth. *International Journal of Behavioral Medicine, 13,* 109-120.

Knack, J.M. & Jensen-Campbell, L.A. (2007, January). Re-Experiencing Social versus Physical Pain and Its Influence on Self-Control. Paper presented at the annual meeting of the *Society of Personality and Social Psychology,* Memphis, TN.

Knack, J.M., Jacquot, C., Jensen-Campbell, L.A., Malcolm, K.T. (2007) The Importance of Having Agreeable Friends (Especially When You are Not). Unpublished manuscript.

Knack, J.M., Rex-Lear, M., Bryant, N., Gomez. M., & Jensen-Campbell., L.A. (2007, March). Personality and Social Competence in Close Friendships during Childhood. Poster presented at the bi-annual meeting of the *Society of Research in Child Development*, Boston, MA.

Knack, J.M., Waldrip, A.M., & Jensen-Campbell, L.A. (2007). Social Support. In R. Baumeister & K. D. Vohs (Eds.), *Encyclopedia of social psychology*. Thousand Oaks, CA: SAGE Publications.

Kochenderfer, B. & Ladd, G. W. (1996). Peer victimization: Cause or consequence of school maladjustment? *Child Development, 67*, 1305-1317.

Kochenderfer-Ladd, B. & Ladd, G. W. (2001). Variations in peer victimization: Relations to children's maladjustment. In J. Juvonen and S. Graham (Eds.), *Peer harassment in school* (pp. 25-48). New York: Guilford Press.

Kudielka, B.M., Schmidt-Reinwald, A.K., Hellhammer, D.H., & Kirschbaum, C. (1999). Psychological and endocrine responses to psychological stress and dexamethasone/corticotrophin-releasing hormone in healthy postmenopausal women and young controls: The impact of age and a two-week estradiol treatment. *Neuroendocrinology, 70*, 422-430.

Kupersmidt, J.B., Coie, J.D., & Dodge, K.A (1990). Predicting disorder from peer social problems. In S.R. Asher & J.D. Coie (Eds.), *Peer Rejection in Childhood* (pp. 274- 305). New York: Cambridge University Press.

Laursen, B., Bukowski, W.M., Aunola, K., & Nurmi, J.E. (2007). Friendship moderates prospective associations between social isolation and adjustment problems in young children. *Child Development, 78,* 1395-1404.

Larson, R. & Richards, M.H. (1994). Divergent realities: The emotional lives of mothers, fathers, and adolescents. New York: Basic Books.

Limber, S.P. & Small, M.A. (2003). State laws and policies to address bullying in schools. *School Psychology Review, 32,* 445-455.

Little, R.J.A., (1988). A test of missing completely at random for multivariate data with missing values. *Journal of the American Statistical Associate, 83*, 1198-1202.

Liu, Y-L. (2006). Paternal/maternal attachment, peer support, social expectations of peer interaction, and depressive symptoms. *Adolescence, 41,* 705 – 721.

Loeber, R., & Dishion, T. J. (1984). Boys who fight at home and school: Family conditions influencing cross-setting consistency. *Journal of Consulting and Clinical Psychology, 52*, 759–768.

Lovallo, W.R. & Gerin, W. (2003). Psycholphysiological reactivity: Mechanisms and pathways to cardiovascular disease.

MacDonald, G., Kingsbury, R., & Shaw, S. (2005). Adding insult to injury: Social pain theory and response to social exclusion. In K. Williams, J. Forgas, & W. van Hippel (Eds.) *The social outcast: Ostracism, social exclusion, rejection, and bullying.* New York: Psychology Press.

MacDonald, G., & Leary, M.R. (2005). Why does social exclusion hurt? The relationship between social and physical pain. *Psychological Bulletin, 131*, 202-223.

MacKinnon, D.P., Lockwood, C.M., Hoffman, J.M., West, S.G., & Sheets, V. (2002). A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods, 7*, 83-104.

Malecki, C.K. & Demaray, M.K. (2003). What type of support do they need? Investigating student adjustment as related to emotional, informational, appraisal, and instrumental support.

Malone, M.J. & Perry, D.G. (1995, April). Features of aggressive and victimized children's friendships and affliative preferences. Poster presented at the biennial meeting of the Society for Research in Child Development in Indianapolis, Indiana.

Maslow, A.H. (1968). Toward a psychology of being (2PNd P Edition). New York: Van Nostrand Reinhold.

