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IMPACT OF EXPOSURE TO VIOLENCE ON URBAN YOUTH: A BIOPSYCHOSOCIAL PERSPECTIVE OF AGGRESSION BY JODI S. HUNTINGTON

Dissertation Committee

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Submitted in partial fulfillment of the requirements of the Degree of Doctor of Philosophy Seton Hall University 2012

SETON HALL UNIVERSITY COLLEGE OF EDUCATION AND HUMAN SERVICES OFFICE OF GRADUATE STUDIES

APPROVAL FOR SUCCESSFUL DEFENSE

Doctoral Candidate, Jodi Huntington, has successfully defended and made the required modifications to the text of the doctoral dissertation for the Ph.D. during this Spring Semester 2012.

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ABSTRACT

IMPACT OF EXPOSURE TO VIOLENCE ON URBAN YOUTH: A BIOPSYCHOSOCIAL PERSPECTIVE OF AGGRESSION

Exposure to violence has pervasive and deleterious effects on children that are just beginning to be understood by the mental health community. The purpose of this study was to address the void in current research regarding potential moderating factors between exposure to violence and aggression in urban youth. A biopsychosocial model was employed to capture the complex and interrelatedness of the contributing factors; specifically, neurophysiological, neurodevelopmental, and psychosocial factors were examined as they related to exposure to violence and aggression. Lastly, risk and protective factors were identified.

DEDICATION

To my parents who gave me roots,

To Christopher who gave me wings,
and to the children, whose courage inspires me,
I share this achievement with you.

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CHAPTER 1

Introduction

Children are being exposed to violence and trauma at an epidemic rate, with those in urban communities at the greatest risk. In some communities, exposure to violence is so pervasive that studies have reported that up to 95% of the children studied had been exposed to violence (Margolin & Gordis, 2004; Skybo, 2005). Other reports assess children's exposure at over 50% in one year alone (Richters & Martinez, 1993).

Overstreet (2000) found that among 75 students from an urban community, 83% of children knew someone killed from violence, 55% witnessed shootings, 43% observed a dead body, and 37% experienced personal violence. In another study, examiners found that almost 80% of middle school children had witnessed a drug deal, while 90% saw someone being assaulted (White, Bruce, Farrell, & Kliewer, 1998). This rate of exposure of inner city children to extreme forms of violence drives an urgent line of inquiry regarding the development consequences of this seemingly unavoidable experience.

Statement of the Problem

One of the most pervasive and observable consequences of violence exposure is the increase in externalizing behaviors seen in boys, specifically, the emergence of aggression. Studies have shown that exposure to violence in the community has been significantly related to the development of antisocial behaviors and peer-directed aggression in school-aged boys over and above the development of posttraumatic stress symptoms, depression, and anxiety (Buka, Stichick, Birdthistle, & Earls, 2001).

Furthermore, this aggressive behavior in adolescents has been linked to antisocial behavior and delinquency in adolescence as well as incarceration and perpetration in adulthood (Dahlberg, 1998), further continuing the cycle of violence.

While some children have environmental buffers within the school or home to mediate the impact of being exposed to violence, for many children no safe place exists. This is particularly true for children that are living in impoverished communities (Jensen, 2009; Lipina & Colombo, 2009; Margolin & Gordis, 2004), where their homes and their community are often characterized by chaos, gang violence, and under-resourced schools. These children must adapt, develop, and learn in the face of profound circumstances resulting in tremendous stress (Jensen). Existing in this perpetual state of stress comes at a considerable cost as adaptations to these experiences can alter development, causing physiological, neurodevelopmental, emotional, behavioral, cognitive, and social impairment (Palmer, Farrar, & Ghahary, 2002; Rothschild, 2000; Solomon & Heide, 2005).

While the amount of exposure varies based on how it is defined in each study, the statistics reflect a significant societal problem with grave implications for the children. Clearly, exposure to violence from a variety of experiences, including gang activity, gunshots, domestic violence, or abuse, has important biological, psychological, and social implications that impact both the individual and their community. Exposure, for example, has been correlated with the development of posttraumatic stress symptoms (Overstreet, 2000), externalizing and internalizing behaviors, poor academic outcomes, and physical and mental illness (Watts-English, Fortson, Gibler, Hooper, & De Bellis,

2006), and juvenile delinquency (Lee & Hoaken, 2007). The scientific literature to date generally focuses on a specific type of violence exposure and narrow consequences (McDonald & Richmond, 2008); leaving a void in how the cumulative impact of exposure can simultaneously impact the inter-related physiological, neurodevelopmental, and social consequences (Dodge & Pettit, 2003).

Not all children will respond to exposure in the same way. Much research has examined changes in neurodevelopment and function as it relates to the child's physiological response to the threat (Solomon & Heide, 2005), the nature of the violent experience (Nader, 2008), or a range of psychosocial factors associated with the child, their family, and community (McDonald & Richmond, 2008); however, few studies have attempted to look at the cumulative effect of exposure (Margolin & Gordis, 2004) and the interrelatedness of the physiological, neurodevelopmental, psychological, and social factors and how they contribute to aggression in urban youth (Perry, 2001; Teisl & Cichetti). The current research attempted to address this scientific gap by employing a biopsychosocial perspective to examine how exposure to violence in children related to aggressive behavior and to what extent biological, psychological, and/or social factors might moderate this relationship. The following sections provide an overview for the existing scientific literature related to this research question and provide the background and rationale for the development of the present study. Specifically, the stress response from exposure to violence will be detailed along with potential physiological and neurophysiological changes. The potential impact that these alterations have on neurodevelopment, cognition, and executive functioning are discussed. In addition, elements that serve as both risk and protective factors are reviewed. Lastly, the

importance of utilizing a biopsychosocial framework to examine the impact of exposure to violence has on the development of aggression are underscored.

Stress Response

It has been well established, across the literature that exposure to violence is a traumatic event, resulting in physiological and psychological stress (Nader, 2008). These types of stress assumptions provide the fundamental principles of neurodevelopment and evidence of the underlying mechanisms that cause functional changes in children exposed to violence (Lee & Hoaken, 2007; Perry, 2005; Rothschild, 2000). Specifically, it has been hypothesized that exposure to violence sets in motion a series of threat-responses within the brain. When in excess, such as being exposed to reoccurring violence, the activation of the neural systems responsible for the threat responses can alter the structures and development of the brain. These adaptive responses to threat, present during exposure to violent experiences, lead to alterations that may result in discernable changes in biological, psychological, and social functioning.

While research related to stress and stress responses is common in the modern literature, the stress phenomenon has roots very early in science. Stress is defined as, "the nonspecific response of the body to any demand whether it is caused by, or results in, pleasant or unpleasant conditions" (Selye, 1984, p. 74). In the case of exposure to violence, when an individual first perceives threat, the body immediately signals the activation of a stress response, which triggers biological, cognitive, and psychological responses (Resnick, 2001). Hans Seyle coined the term "general adaptation syndrome" to describe this process. In the first of three stages, the alarm and mobilization stage, stress

hormones are activated as the body enters fight or flight, which is mediated by the autonomic nervous system (Resnick). During this process the blood flow moves to major muscle groups in preparation for movement, increased heart rate, constriction of blood vessels, and blood glucose is mobilized. The brain then responds by signaling the release of neurochemicals, such as epinephrine and norepinephrine, which act to increase respiration, heart rate, blood pressure, and the consumption of oxygen. This orchestrated response then provides the structure for a fight or flight response (Resnick; Rothschild, 2000). The second stage, the resistance stage, refers to the attempt to return the body to homeostasis, while the third stage, the exhaustion stage, is described as physiologic dysregulation and dysfunction.

The autonomic nervous and endocrine systems are largely involved in the initial stress response, which works simultaneously with the neuroendocrine system. When an individual perceives threat or is under stress, corticotropin-releasing hormones (CRH) and arginine-vasopressin (AVP) are secreted. The fight or flight response of the autonomic nervous system, engage the sympathetic nervous system which creates cardiovascular, respiratory, endocrine, and gastrointestinal responses. The hypothalamic-pituitary-adrenal (HPA) axis is also activated by the release of the CRH and AVP, which, in turn, triggers another series of events (Lee & Hoaken, 2007). The hypothalamus secretes corticotropin releasing factor, which then prompts the release of adrenocorticotropic hormone from the pituitary, and subsequent release of cortisol from the adrenal cortex (Solomon & Heide, 2005). The cortisol serves to terminate the stress response through a feedback loop mechanism.

Neurobiological Alterations

Dysregulation of the HPA axis, as in persistent or prolonged physiological stress, can impact numerous neural regions as well as over and under activation of the HPA axis, resulting in impaired functioning of the limbic systems and neuronal damage (Lee & Hoaken, 2007). It is readily established that child and adolescent exposure to violence can be traumatic for an individual and has the potential for extreme stress and subsequent neurobiological consequences (Saltzman, Pynoos, Layne, Steinberg, & Aisenberg, 2001; Solomon & Heide, 2005).

Exposure to violence and posttraumatic stress symptoms has been positively correlated across the literature (McDonald & Richmond, 2008). The impact of exposure to violence on physiological development in children, has been directly linked to the development of trauma related symptoms, post-traumatic stress (PTS), and post-traumatic stress disorder (PTSD; McDonald & Richmond; Solomon & Heide, 2005). When using community samples, children exposed to violence have been typically described across studies as having symptoms associated with PTS and PTSD, not as having a diagnosis of PTSD (McDonald & Richmond). Given the under-identification of PTSD in a community sample of children, there is a paucity of literature that broadly examines those children that have been exposed to violence to determine the presence or extent of neurophysiological changes (Buka et al., 2001). Based on the inherent exposure to violence and trauma that precipitates the development of PTSD, this literature will be detailed to further potentiate mechanisms of change as it suggests that there are long-term effects of exposure.

A decrease in the size of the amygdala, hippocampus, corpus callosum, and cerebellar vermis as well as an increase in the size of the putamen and lateral ventricles have been found in children that have experienced persistent trauma (McCollum, 2006; McNally, 2003). These changes are suggested to be a result of a prolonged elevation of glucocortisol levels in these structures. Additionally, these areas are high in glucocorticiod receptors (McCollum), making the structures of the limbic system vulnerable during traumatic experiences. Literature supports the interaction between traumatic and stressful environments, enhanced corticosteroid levels, and cell death despite the presence of inconsistencies in pediatric literature (McNally; Nader, 2008).

Advancements in brain imaging technology have elucidated brain regions involved in PTSD (Korn, n.d.). Changes of the structures and functions of the hippocampus and prefrontal cortex are present in children that have experienced sexual trauma and posttraumatic stress disorder (Bremner, 2002). These results suggest that the symptoms of PTSD in children that have experienced abuse can be associated with alterations in brain structure and function. It has been found that traumatized children with PTSD had smaller intracranial and cerebral volumes than the control subjects (Jackowski, de Araújo, Tavares de Lacerda, Mari, & Kaufman, 2009). One study found that "brain volumes positively and robustly correlated with age of onset and negatively correlated with duration of abuse" (Nader, 2008, p. 51). In a related study, children with PTSD did not find smaller hippocampal volume, but did find smaller brain volume and a smaller corpus callosum (Bremner). Similarly, PTSD symptoms and dissociation are found to be elevated when corpus collosum and other regions were reported as decreasing in size (Bremner; Nader).

Structural changes can be further examined in an effort to better understand the aggressive behavior that is often seen in boys growing up in impoverished communities (Lee & Hoaken, 2007). Specifically, much attention has been given to amygdala and hippocampal changes that occur in children that have been exposed to violence. In addition, right brain underdevelopment as well as impairment with and neural connections between the orbitofrontal cortex, anterior cingulatecoretx (ACC), and amygdala have received growing attention (Solomon & Heide, 2005). Within the aggression literature, it has also been determined that individuals that are more aggressive show a decrease in grey matter in the frontal lobes which has been linked to altered decision making skills (Nestor, Kubicki, Nakamura, Niznikiewicz, McCarley, & Shenton, 2010). In addition, possible disruption in the amygdala has been well documented which has been found to contribute to inaccurate facial recognition (Tremblay, Hartup, & Archer, 2005). Hemispheric differences and prefrontal cortex development have been suspected in altered arousal and a biological predisposition to aggression (Critchley, Mathias, Josephs, O'Doherty, Zanini, Dewar, Cipolloti, Shallice, & Dolan, 2003; Raine, 2002).

Neurodevelopment

There is a scientific rationale for the reasons for and mechanisms by which a child might experience neurocognitive changes secondary to exposure to violence.

Specifically, it is well established that a child's brain is most vulnerable to negative environmental experiences and influences because of it plasticity (Lipina & Colombo, 2009; Rothchild, 2000). The maturation and integration of the regions of the brain are

vital for proper functioning and development. Because human development begins as a simple foundation and is shaped by each experience thereafter, interruption or insult of this process has a progressive effect (Palmer Frantz, Armsworth, Swak, Copley, & Bush, 1999; Perry, 2001). Prolonged and persistent stress can negatively affect both cognitive and emotional growth as well the integration of both systems (Nader, 2008).

While DNA is responsible for early brain formation, post-natal experiences determine the pruning of neural components and the formation of brain structures and linkages (McCollum, 2006). Myelination of axons most actively occurs within 6 month to 3 years of life, but continues into childhood and adolescence. As this process increases, the speed and efficiency of the information transmitted between neurons improves. Improvements in the functioning of the frontal lobe are thought to be a result of the pruning and increasing of synaptic connections based on use (Spear, 2000a) and is most active between the ages of seven and sixteen.

The multidimensional process of brain growth and development continues at varying rates until functional specialization of the specific brain region is achieved (Johnson, 2005). There are known critical and sensitive times with peak periods of growth (Thomas & Johnson, 2008). Interference at these stages can have significant and lifelong neurodevelopmental consequences (Markham & Greenough, 2004). Specifically, this has implications in the neural formations in children that have been exposed to stress and trauma. It has been proposed that children that are constantly scanning the environment for potential threats will be less likely to develop pathways for alternative processes (Lee & Hoaken, 2007).

The prefrontal cortex is typically not fully developed until adulthood and undergoes significant remodeling during adolescence, making children at this age uniquely vulnerable (Scarpa & Raine, 2000; Spear, 2000). Given the immaturity of frontal lobe, the child's capacity for self-awareness, self-control, and goal directed behaviors is limited (Spear). In addition, adolescence is a time when children are confronted with growing challenges and may be placed in situations where maladaptive thoughts and physiological dysfunction become more pronounced (Eckes & Radunovich, 2007; Lee & Hoaken, 2007). According to Piaget's stages of cognitive development, at the age of twelve, children are entering into the period of "Formal Operations" (Berger, 2008). This stage is marked by the departure from concrete thinking to the development of abstract reasoning, the ability to generate alternative hypotheses, and recognition of possible outcomes (Berger). Deficits or underdeveloped skills can place children of this age at further risk for delinquency.

Neurodevelopmental Response

Cognition. Exposure to violence creates tremendous physiological stress on the child (McDonald & Richmond, 2008), which can negatively impact learning and memory. According to Perry (2001), a key factor to understanding learning in children exposed to violence is to consider that all people process, store, retrieve and respond to their environment in a "state-dependent fashion" (p. 10). When a child is in a state of arousal from chronic exposure to violence, the brain's ability to process information is very different from a child that is not in arousal. When hearing the same classroom instruction, the child that is in an alarm state will be less able to process verbal

information the teacher is providing than the child that is in a calm state. This child's mental energy will be predominantly spent focusing on non-verbal cues like non-verbal gestures and facial expression, resulting in the appearance of a distracted child. As a result of this "use-dependent" pattern, the child will have disproportionally developed non-verbal skills than verbal abilities. This capacity, often unknowingly deemed as "street smarts," develops out of adaptation as a result of perceived threat for the purposes of survival as children raised in the throws of violence have learned that attending to non-verbal information is more crucial than verbal information (Perry).

In one study, almost 40% of children raised in environments that were chronically traumatic, demonstrated a significant Verbal-Perceptual discrepancy on IQ testing (Perry, 2001). Not only does this have implications for the heightened perceptual skills and attention to threat, it also serves as a risk factor, as the children may not have the verbal abilities to effectively communicate in times of confrontation (Kikas, Peets, Tropp, & Hinn, 2009; Villemarette-Pittman, Standford, & Greve, 2002). Decreased verbal skills are also linked to frustration which, unresolved, can lead to aggression, causing further problems for the already vulnerable child (Tremblay, Hartup, & Archer, 2005).

Executive Function. Executive functions refer to the "higher-order processes of self-regulation of thought, action, and emotion," all dependent on neural systems of the prefrontal cortex (Lee & Hoaken, 2007, p. 283). This has been said to include problem solving, planning and organization, inhibition, shift, selective attention, verbal learning, and visual scanning skills (Tremblay et al., 2005). As a child develops, executive function skills become more developed and refined. By the age of twelve, a child has

developed most executive function abilities similar to adults; however, underlying neural structures continue to mature through the age of 20 to 30 years (Raine, 2002). Potential for dysfunction increases as a child ages because functioning becomes more complex.

Delays in development in any one portion of executive functioning can lead to disruption of metacognition. For example, if a child has hypervigilance toward potential threat cues, his ability to attend to and respond to classroom interactions may be hindered.

Children exposed to violence and resulting traumatic stress, have altered executive functioning as a result of neurophysiological and neurodevelopmental changes previously described. In addition, deficits and poorly established executive function skills have been associated with aggressive behaviors in children (Lee & Hoaken, 2007; Tremblay et al., 2005; Seguin & Zelazo, 2005). In numerous studies, executive function irregularities have been present in children that are more aggressive. Children who exhibit aggressive behaviors were found to be more perseverative and possessed less developed problem solving skill, suggesting that they may have difficulty generating alternative perspectives (Séguin, Arseneault, Boulerice, Harden, & Tremblay, 2002). Under-developed abstract verbal reasoning has also been identified as contributing to aggressive behavior as children may not be able to adequately generate alternative responses and negotiate social relationships (Kikas et al., 2009). Furthermore, young adults that presented with more disruptive behaviors showed less developed organizational and planning abilities (Villemarette-Pittman et al., 2002). While there is a high incidence of attention deficit hyperactivity disorder (ADHD) symptomology in children that show more aggressive behavior, it may be that the children are attending to

potential threat cues in the environment which is constricting their ability to attend to novel, non-threatening material.

The literature demonstrates that exposed children possess a heightened vigilance and attention to aggressive and negative stimuli (Teisl & Cicchetti, 2008) as well as difficulty interpreting non-verbal cues. This has been attributed to both neurophysiological and neurodevelopmental adaptations to the physiological stress response as well as social information processing (Dodge & Coie, 1987). It has been shown that children that have been exposed to personal violence were able to discern angry faces more quickly, with less cueing, than the control group (Pollak & Kistler, 2002). The researchers suggested that, while this may be adaptive for survival, over interpretation of signals could lead to incorrect judgments. This is congruent with other work that has shown that children that were abused were more likely to interpret neutral and ambiguous cues as hostile (Teisl & Cicchetti).

Psychosocial Development

Adolescence is a critical time for psychosocial development. Research has found that children who have experienced trauma show a behavioral regression and a negative attitude towards expectations and future. In addition, as mentioned previously, children who are exposed to violence and maltreatment have been shown to misinterpret social cues, attend to hostile cues, and behave in aggressive and withdrawn manners. While these alterations can be grounded in neurophysiological and neurodevelopmental bases, the result is further impeded social development and interpersonal relationships (Pollack, 2004).

Social cognition and normative beliefs of aggression have been identified as key components of children's development of aggression after exposure to violence (Guerra, Huesmann & Spindler, 2003). According to social learning theory, an individual learns from previous experiences with the environment and develops schemas accordingly (Bandura, 1977; Nader, 2008). If a child is repeatedly exposed to violence or lives in a community where violence is prevalent, the child will experience aggressive behavior as the norm.

Social information processing skills of children that have been exposed to violence has been largely examined. Social information processing model presented by Crick and Dodge highlights a series of processes in which children use when presented with social cues (Teisl & Cicchetti, 2008). It is suggested that when children possess distortions within these processes it increases the likelihood of aggression. In other words, the child inaccurately perceives, interprets, and makes conclusions about social exchanges that promote the use of aggressive behavior (Nader, 2008). Specifically, it has been shown that children that have a history of maltreatment and violence display a heightened attention to non-verbal cues, which can lead to overlooking other non-threatening contextual factors that would normally signal that the interaction was safe (Perry, 2001). In addition, children were also found to misinterpret affect with a bias toward anger, further raising their defenses (Teisl & Cicchetti).

Beliefs about the appropriateness of aggressive behavior have been linked to aggressive behaviors in children (Kikas et al., 2009). Research has found that the more a child believes that aggressive behavior is acceptable, the more likely he is to employ such tactics, with boys having higher "aggression-approval" normative beliefs (Kikas et al.).

Risk and Protective Factors

Biopsychosocial factors can place a child at increased risk for developing aggressive behaviors after exposure to violence. The nature and chronicity of the event, the child's attributes, neuropsychological deficits, and available coping mechanisms have all been found to contribute to aggression (DePrince, Weinzierl, & Combs, 2009; Twardosz & Lutzker, 2010).

Biological risk factors have been documented in aggression populations. Gender differences have been seen in the development of physical aggression (Séguin, Pihl, Harden, Tremblay, & Boulerice, 1995). While girls have been found to be more likely to develop symptoms of PTSD after exposure, boys tend to show more physical aggression (Hanson, Borntrager, Self-Brown, Kilpatrick, Saunders, Resnicj, & Amstadter, 2008). Gender also plays a part in exposure itself as boys are reported as witnessing more violence than girls of the same communities. This could be a due to many factors such as child-rearing differences between genders (i.e., freedoms given, etc.) and internalizing/externalizing of experiences (Skybo, 2005).

Prenatal exposure to toxins, as well as genetic factors, has also been found to influence aggression in children. For example, maternal depression and smoking while pregnant has been linked to later antisocial behavior (Hay, 2005). Another biological factor that has been found to contribute to aggressive behavior is heart rate. Scarpa and Ollendick (2003) found that aggression is related to increased baseline heart rate variability (HRV) and a decreased baseline heart rate (BHR). This factor has also been supported in an intergenerational transmission model with research of antisocial behavior (Scarpa & Raine, 2002). It has been found that a significant number of children with

antisocial parents have lower resting heart rates, which has been correlated with aggression, antisocial behavior, and violent offenses.

While children can be exposed to violence and maltreatment across socioeconomic levels and communities, poverty and stressors from the environment can place children at increased risk (Pollak, 2004); however, research has shown that social and family support can be a moderator in exposure to violence. Children that identified positive social support were less likely to experience posttraumatic stress symptoms and aggression. Conversely, children that had less involved or absent mothers demonstrated a higher amount of aggression and symptomology (Overstreet, Dempsey, Graham, & Moely, 1999).

Just as the factors previously described can put a child at increased risk for the development of aggressive behaviors, each can conversely serve as protective factors. Positive relationships with adults, well developed problem solving skills, and solid cognitive functioning have been identified as key components to resiliency in children (Hamill, n.d.; Wright, 1998). Since these skills are prerequisites to normal development, they too serve as protective factors when a child is faced with hardship.

Biopsychosocial Perspective of Aggression

As discussed in previous sections, exposure to violence has physiological, neuropsychological, and social implications that interact and affect one another to influence the expression of aggressive behavior. These complex and inter-related factors largely align with the biopsychosocial model. This model will be the foundation of the

proposed study for it postulates that result of exposure is multifaceted and should be examined across domains, recognizing the impact that each factor has on aggression.

Child exposure to violence has been consistently linked to aggression, yet there have been only a few attempts to conceptualize how it contributes to the development of aggression (Lee & Hoaken, 2007). Previous literature and research has focused on various aspects of aggression in children and the effects of exposure to violence but to date, none of these studies examined the complexities and dynamic interplay of the biological, psychological, and social systems. Perry (2001) has theorized and underscored the multiple dimensions of insult that occur when children are exposed to violence but has not yet investigated them together.

The Biopsychosocial model of therapeutic treatment has been gaining momentum across healthcare and in a variety of treatment settings. The basic premise of this theory is to address the dynamic and inter-related aspects of multiple areas of clients' lives (Palmer et al., 2002). A criticism of other "more traditional" models is that they address only one aspect of an individual and are not robust enough to look at the client as a whole (Kaplan & Coogan, 2005). Just as the name suggests, the Biopsychosocial Model (BSM) is formed from three areas of influence: biology, psychology, and social-culture (Kaplan & Coogan).