Mason, J.W. (1971). A re-evaluation of the concept of "non-specificity" in stress theory. *Journal of Psychiatric Research, 8*, 323-333.

McBeth. J., Chiu, Y.H., Silman, A.J., Ray, D., Morriss, R., Dickens, C., Gupta, A., & Macfarlane, G.J. (2005). Hypothalaic-pituitary-adrenal stress axis function and the relationship with chronic widespread pain and its antecedents. *Arthritis Research and Therapy, 7*, R992-R1000.

McLeod, B. D., Weisz, J. R., & Wood, J. J. (2007). Examining the association between parenting and childhood depression: A meta-analysis. Clinical Psychology Review, 27, 986-1003.

McEwen, B.S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Science, 840,* 33-44.

McEwen, B.S. (2000). The neurobiology of stress: From serendipity to clinical relevance. *Brain Research, 886*, 172-189.

McEwen, B.S., Conrad, C.D., Kuroda, Y., Frankfurt, M., Magarinos, A., & McKittrick, C. (1997). Prevention of stress-induced morphological and cognitive consequences. *European Neuropsychopahrmacology, 7*, 323-328.

Melzack, R. (1999). Pain – an overview. *Acta Anaesthesiol Scand, 43*, 880-884.

Melzack, R. (2005). Evolution of the neuromatrix theory of pain. The prithvi raj lecture: Presented at the third world congress of world institute of pain, Barcelona 2004. *World Institute of Pain, 5,* 85-94.

Melzack, R. & Wall, P.D. (1965). Pain mechanisms: A new theory. *Science, 150*, 971-979.

Miller, G.E., Cohen, S., Pressman, S., Barkin, A., Rabin, B., S., & Treanor, J.J. (2004). Psychological stress and antibody response to influenza vaccination: When is the critical period for stress, and how does it get inside the body? *Psychosomatic Medicine, 66,* 215-223.

Miller, G.E., Cohen, S. & Ritchey, A.K. (2002). Chronic psychological stress and the regulation of pro-inflammatory cytokines: A golucocorticoid-resistance model. *Health Psychology, 21,* 531-541.

Mischel, W., Cantor, N., & Feldman, S. (1996). Principles of self-regulation: The nature of willpower and self-control. In E. T. Higgins and A. W. Kruglanski (Eds.), *Social psychology: Handbook of basic principles* (pp. 329-360).

Nansel, T. R., Overpeck, M., Pilla, R.S., Ruan, J., Simons-Morton, B., & Scheidt, P. (2001). Bullying behaviors among U.S. youth: Prevalence and association with psychosocial adjustment. Journal of the American Medical Association, 285, 2094-2100.

Olweus, D. (1978). Aggression in the schools: Bullies and whipping boys. Washington, D.C. Hemisphere.

Olweus, D. (1993). Bullying at school: What we know and what we can do. Cambridge, MA: Blackwell. ED 384 437.

Parker, J.G. & Asher, S.R. (1987). Peer relations and later personal adjustment: Are low-accepted children at risk? *Psychological Bulletin, 102,* 357-389.

Pedersen, S., Vitaro, F., Barker, E.D., & Borge, A.I.H. (2007). Early behavioral dispositions and middle childhood peer rejection and friendship: Direct, indirect, and moderated links to adolescent adjustment. *Child Development, 78,* 1037-1051.

Pellegrini, A.D., Bartini, M., & Brooks, F. (1999). School bullies, victims, and aggressive victims: Factors relating to group affiliation and victimization in early adolescence. *Journal of Educational Psychology, 91,* 216-224.

Perry, H.E., Wright, R.O., Shannon, M.W., & Woolf, A.D. (1998). Baclofen overdose: Drug experimentation in a group of adolescents, *Pediatrics, 1001,* 1045-1048.

Perry, D.G., Hodges, E.V.E., & Egan, S.K. (2001). Determinants of chronic victimization by peers: A review and a new model of family influence. In J.Juvonen & S. Graham (Eds.). Peer Harassment in School: The Plight of the Vulnerable and Victimized. (pp 73-104). New York: Guilford Press.

Pervanidou, P. (2008). Biology of post-traumatic stress disorder in childhood and adolescence. *Journal of Neuroendocrinology, 20*, 632-638.

Preacher, K. J. (2002, May). Calculation for the test of the difference between two independent correlation coefficients [Computer software]. Available from http://www.quantpsy.org.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, *40*, 879-891.

Price, D. (2000). Psychological and neural mechanisms of the affective dimension of pain. *Science, 288*, 273-279.

Purvis, K.B. & Cross, D.R (2006). Improvements in salivary cortisol, depression, and representations of family relationships in at-risk adopted children utilizing a short-term therapeutic intervention. *Adoption Quarterly, 10*, 25- 43.

Raudenbush, S.W. & Bryk., A.S. (2002). *Hierarchical linear models* , (2P Nd P Edn.), Sage, Thousand Oaks, CA.

Rigby, K. (1998). The relationship between reported health and involvement inbully/victim problems among male and female secondary schoolchildren. *Journal of Health Psychology, 3,* 465-476.

Rigby, K. & Slee, P. (1999). Suicidal ideation among adolescent school children, involvement in bully-victim problems, and perceived social support. *Suicide and Life-Threatening Behavior, 29,* 119-130.

Rigby, K., Slee, P.T and Martin, G. (2006). Implications of inadequate parental bonding and peer victimization for adolescent mental health. *Journal of Adolescence.*

Rohleder, N., Joksimovic, L., Wolf, J.M., & Kirschbaum, C. (2003). Hypocortisolism and increased glucocorticoid sensitivity of pro-inflammatory cytokine production in Bosnian war refugees with posttraumatic stress disorder. *Biological Psychiatry, 55*, 745-751.

Rose, A.J., Carlson, W., & Waller, E.M.(2007). Prospective associations of co-rumination with friendship and emotional adjustment: Considering the socioemotional trade-offs of co-rumination. *Developmental Psychology, 43*, 1019-1031.

Roy, M.P. (2004). Patterns of cortisol reactivity to laboratory stress. *Hormones and Behavior, 46*, 618-627.

Sapolsky, R.M., Uno, H., Rebert, C.S., & Finch, C.E. (1990). Hippocampal damage associated with prolonged glucocortioid exposure in primates. *Journal of Neuroscience, 10*, 2897-2902.

Selye, H. (1950). Physiology and Pathology of Exposure to Stress. 1st Edition. Acta, Montreal, Canadad.

Selye, H. (1956). The Stress of Life. New York, NY: McGraw-Hill Book Company, Inc.

Seminowicz, D.A., & Davis, K.D. (2006). Cortical responses to pain in healthy individuals depends on pain catastrophizing. *Pain, 120*, 297-306.

Smith, T.W., Gallo, L.C., Goble, L., Ngu, L.Q., & Stark, K.A. (1998). Agency, communion, and cardiovascular reactivity during martial interaction. *Health Psychology, 17*, 537-545.

Smyth, J., Ockenfels, M.C., Porter, L., Kirschbaum, C,, Hellhammer, D.H., & Stone, A.A. (1998). Stressors and mood measures on a momentary basis are associated with salivary cortisol secretion. *Psychoneuroendocrinology, 23*, 353-370.

Spear, L.P. (2000). Neurobehavioral changes in adolescence. *Current Directions in Psychological Science, 9,* 111-114.

Steele, R.G. & Aylward. B.S. (2007). The use of cluster analytic techniques in developmental and behavioral pediatric research. *Journal of Developmental and Behavioral Pediatrics, 28*, 327-329.

Steptoe, A., Owen, N., Kunz-Ebrecht, S.R., & Brydon (2004). Loneliness and neuroendocrine, cardiovascular, and inflammatory stress responses in middle-aged men and women. *Psychoneuroendocrinology, 29,* 593-611.

Stroud, L. R., Salovey, P., & Epel, E. (2002). Sex differences in adrenocortical responses to achievement and interpersonal stress. *Biological Psychiatry, 52,* 318.

Sullivan, H. S. (1953). *The interpersonal theory of psychiatry.* New York: Norton.

Steele, R.G. & Aylward, B.S. (2007). The use of cluster analytic techniques in developmental and behavioral pediatric research. *Journal of Developmental and Behavioral Pediatrics, 28(4)*, 327-329.

Tabachnick, B. G., and Fidell, L. S. (2007). Using Multivariate Statistic, *5th ed.* Boston: Allyn and Bacon.

Tarullo, A.R. & Gunnar, M.R. (2006). Child maltreatment and the developing HPA axis. *Hormones and Behavior, 50,*632-639.