Viewing a person's mental state as, "many interacting processes" has been around for the past 2000 years (Gilbert, 2002, p.13). Greek physicians were said to recognize the importance of considering bodily processes, personality, and life experiences as all contributing to one's mental status (Gilbert). The modern version seems to be attributed to the work of Engel in the 1960s as he applied this model when understanding

cardiovascular disease. The Biopsychosocial model has since been incorporated into medical school teachings but has lagged behind in its inclusion in Counseling Psychology curriculum despite the profession's emphasis on multiculturalism and multi-axial conceptualization (Gilbert). The Biopsychosocial Model has been more recently adopted in research and employed within more multidisciplinary settings where treatment teams include professionals from psychology, medicine, and other social related fields.

Currently, use of this model has been effectively applied to numerous conditions, such as eating disorders, antisocial behaviors, schizophrenia, and substance abuse. The model is postulated to be applicable across settings, addressing the needs of patients in mental health, career, and school counseling settings (Kaplan & Coogan, 2005).

When employing a Biopsychosocial Model to a specific challenge, it is difficult to categorize factors of influence as this model inherently speaks to the dynamic and interactive processes across biological, psychological, and social domains (Palmer et al., 2002). This has also served as a challenge within previous research as some factors have been considered neuropsychological factors and also investigated as social factors. For the purposes of this study, biological factors include physiological and neurophysiological responses to exposure to violence and trauma; psychological factors include neuropsychological and cognitive changes in the exposed children; and social factors center around social support and social cognition.

The current research aimed to add to the literature by simultaneously considering biological, psychological, and social factors that moderate the relationship between exposure to violence and aggression. It was anticipated that this study would provide results that could advance the knowledge in the fields of education, psychology, and

corrections and informed educational planning, psychoeducation, and mental health services for at-risk adolescents.

Research Questions

Given the limitations of the existing research, the following were the specific questions evaluated by the present study.

Question 1. What is the incidence of exposure to violence in an urban population of male adolescents?

Question 2. Is there relationship between exposure and aggression in children exposed to violence?

Question 3: Does physiological stress response moderate aggression in exposed children?

Question 4: Does neurocognitive development moderate aggression in children exposed to violence?

Question 5: Does social support moderate aggression in children exposed to violence?

Research Hypotheses

Hypothesis 1: It is predicted that the population examined in this study will have had exposure to violence.

Hypothesis 2: There will be a significant, positive relationship between exposure and aggression with greater exposure equated with greater levels of aggression.

Hypothesis 3: The effect of exposure on aggression will be moderated by physiological stress response where lower baseline measure of heart rate will be related to greater aggression in children exposed to violence.

Hypothesis 4: The effect of exposure on aggression will be moderated by neurocognitive development where lower scores on cognitive measures will be related to greater aggression in children exposed to violence.

Hypothesis 5: The effects of exposure on aggression will be moderated by social support where greater social support will be related to lower aggression in children exposed to violence.

Definition of Terms

Exposure to Violence: Exposure to violence has been qualified in various ways throughout the literature (Overstreet, 2000). For example, some researchers have differentiated between witnessed and observed or exposure to interpersonal or community violence (Mrug, Loosier, & Windle, 2008). For the purposes of this study, exposure to violence was examined as any exposure, direct, witnessing, or awareness of aggressive and/ or threatening behaviors.

Aggression: Numerous constructs of aggression exist. The definition of aggression for this study was operationally defined as the use of force against another with or without an object (Tremblay & Nagin, 2005).

Traumatic Stress/Post-traumatic Stress Disorder (PTSD). When the body endures extreme or prolonged stress, traumatic stress is the result. Traumatic stress that persists following the experience of a traumatizing event is called posttraumatic stress (Rothchild,

2000). When symptoms develop that are in accordance with DSM-IV criteria, the individual is diagnosed as having Post-traumatic Stress disorder (PTSD). The diagnostic criteria for PTSD include a history of exposure to a traumatic event meeting two criteria and symptoms from each of three symptom clusters: intrusive recollections, avoidant/numbing symptoms, and hyper-arousal symptoms. A fifth criterion concerns duration of symptoms and a sixth assesses functioning. Specifically, DSM-IV-TR criteria is as follows (American Psychiatric Association, 2000).

A: The person has been exposed to a traumatic event in which both of the following have been present: (a) The person has experienced, witnessed, or been confronted with an event or events that involve actual or threatened death or serious injury, or a threat to the physical integrity of oneself or others. (b) The person's response involved intense fear, helplessness, or horror. Note: in children, it may be expressed instead by disorganized or agitated behavior.

B: The traumatic event is persistently re-experienced in at least one of the following ways: (a) Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. Note: in young children, repetitive play may occur in which themes or aspects of the trauma are expressed; (b) Recurrent distressing dreams of the event. Note: in children, there may be frightening dreams without recognizable content; (c) Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur upon awakening or when intoxicated). Note: in children, trauma-specific reenactment may occur; (d) Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of

the traumatic event; and (e) Physiologic reactivity upon exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event

C: Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by at least three of the following: (a) Efforts to avoid thoughts, feelings, or conversations associated with the trauma; (b) Efforts to avoid activities, places, or people that arouse recollections of the trauma; (c) Inability to recall an important aspect of the trauma; (d) Markedly diminished interest or participation in significant activities; (e) Feeling of detachment or estrangement from others; (f) Restricted range of affect (e.g., unable to have loving feelings); and (g) Sense of foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).

D: Persistent symptoms of increasing arousal (not present before the trauma), indicated by at least two of the following: (a) difficulty falling or staying asleep, (b) irritability or outbursts of anger, (c) difficulty concentrating, (d) hyper-vigilance, and (e) exaggerated startle response.

E: Duration of the disturbance (symptoms in B, C, and D) is more than one month.

F: The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Further, you need to specify if either acute (if duration of symptoms is less than three months) or chronic (if duration of symptoms is three months or more). You also need to specific if it is with or without delay onset (i.e., onset of symptoms at least six months after the stressor) or with a moderator (i.e., a variable that "affects the direction

and/or strength of the relation between the independent or predictor variable and the dependent or criterion variable" (Baron & Kenny, 1986, p 1174).

Physiological stress response: For the purpose of this study, physiological stress was defined by baseline heart rate measure, heart rate variability, and self-report scale of stress symptoms. The equipment used in this study was manufactured by AD Instruments and was specifically designed for use in research and clinical practice applications with human subjects. The equipment met all safety and regulatory standards of the ISO9001: 2008 quality management system.

Neuropsychology: Neuropsychology is defined as the study of the brain function and behavior (Zillmer, Spiers, & Culbertson, 2008).

Executive Function: Executive functions refer to the higher-order processes of thinking that includes problem solving, planning and organization, inhibition, shift, selective attention and verbal learning, and visual scanning (Lee & Hoaken, 2007).

Delimitations

The proposed research studied a convenience sample of adolescent boys attending a public school in an urban city in the Northeast region. Due to the complex and relatedness of contextual factors, the study did not employ use of a control group but instead looked at the intensity of symptomology as it related to level of exposure. The study was proposed to examine for moderation using multiple regression models, in effort to allow for an examination of the moderating effect that physiological, neuropsychological and psychosocial factors may have had on aggression. This study

aimed to contribute to the literature and promote a multifaceted view of aggression and exposure to violence in our youth.

Previous research findings on the effects of exposure and aggression are difficult to discern as various constructs have been used in investigations (Lee & Hoaken, 2007). Exposure has included variables such as direct abuse and neglect while others have included witnessing or having knowledge of violence and traumatic events (Mrug et al., 2008; Overstreet, 2000). Similarly, aggression has been defined as a range of behaviors to diagnosis conduct disorder across literature. The lack of consistency in the definition of these variables is addressed in the discussion and results of the study were generalized with appropriate caution.

CHAPTER II

Review of Related Literature

The purpose of this chapter was to provide a critical review and thoughtful discussion of the literature that is most relevant to the current study. Researchers and clinicians alike have just begun to understand the pervasive and pernicious effects that exposure to violence has on children. While ample studies document that exposure is related to behavioral, academic, and social-emotional changes in children, it is only in the recent years that the inclusion of neurophysiological and neurodevelopmental factors have been emphasized as significant contributors, even fundamental underpinnings, of the adaptive response to traumatic stressors (Perry, 2001).

Exposure to Violence

Research on exposure to violence often examines a specific type or level of exposure but does not address and acknowledge the cumulative effect of exposure or the multiple insults in urban areas for which children are at risk. Specifically, children living in high-risk communities may be exposed to multiple forms of violence, which can be overlooked when only attending to a specific type of exposure, and therefore underscore the necessity to consider exposure together (Rosenthal, 2000). While the impact of exposure to violence has been theorized (Lee & Hoaken, 2007; Nader, 2008; Perry, 2001; Perry, 2005; Rothschild, 2000), a scarcity of research exists. Studies that have examined the impact of exposure do so in a single dimension and in dichotomous models,

neglecting to look at interacting and overlapping factors. This also impedes the generalizability of findings across the exposed population.

An area of investigation that has received much attention is exposure to community violence. In a meta-analysis done by McDonald and Richmond (2008), the authors found inconsistencies in what constituted exposure, witnessing versus experiencing violence, and the proximity of the time frame of exposure, contributed to the difficulty in capturing the impact of exposure. They also highlighted the lack of consistency that researchers have found when they look at witnessing and victimization separately, further emphasizing the need to consider the factors together.

Lynch and Cicchitti (1998) described an ecological-transitional model that places the child at the center of a circle and progresses outward, having the microsystem of family, then exosystem of the community and neighborhood, and finally the macrosystem of culture and society surrounding him or her. While this model is useful in conceptualizing the potential impact of proximity to perpetrator and/or exposure, findings have not been consistent and do not begin to address the complexities of biological, psychological, and social factors.

Although Lynch and Cicchitti (1998), found that children (aged 7-12) did not exhibit externalizing behaviors after community exposure; a later study by McCabe, Lucchini, Hough, Yeh, and Hazen (2005) found that exposure to community violence had the strongest relationship, over witnessing intimate partner relationships within the home, with the development of conduct disorder. The authors further suggested that previous studies that did not include exposure to community violence may have lead to faulty conclusions about the impact of direct exposure.

Mrug and colleagues (2008) examined exposure to violence across contexts (i.e., home, school, and community) and conditions (i.e., witnessing, threat, or actual violence) in an effort to differentiate effects of each. Outcome variables included anxiety symptoms, depressive symptoms, aggressive fantasies, and overt aggression. While the researchers found relationships between variables, findings suggest that cumulative exposure is a better predictor of child outcomes then context specific information.

Exposure and Aggression

Numerous terms have been used throughout the scientific literature to describe aggression, which impedes the generalizability of the findings. While not interchangeable, terms such as externalizing behavior, antisocial behavior, hostility, anger, and aggressive behavior have all been used in the measurement of aggression (McDonald & Richmond, 2008). Despite these discrepancies, biological, psychological, and social factors have been linked to aggression in children, which point to potential moderating factors between it and exposure to violence.

While it is difficult to generalize findings across literature given the numerous definitions and constructs used, exposure to violence has been consistently linked to altered behavior, specifically, aggression. McCabe and colleagues (2005) examined the relationship between exposure to community violence, partner violence, and child maltreatment, in youths aged 12 to 17 years. A logistical regression was utilized to examine the contributions of each on predicting conduct disorder and externalizing behaviors in children after 2 years. The investigators found that exposure to community violence predicted conduct disorders and externalizing behaviors, where increased

exposure was related to higher likelihood of each.

Biopsychosocial Model

Child exposure to violence has been consistently linked to aggression, yet there have been only a few attempts to comprehensively conceptualize how it contributes to the development of aggression (Lee & Hoaken, 2007). Previous literature and research have focused on various aspects of aggression in children and the effects of exposure to violence, but to date, none of these studies examined the complexities and dynamic interplay of the biological, psychological, and social systems. While some have theorized and underscored the multiple dimensions of insult that occurs when children are exposed, none have yet investigated them together.

Biological, psychological and environmental factors are undistinguishable as each of these factors can have a direct or indirect affect on the others (Scarpa & Raine, 2000). For example, some biological factors can be caused by environment, such as in the case of injury to the brain from physical altercation. Conversely, biological factors can also result in neuropsychological changes, such as decreased inhibition, which could result in increased incidence of engaging in physical violence. From this example alone, the complex and inter-related nature of aggression can be elucidated, highlighting the need to move away from dichotomous research toward a biopsychosocial perspective.

Neurophysiological Response to Exposure

Brain Maturation in Children

There is a scientific rationale for the reasons and mechanisms by which a child might experience neurocognitive changes secondary to exposure to violence.

Specifically, it is well established that a child's brain is most vulnerable to negative environmental experiences and influences because of its plasticity (Rothschild, 2000).

The maturation and integration of the regions of the brain are vital for proper functioning and development. Since human development begins as a simple foundation and is shaped by each experience thereafter, interruption or insult of this process has a progressive effect (Lipina & Colombo, 2009). Prolonged and persistent stress can negatively affect both cognitive and emotional growth as well the integration of both systems (Nader, 2008).

While DNA is responsible for early brain formation, post-natal experiences determine the pruning of neural components and the formation of brain structures and linkages (McCollum, 2006; Spear, 2000). Myelination of axons is most actively occurring within 6 months to 3 years of life. The child is learning and acquiring skills for motor and cognitive processes as well as building strategies for self-regulation.

Interference at this stage of life can have significant and lifelong neurodevelopmental consequences (Nader, 2008). As the brain is developing, it is uniquely vulnerable to affective and sensory experiences. Lee and Hoaken (2007) purport that when the activation of neural responses become repetitious, those are the pathways that are established and maintained.

Adolescence is a period of tremendous growth and development, making it also a uniquely vulnerable time (Raine, 2002). During this time, children are faced with increased social pressure along with hormonal and cognitive changes (Eckes & Radunovich, 2007; Papalia, Olds, & Feldman, 2007). Given that the prefrontal cortex is not yet developed, decision making and problem solving skills may not be adequately developed to negotiate environmental, biological, and psychosocial factors. In the absence of support and safety, a child can become overwhelmed and exhibit maladaptive and aggressive behaviors.

Mechanisms of Response to Stress in the Body

It has been well established that there is a high incidence of child and adolescent exposure to violence that often results in traumatic levels of stress among those impacted (Saltzman et al., 2001). Further, there are numerous neurophysiological and biological correlates associated with the experience of stress. In order to fully comprehend the impact of violence exposure, it is necessary to first detail the mechanisms and processes by which exposure-related stress impacts physiology. The purpose of the ensuing sections shall be to detail these implications of stress.

Neurophysiological Response. Traumatic and stressful experiences activate stress response in the central and peripheral nervous system (Resick, 2001). The Reticular Activating System (RAS) comprised of a multi-symptom network of "ascending arousal related neural systems" is involved in arousal and anxiety as well as limbic and cortical process modulation (Perry, 2001; Perry, 2005). The RAS is activated by the traumatic

experience and mobilizes anxiety-related modulation. The Locus Coeruleus (LC) is the nuclei of the norepinepherine-containing neurons and is a key component of the RAS. These neurons originate in the pons and send axonal projections to all major brain regions thus playing a major role in the determination of the incoming sensory information (Perry, 2001). The Ventral Tegmental Nucleus (VTN) also has a role in this regulation. Intense stressors cause an increase in both LC and VTN activity. This activity results in the release of norepinepherine, which in turn impacts the brain and then the body. The regulation of arousal, affect, attention, locomotion, sleep, and startle response are greatly influenced by the LC and VTN. In addition, the level of arousal from stress is proportionately reflected in the level of LC activity resulting in an elevation of norepinephrine in the "LC and VTN terminal fields throughout the brain" if fear is increased (Perry, 2001, p. 227). The LC is responsible for hypervigilance toward critical information as well as mobilizing the process of autonomic nervous system activation, adrenocorticotropin and cortisol release from the activation of the HPA axis, and the immune system (Perry, 2005).

Limbic System. The limbic system is comprised of the amygdala, hippocampus, cingulate gyrus, thalamus, hypothalamus, and putamen and regulates the fight or flight responses in the body (Carlson, 2007). The integration of emotional and physical reactions, are guided by the limbic system with the hippocampus playing a critical part in the response to fear, memory and learning (Resnick, 2001). This structure is necessary for short term and declarative memory. Since stress hormones and stress related neurotransmitter systems from LC have the hippocampus as a target, it is at risk for

changes secondary to the stress response and subsequent impairment of memory.

Equally as important in the functioning of the limbic system is the amygdala. This structure plays a key role in the processing of information received from the thalamus, hippocampus, and the entorhinal and sensory cortexes (Resick). The amygdala is the primary center for processing and interpretating affective information as well as emotional memory (Rothschild, 2000).

Hypothalamic-Pituitary-Adrenal Axis. The hypothalamic-pituitary-adrenal (HPA) axis has received much attention in traumatic stress research and literature (Anisman, Griffiths, Matheson, Ravindran, & Merali, 2001). The HPA axis is a feedback loop that includes the hypothalamus, pituitary, and the adrenal glands. The main hormones that activate the HPA axis are corticotropin-releasing factor (CRF), arginine vasopressin (AVP), and adrenocorticotropin hormone (ACTH). The hypothalamus releases corticotropin-releasing factor (CRF), which in turn causes the pituitary to release adrenalcorticotrophic hormone (van Voorhees & Scarpa, 2004). This ACTH then stimulates the release of cortisol from the adrenal glands. This constitutes the completion of the loop via the negative feedback of cortisol on the hypothalamus and pituitary.

Impact of Exposure to Violence on the Brain

While the systemic physiological response to stress has been detailed above, there remains a need to understand how exposure to trauma can also have deleterious impact on the central nervous system. As a child experiences a traumatic event, the brain orchestrates an adaptive stress-mediating neural system, including the hypothalamic-

pituitary-adrenal (HPA) axis, central nervous system (CNS) noragrenergic (NA), dopaminergic (DA) systems and associated central and peripheral nervous systems (Carlson, 2007; van Voorhees & Scarpa, 2004). This well orchestrated response provides the adaptive emotional, behavioral, cognitive and physiological changes necessary for survival (Perry, 1999, p. 2). The neural system responds to prolonged and persistent stress by altering neurophysiological responses. After the occurrence of a traumatic event the physiological effects alter homeostasis, causing the child to become symptomatic. With repeated activation, the networks can become modified and the longer this altered state persists, the less likely the body is to return to its pre-trauma state, leading to subsequent clinical dysfunction (Resnick, 2001).

Neurochemical Changes in the Brain

There are a variety of physiological mechanisms by which aggression might occur in children exposed to trauma. It has been found, for example, that abundant stress-provoked release of cortisol into the circulation has a number of effects (Carion, Weems, & Reiss, 2007; van Voorhees & Scarpa, 2004). While an appropriate response and release of cortisol is necessary for survival, prolonged hypothalamic-pituitary-adrenal functioning can cause damage to the brain. In addition, cortisol causes an elevation of blood glucose. Cortisol negatively affects the immune system & prevents the release of immunotransmitters (Resick, 2001).

Blunted cortisol has been linked to the development of PTSD as a low cortisol response may allow arousal to perpetuate within the system therefore continuing the cycle of traumatic stress (Carlson, 2007). While studies with children and of

neurochemicals in general, have had inconsistent results, literature supports the interaction between traumatic and stressful environments, enhanced corticosteroid levels, and cell death (McNally, 2003; Nader, 2008).

Chronic stress has been linked to an overproduction of dopamine and decreased production of serotonin. While normal levels of dopamine are necessary to activate areas of the prefrontal cortex during the stress response process, too much has been suggested to cause decreased attention and learning capability, hyper-vigilance, and psychosis (McCollum, 2006). Serotonin is critical for the regulation of anxiety, fear, mood, and appetite (Nader, 2008); however, decreased levels in the prefrontal cortex have been attributed to persistent presence of stress leading to depressive and suicidal thoughts as well as aggression in youth (Scarpa & Raine, 2000).

Structural Changes in the Brain

The limbic system, as previously discussed, is comprised of the amygdala, hippocampus, cingulate gyrus, thalamus, hypothalamus, and putamen. These structures are most susceptible to the adverse effects of traumatic experiences during childhood (McCollum, 2006). The control and inhibition of emotion, along with interpretation of facial expression, fight or flight responses and the integration of emotional and physical reactions, are guided by the limbic system. The functioning of the limbic system also impacts implicit and explicit components of memory as well as learning.

The orbitofrontal and anterior cingulate cortexes have received recent attention in exposure and aggression literature (Solomon & Heide, 2005). The orbitofrontal cortex typically regulates areas of the hypothalamus that are associated with aggression.

Atypical development of the cortex and subsequent connections with the limbic system has been suggested to contribute to the dysregulation of affect and aggressive impulses. Anterior cingulate cortex activity has been linked to traumatic experiences and aggression as well (Solomon & Heide). Impaired connections between this area and the amygdala have been found to alter the inhibition of rage, leading to violent and aggressive behavior. While much remains unclear, anterior cingulate cortex functioning has also been linked to altered cognition, apathetic behavior, and the regulation if sympathetic activity (Luu & Posner, 2003).

Physiological Response

Heart Rate. While literature exists that documents the body's general response to stressors, the physiological response to exposure to violence has been under examined. In effort to better understand this interaction, Murali and Chen (2005) investigated the relationship between exposure and basal and reactive cardiovascular measures. The sample consisted of 115 high school students, where heart rate was obtained during two different tasks; a puzzle task and debate. Heart rate approached significance (p = .06), where increased basal heart rate was higher with increased frequency of exposure to violence, not proximity and severity of exposure. Heart rate has been identified as the most consistent physiological correlate to conduct and antisocial behaviors in children and adolescents (Raine, 2002); but has not been studied relative to its moderating potential between exposure to violence and the development of aggression. Scarpa and Ollendick (2003) found that aggression is related to increased baseline heart rate variability (HRV) and a decreased baseline heart rate (BHR). While heart rate variability

has been inconsistently replicated (Raine); decreased heart rate has been associated with aggression in adolescence and criminal activity in young adults, making it a risk factor even in the absence of social risk. Conversely, heart rate has been shown to be a protective factor against criminal behavior.

While the etiology of lower heart rate and aggression in less understood, a number of mechanisms have been speculated. It has been proposed that a decreased resting heart rate may be attributed to trait, biological, neurodevelopmental, and environmental factors (Raine, 2002; Scarpa & Raine, 2000). Specifically, it has been theorized that a lower resting heart rate could be due to autonomic under-arousal; therefore, requiring children with lower heart rates to seek out more stimulation than their peers. Raine, Reynolds, Venables, Mednick, and Farrington (1998) found that children with lower resting heart rates exhibited an increase in stimulating-seeking behaviors at age three and aggressive behaviors at age eleven. Reduced noradrenergic functioning and subsequent underarousal of the sympathetic nervous system has also been suggested. Fearlessness theory postulates that low levels of arousal during stress are indicative of lower levels of fear and that children that readily engage in aggressive and violent behaviors have an absence of fear regarding consequences and punishment (Raine).

Reduced right hemispheric functioning has been identified as a possible factor that contributes to lower heart rates given that it is the dominant hemisphere in controlling autonomic functions. Raine (2002) underscores lesions, neuropsychological, and imaging studies that support this claim. In addition, it has been previously shown that decreased right hemispheric functioning has been related to inefficiencies in systems that support withdrawal from danger. In lesions studies, patients with right hemisphere

damage show a significantly lower heart rate in response to negatively charged films (Raine).

Perceived Stress

Skybo (2005) examined the impact of exposure to violence on children, aged 7 through 14 years, from low-income areas. A cross-sectional, correlational design was employed to examine the relationship between witnessing violent acts and self-reported biopsychosocial reactions, which included stomachaches, anger, confusion, fatigue, and nervousness. Skybo found that nearly 95% of the children recruited witnessed acts of violence, which had positively correlated with self-reported stress symptoms.

Limitations in this study included the use of a convenience sample and self-report only measures of stress.

Manifestations of Post-Traumatic Stress Disorder

The trauma that is elicited in chronic and persistent exposure to violence is not captured in assumptions that underlie the current conceptualization of PTSD (Buka et al., 2001; Margolin & Vickerman, 2007). In much of the literature the use of this clinically descriptive terminology limits the ability to generalize findings to those that are exposed to ongoing urban violence (Buka et al.). While a community sample may not either meet criteria or even be screened for PTSD given that there may not be a single, identifiable, life-threatening event, the cumulative exposure and knowledge of imminent future threat, can cause a prolonged stress response in the body with pervasive consequences for some children (Margolin & Vickeman).