Taylor, S.E. (2002). *The tending instinct: How nurturing is essential to who we are and how we live.* New York: Holt.

Taylor, S.E. (2007). Social support. In H.S. Friendman & R.C. Silver (Eds.), *Foundations of health psychology* (pp. 145-171). New York: Oxford University Press.

Taylor, S.E., Klein, L.C., Lewis, B.P., Gruenewald, T.L., Gurung, R.A., & Updegraff, J.A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review, 107,* 411-429.

Taylor, S.E., Lerner, J.S., Sage, R.M., Lehman, B.J., & Seeman, T.E. (2004). Early environment, emotions, responses to stress, and health. *Journal of Personality, 72*, 1365-1393.

Tops, M., Riese, H., Oldehinkel, A.J., Rijsdijke, F.V., & Ormel, J. (2008). Rejection sensitivity relates to hypocortisolism and depressed mood state in young women. *Psychoneuroendocrinology, 33*, 551-559.

Turk, D.C. & Monarch, E.S. (2002). Biopsyschosocial perspective on chronic pain. In : D.C. Turk & R.J. Gatchel (Eds.). *Psychological Approaches to Pain Management: A Practitioner's Handbook.* New York: Guilford Press, pp 1 – 29.

Uno, H., Tarara, R., Else, J.G., Suleman, M.A., & Sapolsky, R.M. (1989). Hippocampal damage associated with prolonged and fatal stress in primates. *Journal of Neuroscience, 9*, 1705-1711.

Vaillancourt, T., Duku, E., Decatanzaro, D., Macmillan, H., Muir, C., & Schmidt (2008). Variation in hypothalamic-pituitary-adrenal axis activity among bullied and non-bullied children. *Aggressive Behavior, 34,* 294-305.

van Lier, P.A.C., Vitaro, F., Wanner, B., Vuijk, P., & Crijnen, A.A.M. (2005). Gender differences in developmental links among antisocial behavior, friends' antisocial behavior, and peer rejection in childhood: Results from two cultures. *Child Development, 76*, 841-855.

Waldrip, A., & Jensen-Campbell, L.A. (2007a). Why Do I Feel so Bad After Being Excluded? The Mediational Effects of Threatened Needs on Social Exclusion. Manuscript under Review.

Waldrip, A., & Jensen-Campbell, L.A. (2007b) The Influence of Belongingness on Reactions to Social Exclusion. Manuscript under Review.

Waldrip, A.M., Malcolm, K.T., & Jensen-Campbell, L.A. (2008) Social Adjustment as Predicted by Victimization, Peer Acceptance, and Friendships. *Social Development.*

Walker, E.F. (2002). Adolescent neurodevelopment and psychopathology. *Current Directions in Psychological Science, 11,* 24-28.

Walker, E.F., Walder, D.J., & Reynolds, F. (2001). Developmental changes in cortisol secretion in normal and at-risk youth. *Development and Psychopathology, 13*, 721-732.

Weekes, N.Y., Lewis, R.S., Goto, S.G., Garrison-Jakel, J., Patel, F., & Lupien (2008). The effect of an environmental stressor on gender differences on the awakening cortisol response. *Psychoneuroendocrinology, 33*, 766-772.

Williams, K.D. (2001). *Ostracism: The Power of Silence.* New York: Guilford.

Williams, K.D. (2007). Ostracism. *Annual Review of Psychology, 58*, 425-452.

Williams, K., Chambers, M., Logan, S., & Robinson, D. (1996). Association of common health symptoms with bullying in primary school children. *British Medical Journal, 3,* 17-19.

Wolke, D., Woods, S., Bloomfield, L., & Karstadt, L. (2001). Bullying involvement in primary school and common health problems. *Archives of Disease in Childhood, 85,* 197-201.

BIOGRAPHICAL INFORMATION

Jennifer M. Knack received her bachelor of arts from Saint Bonaventure University in Olean, New York where she majored in psychology. She then moved to Texas where she earned her master of science in experimental psychology from the University of Texas at Arlington. Her primary research interests center on understanding how social pain experiences such as peer victimization influence biological functioning. She hopes to expand her research to other species such as primates and larger marine animals. Jennifer plans to continue her research training as a post doctoral scholar in Canada where she will continue to examine the influence of peer victimization on biological functioning. She is lives with her cat named Nimbus.