A study conducted by Lyons (as cited in Buka et al., 2001) regarding PTSD symptomatology found that children met criteria with as little as one traumatic event. These children exhibited decreased concentration, disruptions in sleep, hypervigilance, flash backs, increase in startle response, and altered attachment. In another study of children aged 6-12, those who witnessed domestic violence had at least a moderate amount of PTSD symptomatology. Observed frequencies compared to predicted frequencies showed a significant association between witnessing and a diagnosis of PTSD when compared to non-witnessing controls (Kilpatrick, Litt, & Williams, 1997).

Neurodevelopmental Changes from Exposure

Cognition, Exposure, and Aggression

Physiological stress has been shown to have a negative impact on learning and memory (DeBellis, Hooper, Spratt, & Woolley, 2009; Palmer et al., 1999). According to Perry (2001), a key factor to understanding learning in children exposed to violence is to consider that all people process, store, retrieve and respond to their environment in a "state-dependent fashion" (p. 10). When a child is in a state of arousal from chronic exposure to violence, the brain's ability to process information is very different to a child that is not in arousal. When hearing the same classroom instruction, the child that is in an alarm state will be less able to process verbal information the teacher is providing than the child that is in a calm state. This child's mental energy will be predominantly spent focusing on non-verbal cues like non-verbal gestures and facial expression, resulting in the appearance of a distracted child. As a result of this "use-dependant" pattern, the child will have disproportionally developed non-verbal skills than verbal abilities. This

capacity, often unknowingly deemed as "street smarts," develops out of adaptation resulting from perceived threat for the purposes of survival (Perry, 2005, p. 4).

Specifically, children raised in an environment of violence often learn that attending to non-verbal information is more crucial than verbal information (Perry, 2005).

Research has shown that almost 40% of children raised in environments that were chronically traumatic demonstrated a significant Verbal-Perceptual discrepancy on IQ testing (Perry, 2001). Not only does this have implication for the heightened perceptual skills and attention to threat, it also serves as a risk factor as the children may not have the verbal abilities to effectively communicate in times of confrontation (DeBellis, Hooper, Spratt, & Woolley, 2009; Villemarette-Pittman et al., 2002). A study highlighted by Henry, Sloane, and Black-Pond (2007) suggested that preschool aged children who experienced trauma were seven times more likely to be referred for therapy services secondary to delays in grammar, comprehension, and semantics.

Developed abstract verbal reasoning skills in children suggest an inherent ability to generate alternative ideas and subsequently it has been theorized that this skill can help a child to find and employ non-aggressive ways to manage conflict. Underdeveloped verbal reasoning skills have been specifically identified in children that exhibit aggressive behavior. Kikas and colleagues (2009) examined verbal reasoning skills, normative beliefs about aggression, and aggressive behavior in a group of fifth, seventh, and ninth grade children. The investigators found that lower verbal reasoning skills predicted aggressive behavior across grades, with the exception of indirect aggression in seventh graders.

Executive Function, Exposure, and Aggression

The prefrontal cortex primarily supports executive functioning while the striatum and limbic system play a critical role in the coordination of intellectual and emotional mechanisms (Paschall & Fishbein, 2002). Initiation, problem solving, and planning are key functions of the executive functioning system with deficits resulting in impaired regulation of behavior, diminished social skills, and poor judgment. Undeveloped executive function skills have been associated with maltreatment and exposure to violence as well as contributing to aggressive behaviors in children (Paschall & Fishbein; Seguin & Zelazo, 2005).

Teisl and Cicchetti (2008) found that in a number of studies, exposed children possessed a heightened vigilance and attention to aggressive stimuli. In addition, children who exhibit externalizing behaviors were found to be more perseverative suggesting that they may have difficulty in seeing things from different perspectives (Sequin et al., 2002). Working memory, non-verbal and verbal, have also been linked to aggressive behavior.

It is documented that children exposed to trauma and violence can misinterpret non-verbal cues because the tendency is to focus consistently on them, which can lead to overlooking other non-threatening contextual factors (Perry, 2001). In addition, it has been hypothesized that the diminished capacity to accurately interpret facial affective cues is linked to underlying neurodevelopment, neurophysiological, and executive functioning (Hoaken, Allaby, & Earle, 2007). Theories of social information processing and social cognition further cloud the ability to pinpoint one underlying mechanism in affective misrecognition but do not negate the functional implications.

In a study conducted by Pollack and Kistler (2002), affect recognition of children who had a history of direct exposure to violence (i.e., physical abuse) was compared to a group that had no reported exposure. The children were exposed to a series of computer-generated images that depicted faces with varying degrees of affective cues of happy, angry, sad, and fearful expressions. The investigators found that children with a history of physical abuse, tended to over identify anger.

Psychosocial Consequences of Exposure

Research has found that children who have experienced trauma show a behavioral regression and a negative attitude towards expectations and the future. In addition, as mentioned previously, children who are exposed to violence and maltreatment have been shown to misinterpret social cues, attend to hostile cues, and behave in an aggressive and withdrawn manner. Regardless of the cause of this alteration, all can impede social development and hinder interpersonal relationships (Pollak, 2004).

Social-Cognition

Children that have been exposed to violence have behavioral models that are aggressive. It is believed that a child may be able to reject these models if they have a sense of safety and trust present in their family (McCabe et al., 2005). Overstreet and colleagues (1999) examined the role of family support in moderating the effects of a child's exposure to violence in the community. African American children aged 10 through 15 were asked about maternal presence, family size, stress and depression, and exposure to community violence. The investigators used a regression analysis to assess

the moderating effects of family support and found that maternal presence did not significantly protect the child from developing PTSD symptomatology when there was an increase in exposure. This study emphasizes the need to consider other types of exposure that children may encounter in addition to community violence as well as the quality of familial relationships or support outside of the home as a potential moderator.

Social Information Processing

Social information processing's, as stated by Crick and Dodge (1994), purports that experiences shape one's cognitive schemas, beliefs, and attitudes that influence the way they behave and interact with others. In aggressive children, it is believed that ambiguous social situations and interactions are interpreted as hostile and threatening, resulting in aggressive behavior (Bradshaw, Rodgers, Ghandour, & Garbarino, 2009). Previous social information processing research has supported the idea that aggressive children attend to negative and threatening cues in the environment and overlook neutral ones. According to social learning theory, exposure to violence can inform, maintain, and perpetuate maladaptive social information processing biases (Bandura, 1977).

In a study conducted by Shahinfar, Kupersmidt, and Matza (2001), social information processing and exposure to violence was explored in a highly aggressive group of incarcerated adolescents. The investigators found that greater exposure and victimization was significantly related to increased hostile attribution of social information processing as well as increased aggressive beliefs.

Normative Beliefs

The importance of cognitive schemas, cognitive scripts, and the normative beliefs has been underscored in the way that a child interacts with his environment (Guerra, Huesmann, & Spindler, 2003). A child's belief that his or her surroundings are hostile and that aggression is acceptable, coupled with deeply established aggressive social scripts, sets the stage for aggressive behavior. Kikas and colleagues (2009) examined normative beliefs and children's expression of aggressive behaviors. Using a modified version of Housmann and Guerra's Normative Beliefs About Aggression Scale, the researchers assessed the beliefs of children ages eleven to sixteen. The researchers found that normative beliefs were related to physical and verbal aggression across age ranges, where those who perceived aggression as more acceptable, were more aggressive. Similarly, Guerra, Huesmann, and Spindler (2003) concluded that witnessing community violence had a detrimental effect on children's behavior as a result of imitation and the establishment of related cognitions.

Summary

As previously stated, exposure to violence has pervasive and deleterious effects on children that are just beginning to be understood by mental health care professionals and researchers. The purpose of this chapter has been to provide a discussion and critical review of literature relevant to the current study and demonstrate a gap in current research regarding the implications of exposure to violence. This chapter has detailed neurophysiological development and response to stress and exposure. An overview of the biopsychosocial model, which underlies the framework for the proposed study, was

provided. The specific neurophysiological, neurodevelopmental, and psychosocial factors that will be examined in this study were reviewed as they related to exposure to violence and aggression. Lastly, risk and protective factors were identified.

CHAPTER III

Methodology

The purpose of this chapter was to provide a detailed description of the methodological procedures to be employed in this study. Specifically, this chapter details the population of interest, the method of sampling, assessment procedure, and the psychometric properties of measures used. In addition, the study design, hypothesis testing, and statistic analysis for all hypotheses are provided.

Research Design

This investigation was an observational study that employed a quasi-experimental design to identify if a relationship exists between exposure to violence and aggressive behavior in young males reared in an urban setting. Specifically, measures of neurophysiological, neuropsychological, and social factors were examined by appropriate analyses to understand how these factors may or may not moderate the expression of aggressive behaviors.

Participants

A convenience sample of children, ranging in age from 12 to 15 years, were recruited from an urban elementary school located in northern New Jersey. The sample that was recruited had almost two times the national average in the amount of reported

violent crimes (City Rating, 2002). The median income is approximately \$26,000 with nearly 30% of the local community living below the poverty line (US Census Bureau, 2002).

The school serves approximately 500 students, ranging from Pre-kindergarten through eighth grade, and mostly of African American or Hispanic ethnicity. The vast majority (97%) of the students are eligible for discounted/free lunch, which indicates that many to most of the students and their families have median household incomes at or below the poverty level (State of New Jersey, 2009), thereby placing the children at risk for many poverty associated risk factors (Jensen, 2009).

Children and children of primary caregivers whose primary language was other than English were excluded from the study, as translation services were not accessible. In addition, children with severe developmental disabilities were also excluded from the study.

Procedure

Recruitment

After permission was granted from the appropriate IRB committees, permission was obtained from the principal of the school to hand out recruitment letters to the parents of children aged 12-15 in grades fifth, sixth, seventh, and eighth. Multiple forums were used in order maximize the number of participants. First, recruitment letters were provided to parents via students, who received the letter during their homeroom period. Second, the school social worker, parent liaison, and central office staff also provided letters for distribution to parents of students in grades fifth, sixth,

seventh, and eighth. Thirdly, letters were also given out to parents by the primary researcher at information sessions, parent-teacher organization meetings, and school functions. After a low response, permission was obtained to have information included in a school broadcast telephone message sent by the parent liaison. Interested parents were directed to the principal investigator for details of study participation. A sign-up sheet was also kept in the social work office to encourage participation and protect anonymity.

During the initial exchange, the principal investigator briefly summarized the purpose and nature of the study and offered the parent an opportunity to participate.

Parents were informed that they will receive \$20 compensation in the form of a gift card and the child participant would have a choice to receive a \$5 coupon for McDonalds or Dunkin Donuts following testing. Of the caregivers that responded, 5 chose not to have their children participate due to the content and disclosed obligation of the investigator to explore reports of abuse.

Parents who agreed to allow their child to participate completed the Informed Consent Form as it was reviewed and read aloud by the principal investigator to ensure understanding of materials. Parent and child permission were received independently so the child would not be aware that the study is about "aggression" to avoid influencing his behaviors. After parental or guardian consent was obtained, the child was asked to join the principal investigator in the testing room and was given the assent form. The principal investigator reviewed the form with the child to ensure comprehension of the informed consent and assent forms prior to issuing the assessment instruments. The child's teacher was asked to complete the Behavior Assessment Scale for Children, Second Edition – Teacher Report (BASC-2: TRS-A; Reynolds & Kamphaus, 2004).

Following completion of informed consent and assent procedures, testing commenced. First, the child was asked to take a seat at a small table located in the testing room. The room was free of potential environmental distractions. As this study incorporates both self-report and physiological instruments, care was taken to ensure the child participant understood the equipment being utilized. Thus, the heart rate equipment was explained by the examiner and then modeled for application and use on the examiner so the child knew what to expect. To begin the actual study, the lead for the heart rate monitor was placed on the child's finger. Next, the child observed a series of nature landscape images for 3 minutes total to collect a baseline heart rate. After completion, the lead was removed and the child was brought to a desk to complete the remaining measures. The assessments were administered in the following order by the principal investigator: (1) demographics, (2) Wechsler Abbreviated Scale of Intelligence (WASI, PsychCorp, 1999), (3) Animal Sorting subtest of the NEPSY-II (Korkman, Kirk, & Kemp, 2007), (4) Affect Recognition subtest of the NEPSY-II (Korkman et al., 2007), (5) Roberts Apperception Test for Children - Second Edition (Roberts-2; Roberts & Gruber, 2005), (6) Child Report of Post-traumatic Symptoms scale (CROPS, Greenwald, 1997), (7) Normative Beliefs about Aggression Scale (Huesmann, Guerra, Miller, & Zelli, 1989), (8) Screen for Adolescent Violence Exposure (SAVE, Hastings, 1996), (9) and the social support scale of the Resiliency Scale for Children and Adolescents (RSCA; Prince-Embury, 2005). Upon completion of the instruments, the gift card was provided. The entire assessment session lasted an average of 60-90 minutes in duration, including set up and removal of the physiologic equipment. No child demonstrated any signs of distress nor requested that the study be discontinued.

Research Instruments

The research battery consisted of self and teacher report measures, cognitive and neuropsychological measures, and physiological response measures which are detailed in this chapter. Specifically, the following were used: (a) demographic form, (b) Behavior Assessment Scale for Children-2nd Edition-Teacher Report (BASC-2: TRS-A; Reynolds & Kamphaus, 2004) (c) Screen for Adolescent Violence Exposure (SAVE; Hastings, 1996), (d) Child Report of Post-traumatic Symptoms scale (CROPS; Greenwald, 1997), (e) Wechsler Abbreviated Scale of Intelligence (WASI; PsychCorp, 1999), (f) NEPSY-II, affect recognition subtest (Korkman et al., 2007), (g) NEPSY-II, animal sorting subtest (Korkman et al., 2007), (h) Roberts Apperception Test for Children – Second Edition (Roberts-2; Roberts, 2006), (i) Resiliency Scale for Children and Adolescents, select subtest (RSCA; Prince-Embury, 2005), (j) measure of heart rate (Thought Technology-Procomp Infiniti, T7500M), (k) Normative Beliefs about Aggression Scale (Huesmann, Guerra, Miller, & Zelli, 1989).

Demographic Information

Demographic information was collected from the caregiver at the time of consent (Appendix A). Information regarding parent age, race, income, and education were collected. Children also completed a demographic form detailing age, handedness, grade, household members, social support, and sleep (Appendix B).

Measure of Aggression

that children and adolescents underestimate their aggression, a teacher report was used (Kikas et al., 2009). The Behavioral Assessment System for Children, Second Edition (BASC-2: TRS-A; Reynolds & Kamphaus, 2004) is used to ascertain information from a child's teacher regarding the child's attitude toward school, internalizing behaviors, inattention/hyperactivity, emotional symptoms, and personal adjustment. The teacher is required to answer whether statements are true or false as well as report frequency of occurrence to other items using a likert scale of Never, Sometimes, Often, or Almost Always response. The aggression scale was used for the purposes of this study. This scale looks at the child's tendency to act in a hostile manner, verbal or physical that is threatening to others (Reynolds & Kamphaus). Items assessing for aggression refer behaviors such as arguing, name-calling, threatening others, breaking others possessions, and hitting. For example, an item from this scale is, "Threatens to hurt others."

Standardization of the BASC-2 – TRS took place over a two-year period (2002-2004). Normative data was obtained from a sample size of 13,000 cases from over 375 sites across 257 cities and 40 states from various settings (Reynolds & Kamphaus, 2004). The general norm sample consisted of equal numbers of boys and girls and included 16.3% African American children. The BASC-2, TRS - Adolescent Form has good psychometric properties. Test-retest reliability had coefficients that ranged from .81 to .92 for the composite scales and had a reliability of .89 for the Aggression scale (Reynolds & Kamphaus). Inter-rater reliability for composite scores ranged from .55-.70 and .42 for Aggression scale. The Aggression scale is correlated with the oppositional

scale of the Conners' Teacher Rating Scale-revised at .94. Responses were entered into the BASC-2 computer scoring system and verified for accuracy. For this study, the aggression T-score was obtained from the results table was used in the analysis.

Measure of Exposure to Violence

Screen for Adolescent Violence Exposure (SAVE). Literature has consistently found that parental reports of their children's exposure to violence significantly underestimated exposure (Ceballo, Dahl, Aretakis, & Ramirez, 2001), specifically in the adolescent and young adult population where activities are less supervised (Crouter et al., 1999 as cited in Ceballo et al.). For this reason, child report of exposure is presumed to be the most reliable measure of exposure to violence.

The SAVE (Hastings, 1996) was developed to assess levels of exposure to violence in the home, school, and community (Hastings & Kelley, 1997). The SAVE has 37-items that utilize a 5-point Likert-style scale, and requires the child to assess the occurrence of violence exposure. An example of an item from this scale is, "Someone my age has threatened to beat me up." Each setting scale (i.e., home, school, and community) can be summed together, with total exposure ranging from zero to one hundred sixty. Higher scores reflect greater exposure to violence. Subscales can be further generated from each setting, but were not examined in this study. The SAVE has been shown to have strong reliability with .65 to .95 as the alpha coefficients range. Test-retest coefficients ranged from .53 to .92. Convergent and divergent validity was examined and determined to be sufficient given its significant correlations to related measures of violence and low correlations with unrelated ones (Hastings & Kelley).

Physiological Measures

Heart Rate Measure. Baseline heart rate measures were used to assess the child's physiological state. Thought Technology - Procomp Infiniti-T7500M software and equipment was used. For this study, the physiological measures of heart rate were ascertained as this is most supported in literature examining aggression in children and exposure to violence (Scarpa, Tanaka & Haden, 2008). Baseline heart rate was obtained from each participant by attaching the lead to the child's finger. Data was then sent to a DELL laptop computer using the accompanying technology software. Baseline heart rate was retrieved from the generated charts and tables and used for analysis.

Child Report of Post-traumatic Symptoms Scale (CROPS). The CROPS scale (Greenwald, 1997) was developed to serve as a brief, yet targeted tool to address the broad symptoms that children experience after exposure to traumatic events even when the traumatic event is not disclosed (Greenwald & Rubin, 1999). This measure is based on child trauma literature as well as the Diagnostic and Statistical Manual of Mental Disorders -Fourth Edition (DSM-IV; American Psychiatric Press, 1994) criteria for Post-Traumatic Stress Disorder (PTSD) but intended to serve both a clinical and community population. Children were required to rate, on a three-point scale, the presence of symptoms experienced in the last 7 days. A sum total was obtained and used for analysis. A sample item found on the scale is, "I find it hard to concentrate." Psychometric properties were sound for the CROPS measure. Item correlations ranged from .36 to .66 and were significant at the .001 level. Test-retest validity was .80 (p < .001). Criterion validity was supported for the measure as well (.60, p < .001) when compared to the Lifetime Incidence of Traumatic Events (LITE).

Neurodevelopmental Measures

Wechsler Abbreviated Measure of Intelligence (WASI). The Matrix Reasoning. Block Design, Similarities, and Vocabulary subtests were used from the WASI (PsychCorp, 1999; Sattler, 2001) to assess cognitive skills and specifically the presence of a verbal and performance discrepancy. For the purposes of this study, subtests were given and then the scores were used to determine if a discrepancy exists. Vocabulary is a subtest assessing verbal learning and requires the child to verbalize the meaning of words presented visually and orally. Vocabulary is believed to be a stable assessment therefore, making it a widely used indicator of intelligence; however, performance is influenced by schooling and culture (Groth-Marnat, 2009). The Similarities subtest assesses the child's verbal concept formation and abstract reasoning skills. The Block Design subtest measures the child's perceptual organization and requires the child to construct threedimensional block patterns from two-dimensional designs. The Matrix Reasoning subtest is a measure of nonverbal abstract reasoning and visual processing (Sattler, 2001). This subtest is considered less influenced by culture and requires only minimal visuomotor abilities (Groth-Marnat, 2009).

Standardization of the WASI was completed on a stratified national sample based on the 1997 U. S. Census and included 2,245 participants (Sattler, 2001). The WASI yields T-scores and IQ scores for the verbal and performance scales. The WASI has satisfactory internal consistency for subtests. Specifically, the reliability for Vocabulary = .89, Similarity = .87, Block Design = .90, and Matrix Reasoning = .92. In the children's sample, the stability coefficients for the subtests range from .76 to .84. The WASI has been correlated with other assessments of intelligence and ability.

Correlations between the WASI 4-subtest IQ and the WISC-III Full Scale IQ are reported as being .87 (Sattler, 2001). T-scores for the subtests were calculated from the raw score and then converted to VIQ and PIQ scores. The discrepancy between the scores were obtained and used for analysis.

The use of cognitive measures with ethnic minority children is an area that generates much debate (Groth-Marnat, 2009; Manly, 2005). It is posited that psychological tests and intelligence tests in particular, favor and reflect values of middle class, European Americans. While it has been found that European Americans have scored 12 to 15 points higher than their African American peers, underlying differences are often debated. In an effort to help mediate potential bias in assessments, tests have been developed that are considered more comparable across cultures. When intelligence tests are used, results must be interpreted cautiously and cultural bias must be considered.

For the purposes of this study, participating children were from the same community, thus helping to eliminate differences within the group of participants. In addition, Full Scale IQs were not reported. Subtest performance, specifically Vocabulary, may have been impacted by culture and environmental factors related to socioeconomic status but were analyzed as a moderating factor between exposure to violence and aggression.

NEPSY-II, Affect Recognition Subtest. The NEPSY-II was developed to serve as a complete assessment of neuropsychological functioning. For the proposed study, the Affect Recognition subtest will be used to examine the children's ability to discern between affective information. Research has shown that children that have been exposed

to violence have alterations that effect interpreting affective information as well as show a bias toward angry faces (Pollak & Kistler, 2002). Reading affective facial cues accurately is a critical component of social interaction (Johnson, Grossmann, & Kadosh, 2009; Korkman, Kirk, & Kemp, 2007).

The Affect Recognition subtest was designed to assess a child's ability to discern between common facial expressions that signal happy, sad, neutral, angry, disgust, and fear. The child is required to decide if expressions are the same or different between presented pictures of children's faces by pointing to their response which reduces the impact of language on this recognition task (Korkman et al., 2007).

Norms were developed from a US population of children aged 3-16 years (Korkman et al., 2007). Stratification of the sample was based on the 2003 census. Reliability coefficients were obtained utilizing split-half and alpha methods. Reliability for affect recognition had coefficients that ranged from .84 to .85 for ages twelve through fourteen. Standard error of measurement is 1.20. Stability coefficients were assessed to be .66 for 12 to 12:11 year olds and .49 for 13:0 to 16:11 year olds. Overall reliability and validity were good for the Affect Recognition subtest. Concurrent validity was assessed to be low with the WISC suggesting that Affect Recognition has discriminant validity, which supports this as a measure related to social perception and not intelligence.

The Affect Recognition Total Score was calculated by adding the total number of correct responses. This score was then transformed into a scaled score and used in analysis. A low score was interpreted as having less developed abilities to interpret

affective expressions. Raw scores for the incorrect identification of each emotion were obtained and analyzed for differences.

NEPSY-II, Animal Sorting Subtest. Literature has identified abstract thinking. problem solving, and perseveration as key components of impairment in those that exhibit aggression and are therefore, being examined in this study (Tremblay & Nagin, 2005). The Animal Card Sorting task is a measure of executive functioning that assesses initiation, cognitive flexibility, and self-monitoring (Korkman et al., 2007). Animal Sorting requires the child to sort cards into two groups of four cards using self-directed sorting criteria, assessing the child's concept formulation and ability to shift set. This test was designed specifically for children, depicting pictures of animals in various contexts. It requires the child to work intermittently for 360 seconds. The primary score, Combined Scaled Score, was generated from Total Correct Sort and Total Errors scores. The raw score was then converted into a scaled score, which was used in analysis, where low combined scale scores will be interpreted as less developed cognitive flexibility, selfmonitoring, and conceptual knowledge. The process score of Repeated Sort Errors was obtained by summing sorts that are repeated. This raw score was used to further assess cognitive flexibility and self-monitoring and was be interpreted where high scores suggest poorer flexibility and monitoring (Korkman et al.).

Standardization for the NEPSY-II, as previously described, was conducted based on the 2003 census (Korkman et al., 2007). Reliability on the Animal Sorting subtest was assessed to be .96 for children aged twelve through fourteen. Standard error of measurement was reported as 1.56 for 12 year olds and 1.80 for those 13 to 14 years of

age. Stability coefficient (corrected $r^b = .73$) for those 11-12:11 and .64 for those 13:0-16:11 years of age.

Psychosocial Measures

Roberts Apperception Test for Children-2 (Roberts-2). The Roberts-2 cards (Roberts, 2006) have been used to assess a child's social perceptions and social understanding by requiring them to tell a story about a situation that is depicted on the stimulus cards. The story is then interpreted as a reflection of the child's social cognitive competence. The psychometric properties for the Roberts-2 have been strengthened from the original version. The standardization sample for the Roberts-2 included 1,060 individuals ranging from 6 to 18 years of age. A representative sample, determined from the US Census, was used and was sensitive to parent educational level and ethnicity (Roberts & Gruber, 2005). Retest reliability was assessed at .75 with broad scale coefficients ranging from .80 to .85.

In the current study, children were asked to view 6 cards, 3 that specifically pull for aggression (#9, 13, & 14) and three that are neutral or positive (#2, 5, & 16). Their aggression score was calculated from the total number on aggressive and angry attributes mentioned in the narrative of the cards. A total number was generated and used for analysis. Since a possibility exists that prior exposure to negative images can prime an individual to further report negative responses in subsequent images (Green, 1981), order of stimulus presentation were altered.

Resiliency Scales for Children and Adolescents, Select Subtest (RSCA). The Resiliency Scales for Children and Adolescents developed by Sandra Prince-Embury (2005) was designed to assess characteristics of personal resiliency in children and adolescents between the ages of nine and eighteen. It is composed of three self-report questionnaires: Sense of Mastery, Sense of Relatedness, and Emotional Reactivity. The present study examined the total score on the Sense of Relatedness Scale only to assess the child's perception of social support. Relatedness has been linked to resiliency after adverse events (Prince-Embury, 2010). This scale specifically aims to assess the child's perceived social support, ability to trust others, and tolerance to other's differences. Raw scores were totaled and transformed in to T-scores, which were used in analysis.

The scales of the RSCA were standardized with nine norm groups: total sample, females divided into four age strata, and males divided into four age strata (Prince-Embury, 2008). This group consisted of 450 (9-14) children and 200 (15-18) adolescents who comprised a group that was representative of the general population, including 5% with clinical diagnoses, but not in treatment. The sample was also matched to the general population on race/ethnicity and caregiver education level. Ethnicities broke down as follows: 59% White, 18% Hispanic, 17%African American, and 6% other (Prince-Embury). Chronbach's alpha coefficient ranged from .93 to .95 for the total sample, suggesting good internal consistency. Test-retest reliability ranged from .70 to .92 on a sub-sample of adolescents. Convergent and divergent validity were established among resiliency measures (Brown, 2001).

Normative Beliefs about Aggression Scale. This 20 question, self-report scale, utilizes a Likert scale assessing the child's beliefs about aggressive behaviors (Huesmann, Guerra, Miller, & Zelli, 1989). This scale can be administered in both individual and group settings and has been developed for individuals aged 6 through 30 years. For this study, the scale was administered individually. A total score was obtained by adding responses and used for analysis. The total score represents the child's beliefs about aggression where higher scores are indicative of a belief that aggressive behavior is normal. Items from this scale give a short, one sentence scenario and ask such questions as, "Do you think it is OK for John to hit him?"

Norms were derived using a sample of 1,550 individuals from mid-size and large Midwestern cities, which were predominantly African American, Caucasian, and Hispanic of lower socioeconomic level (Huesmann et al., 1989). While validity information is not published, internal consistency was measured at 0.65 to 0.85. The author granted permission for use of the scale.

Power Analysis

Power analyses for this study were performed using the computer program G*Power (Faul, Erdfelder, Buchner, & Lang, 2009) for each research hypothesis according to statistical analysis procedures. Details for each analysis are provided below, aggregated by hypothesis. In order to ensure sufficient participants were recruited, the hypothesis requiring the largest number of participants was used to establish the minimum sample size required.

Hypothesis 1: It is predicted that the population examined in this study has been exposed to violence. Descriptive statistics were used to assess exposure to violence, thus a power analysis was not performed for this prediction.

Hypothesis 2: There will be a significant relationship between exposure and aggression with greater exposure equated with greater levels of aggression. This was proposed to be tested using a Pearson bi-variate correlation. Power analysis indicated that a sample size of 64 participants has a power of 0.80 and an alpha = .05.

Hypothesis 3: The effect of exposure on aggression will be moderated by physiological stress response where greater self reported stress responses and lower baseline heart rate are related to greater aggression in children exposed to violence. A multiple regression analysis was originally proposed to examine this hypothesis. Power analysis indicated that a regression model with 3 variables, effect size of .30, alpha = .05, will have a power of .80 with 41 participants (45 participants with power of .85).

Hypothesis 4: The effect of exposure on aggression will be moderated by neurodevelopment where higher verbal-perceptual discrepancy, lower executive functioning scores, and increased perseveration scores will be related to greater aggression in children exposed to violence. This hypothesis was intended to be assessed using a multiple regression analysis. Power analysis indicated that a regression model with 5 variables, alpha = .05, and effect size of .30 will have a power of .80 with 49 participants (54 participants will yield power of .85).

Hypothesis 5: The effects of exposure on aggression will be moderated by social support where lower social support, higher scores of perceptions of aggression in social scenes, and higher normative values of aggression will be related to greater aggression in

children exposed to violence. It was anticipated that this hypothesis would be assessed by using a multiple regression analysis. Power analysis indicated that a regression model with 4 variables, alpha = .05, would have a power of .80 with 45 participants (50 participants will yield a power of .85).

Overall, these analyses suggested that a sample size of 64 participants would yield sufficient power to test the study hypotheses with medium effect size.

Statistical Analysis Plan

Descriptive analyses were computed in the form of frequency counts, range, means, and standard deviations, as appropriate, for primary demographic variables and primary study outcome variables. Following computation of descriptive statistics, inferential analyses were conducted to test study hypotheses.

Hypothesis 1, which examined the level of violence participants were exposed to, was assessed using descriptive statistics. Hypothesis 2, which examined the bi-variate relationship between exposure to violence and aggression, was tested using a Pearson bi-variate correlation. Subsequent hypotheses, which examine for a moderating relationship among study variables, were proposed to be assessed using the well-documented, Baron and Kenny methodology (1986) (Frazier, Tix, & Barron, 2004). This technique holds that a moderating relationship is present when a number of pre-specified assertions about the variables are all demonstrated to be true. For the purposes of the present study, a multiple regression analysis, with standard entry of predictors, was initially planned to test the assertions and determine if physiological, neuropsychological, and/or psychosocial factors moderate the relationship between violence exposure and aggression

(Figure 1). In order to utilize the Baron and Kenney methodology, predictors are entered, along with moderator terms, which then are computed as

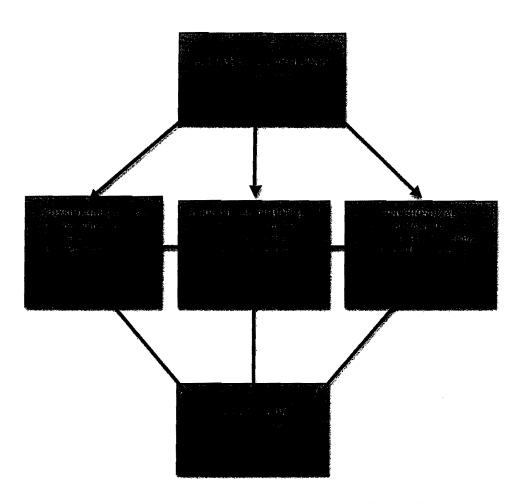


Figure 1: Biopsychosocial Model Of Aggression In Children Exposed To Violence

the product of the predictor and the moderator variable. Independent variables and product terms (i.e., moderators) were proposed regress simultaneously on dependent variables in order to understand the presence of a moderation effect.

Statistical analyses for each of the study hypotheses, additional exploratory analyses involving demographic factors were conducted, as appropriate, to understand how primary study variables and findings might vary as a function of demographics.

This hypothesis generating exercise served to provide data to suggest areas for future

exploration.

Significant demographic relationships should not have influenced regression analyses, as this study had been powered to detect effects for the number of pre-planned predictors, thus, potentially expanding the number of predictors may negatively impact statistical power and create the possibility for Type II error.

Summary

The purpose of this chapter has been to detail the methodology that was initially proposed in the current study to examine the moderating effects of physiological, neurodevelopmental, and psychosocial variables between exposure to violence and aggression. The participants that were recruited and the population of interest were discussed. The details of sample size and power analysis were included. The procedures for data collection, assessment battery, and psychometric properties of the measures being used were reported. Lastly, the study design and anticipated statistical analysis for each hypothesis was detailed.

CHAPTER IV

Results

Descriptive Statistics

Study Sample

The present study recruited 27 participants from an urban community pre-kindergarten to eighth grade school in the New York area. An *a priori* power analysis indicated that 64 children were required to adequately power the study. Among the parents that were provided information on the study, consent was obtained for 29 children to participate, of which 27 completed the study and comprised the per protocol sample used for data analysis. One child transferred to another school prior to assessment session and one child incorrectly characterized his age, thereby disqualifying him for the study. This study encountered significant recruitment challenges that resulted in a study sample that was smaller than planned. Implications for statistical analyses employed and data interpretation shall be detailed later within this Chapter and the Discussion.

Demographics

Table 1 and Table 2 present demographic data for the overall sample. As observed in the Table, the overall sample was comprised of males, between the ages of 12 and 15, with at least 5th grade education. The average age of participants was 13.1 years. The

highest education attained by caregivers was a high school diploma (n = 12, 46.2%). Seventy-five percent of caregivers had a household income of \$30,000 or less.

Table 1

Demographic Characteristics of the Sample (n = 27)

	M	SD	f
Child Age	13.1		
Participant Gender			
Male			27
Female			0
Race/Ethnicity			
African American			26
White			0
Hispanic			1
Pacific Islander			0
Asian			0
American Indian/ Alaskan			0
Native			

Table 2

Demographic Characteristics of Caregivers

The state of the s	М	SD	\overline{F}	%
Caregiver Age	36.6	6.5	***	
Relationship Status				
Married			3	11.5
Single			12	46.2
In a relationship			5	19.2
Living with partner			2	7.7
Divorced/Separated			3	11.5
Partner/Spouse deceased			1	3.8
Highest Level of Education				
Grammar School			1	3.8
High School			12	46.2
High School Equivalent			6	23.1
College Degree			5	19.2
Graduate Degree			2	7.7
Current Employment				
Full time			9	42.9
Part time			5	23.8
Not employed			6	28.6
Full time student			1	4.8

M	SD	F	%
		7	35
		8	40
		3	15
		0	0
		0	0
		2	6.7
		0	0
		0	0
	M	M SD	7 8 3 0

Primary Study Variables

Prior to conducting inferential statistics, descriptive statistics for the primary study variables were obtained and displayed in Table 3.

Table 3

Descriptive Statistics for Primary Study Variables

	Mean	Standard Deviation	Skewness	Kurtosis
BASC Aggression	58.93	15.69	1.52	1.95
Exposure to Violence	61.93	52.79	1.03	.17
Heart Rate	80.33	13.92	003	.87
WISC Verbal- Performance Discrepancy	4.037	13.05	.226	189

	Mean	Standard Deviation	Skewness	Kurtosis
NEPSY Affect Recognition Scaled Score	8.26	2.90	.129	038
NEPSY Animal Sorting	6.37	3.25	068	05
Roberts Total Aggression Score	10.33	5.64	2.99	11.98
CROPS Total Score	18.41	7.56	039	.586
Related Scale T-score	35.33	14.49	271	047
Normative beliefs about Aggression	37.41	8.19	.633	332

Hypothesis Testing

Hypothesis 1. This hypothesis predicted that among the sample recruited for this analysis, the majority of participants would endorse exposure to violence. Descriptive statistics were examined to assess the degree to which participants endorsed exposure and are detailed in Table 3. As shown in table, participants of this study endorsed that they were exposed to violence (n = 27; mean = 61.93; SD = 52.79); however, the degree of exposure was less than predicted. This level of endorsement may represent an underestimation of actual exposure as significant limitations existed related to participant response and will be detailed in the Discussion chapter.

Hypothesis 2. This hypothesis stated that there will be a significant relationship between exposure to violence and aggression. This hypothesis was tested using a Pearson Bi-variate correlation to determine if a relationship exists between the variables of interest.

This study did not find support for this hypothesis. There was no correlation between exposure to violence and aggression, r(2) = .203, p = .310 (Table 4). As a further examination of the data, the analysis was repeated using a one-tailed approach. As observed in Table 4, even when using a one-tailed test the two primary variables are not significantly related, r(2) = .203, p = .155. The lack of statistical significance within this hypothesis has impact on subsequent study hypotheses, which proposes a moderating relationship between study variables. Specifically, the other proposed factors cannot be moderators given the non-significance of this hypothesis.

Table 4

Bivariate Pearson Correlations of Primary Variables (One-Tailed and Two-Tailed)

	Pearson Correlation	Significance (2-tailed)	Significance (1-tailed)		
Aggression and Exposure to	.203	.310	.155		
Violence					

Hypothesis 3. Hypothesis 3 stated that the effects of exposure to violence on aggression will be moderated by physiological stress response where greater self reported stress responses and lower baseline heart rate will be related greater aggression in children exposed to violence. This hypothesis was originally planned to be tested by

using a multiple regression analysis. Given the previously discussed challenges in recruitment and lack of significance between the primary study variables, Pearson Bivariate Correlations were used. Results are represented in Table 5 below.

Table 5

Table of Bivariate Correlations for Physiologic Variables

		Heart Rate	Reported Stress (CROPS Total Score)
Exposure to Violence	PearsonCorrelation	119	.509
	Sig. (2-tailed)	.554	.007
	N	27	27
Aggression	PearsonCorrelation	066	087
	Sig. (2-tailed)	.742	.665
	N	27	27

Note. CROPS = Child Report of Post-Traumatic Symptoms Scale

Results of this study did not support the relationship between baseline heart rate and exposure to violence, r(2) = -.119, p = .554 or baseline heart rate and aggression, r(2) = -.066, p = .742. However, when a bi-variate correlation was employed to the variables of exposure to violence and the self-report measure of post-traumatic symptoms, a significant relationship was found, r(2) = -.509, p = .007. No relationships were present between the self-report measure of exposure and aggression, r(2) = -.087, p = .665.

Hypothesis 4. Hypothesis 4 predicted that the effect of exposure to violence will be moderated by neurodevelopment where higher verbal-perceptual discrepancy, lower executive functioning and increased repeated error scores will be related to increased aggression in children exposed to violence. This hypothesis was also originally planned to be tested by using a multiple regression analysis. For reasons previously mentioned, Pearson Bivariate Correlations to analyze variables. Results are represented in Table 6 and Table 7 below.

Table 6

Table of Bivariate Correlations for Neurodevelopmental Variables

Exposure to Violence	Pearson Correlation	WASI Verbal- Performance Difference .009	NEPSY Animal Sorting 015	NEPSY Affect Recognition .260
	Sig. (2-tailed)	.963	.941	.191
	N	27	27	27
Aggression	Pearson Correlation	420	.233	.186
	Sig. (2-tailed)	.029	.242	.352
	N	27	27	27

Note. WASI = Wechsler Adult Scale of Intelligence; NEPSY = A Developmental Neuropsychological Assessment.

Table 7

Table of Bivariate Correlations for Neurodevelopmental Process Variables

		NEPSY Repeat Sort	Happy Errors	Sad Errors	Neutral Errors	Fear Errors	Anger Errors	Disgust Errors
Exposure to Violence	Pearson Correlation	106	187	370	035	319	170	399
VIOICHEC	Sig. (2-tailed)	.597	.351	.057	.862	.105	.395	.039
	N	27	27	27	27	27	27	27
Aggression	PearsonCorrelation	082	357	.155	253	417	.013	038
	Sig. (2-tailed)	.685	.068	.441	.203	.030	.948	.852
	N	27	27	27	27	27	27	27

Note. NEPSY = A Developmental Neuropsychological Assessment.

Bi-variate analysis showed a significant relationship between Verbal-Perceptual (V-P) discrepancy and the amount of aggression reported, r(2) = .420, p = .029 yet a relationship did not exist between V-P discrepancy and exposure, r(2) = .009, p = .963. Analysis did not show a significant relationship between NEPSY animal sorting and the amount of aggression reported, r(2) = .015, p = .941 nor was there a relationship between NEPSY animal sorting and exposure, r(2) = .233, p = .242. While a relationship did not exist between the primary variables of NEPSY Affect Recognition, aggression, and exposure, secondary relationships did exist. Within the affect recognition task, a significant relationship existed between exposure to violence and disgust errors, r(2) = .399, p = .039 as well as between aggression and fear errors, r(2) = .417, p = .030.

Hypothesis 5. Hypothesis 5 predicted that the effect of exposure to violence will be moderated by psychosocial factors where lower social support, higher scores of perceptions of aggression in social scenes, and higher normative values of aggression will be related to increased aggression in children exposed to violence. This hypothesis was also originally planned to be tested by using a multiple regression analysis. As discussed earlier, Pearson Bivariate Correlations were used to analyze variables. Results are represented in Table 8.

Table 8

Table of Bivariate Correlations for Psychosocial Variables

**************************************		Normative Beliefs Scale	Relatedness Scale	Roberts Cards
Exposure to Violence	PearsonCorrelation	.563	.337	054
10101100	Sig. (2-tailed)	.002	.085	.788
	N	27	27	27
Aggression	PearsonCorrelation	.172	322	.128
	Sig. (2-tailed)	.390	.101	.525
	N	27	27	27

Analysis showed that exposure to violence and aggressive themes embedded in stories were not significantly related, r(2) = -.054, p = .788. The relationship between aggressive themes and reported aggression were also not significant, r(2) = .128, p = .525. Bivariate correlations revealed a significant relationship between exposure to

violence and children's normative beliefs about aggressive behaviors, r(2) = .563, p = .002.

Exploratory Analysis

In an effort to further examine for relationships among study variables, additional exploratory analyses were employed, outside of *a priori* study hypotheses. Specifically, Table 9 displays intercorrelation values for primary study variables to determine if there are any additional significant relationships that might support future studies. The results of this family-wise correlation analysis suggest a number of incremental significant findings; however, inspection of significant findings indicates that most relationships were between subscales of the intelligence assessment. It was expected that WASI scores should correlate, and therefore this did not provide for additional unexpected findings or results that suggest new or additional hypotheses for further inquiry.

Table 9
Intercorrelation Values for Primary Study Variables

	1	2	3	4	5	6	7	8	9	10
BASC Total Score	1.00	-0.34	0.04	-0.16	0.09	0.13	-0.09	0.14	-0.34	0.18
2. WASI Verbal IQ		1.00	0.41*	0.84**	0.55**	-0.16	-0.01	-0.16	0.43*	0.08
3. WASI Performance IQ			1.00	0.82**	0.32	-0.10	0.05	-0.30	0.40*	0.05
4. WASI Full 4 IQ				1.00	0.48*	-0.16	0.03	-0.22	0.50*	0.08
5. NEPSY Affect Recognition Raw					1.00	0.01	0.21	-0.19	0.33	0.29
6. Roberts Total Score						1.00	0.47*	-0.07	-0.23	-0.06
7. CROPS Total Score							1.00	0.39	0.26	0.51*
8. Normative Beliefs About Aggression								1.00	0.10	0.54**
9. Rel Scale Raw									1.00	0.39
10. SV Total Score										1.00

Note. * denotes p < 0.05. ** denotes p < 0.001.

CHAPTER V

Discussion

It has been well established that children are being exposed to violence at an epidemic rate, with those in urban communities at the greatest risk. Exposure has been linked to the development of post-traumatic stress symptoms (Overstreet, 2000), externalizing and internalizing behaviors, poor academic outcomes, physical and mental illness (Watts-English et al., 2006), and juvenile delinquency (Lee & Hoaken, 2007). Exposure to violence has been associated with behavioral consequences, neurobiological and neurocognitive changes, and altered social perception. The present study attempted to address the gap in scientific knowledge by employing a biopsychosocial perspective to examine how exposure to violence in children relates to aggressive behavior. Specifically, the extent to which these biological, psychological, and/or social factors moderate the relationship between exposure and aggression was examined.

This study was based on the premise that there would be a relationship between child exposure (self-report) and a teacher report of aggression. This study did not find such a relationship. By extension, there was no evidence of a moderating effect of physiological, neurocognitive, and social factors. Numerous factors may account for this lack of significance. Specifically, underreporting of exposure by children in the recruited sample was a concern as children were observed to not endorse any exposure at home and school despite investigator knowledge to the contrary. Exposure in the community may have also been underreported as crime rates of the city are significantly higher when

compared to the United States average (Neighborhood Scout, 2012). Another limiting factor was low participation secondary to the poor parental response and amenability to have their children participate, a phenomenon that will be described further within the study limitations section. Given these challenges, the results of the study should be interpreted with caution and viewed within the context of these limitations.

Discussion of Hypotheses

Hypothesis 1

The initial hypothesis sought to determine if and to what extent the participants were exposed to violence as measured by a self-report questionnaire. Literature and statistics suggest that children living in an urban population are exposed at a pervasive rate (Margolin & Gordis, 2004; Overstreet, 2000; Skybo, 2005). While exposure was not defined consistently in previous research, the present study was aimed to capture the cumulative exposure of the adolescent boys.

As expected, children did endorse exposure to violence; however, it was not at a rate consistent with community crime statistics, examiner experience of violence in the school, nor, in some cases, knowledge of reported violence at home. Community crime rates are estimated to be 5.9 for robbery, .32 for murder, and 4.10 for assault per 1,000 people within the community from which this sample was obtained. These rates are significantly higher when compared to the United States average, which is 1.19, .05, and 2.25 respectively. A total of violent and property crimes for the community sampled was 45.84 where as the national average was a mere 4.0 (per 1,000 people) (Neighborhood Scout, 2012). Despite these facts, some children endorsed little or no exposure to

violence.

It was observed that some children completed the questionnaire by first circling "0's" for all of the items related to violence at home and then completed the other items. Children were also observed to cover their answers with their hand suggesting a level of distrust or discomfort with the examiner. Therefore, while exposure to violence was endorsed, the actual reported cumulative exposure in this study may be an underrepresentation of actual experiences, which may have impacted the results of subsequent analyses.

Hypothesis 2

The second hypothesis postulated that a relationship would exist between endorsed level of exposure and teacher reported aggression levels where higher levels of exposure would be correlated to higher levels of aggression. Previous literature has provided inconsistent findings between children exposed and the presence of externalizing behaviors, where it has been found that children aged 7-12 did not exhibit externalizing behaviors after community exposure yet others found that a significant relationship existed between exposure to community violence and the development of conduct disorder (Lynch & Cicchitti, 1998; McCabe et al., 2005). It was suggested that inconsistencies might have been an effect of the inclusion or exclusion of different contexts of exposure (community violence, school) leading to faulty conclusions about the impact of direct exposure. When exposure was examined across contexts (i.e., home, school, and community) it was found to be more predictive of outcomes than context specific information (Mrug et al., 2008). Thus, this current study was aimed to capture

cumulative exposure in an effort to gain a more accurate level of exposure in children of urban communities.

The results of Hypothesis 2 did not show that a significant relationship existed between exposure to violence and aggression in this study. As stated previously, inconsistencies were present in literature; however, by examining cumulative exposure, this was not an expected outcome. Some possible explanations may account for this insignificant finding. It can be speculated that endorsed exposure may not adequately represent actual exposure. Another issue may be related to the teacher report of aggression. While studies have shown that teacher report is more reliable than adolescent self-report (Kikas et al., 2009), the possibility exists that teacher bias and/or normalization of behaviors in the environment may have been a confounding factor.

While recruitment was a major challenge and resultant limitation, the degree of insignificance between the main study variables as well as observed behavior during the completion of the self-report measure, suggest that there was not even a weak relationship. Since there was not a significant relationship between the primary study variables, Hypotheses 3, 4, and 5 were not proven.

Hypothesis 3

The third hypothesis stated that the effect of exposure on aggression would be moderated by physiological stress response where greater self reported stress responses and lower baseline heart rate are related to greater aggression in children exposed to violence. Since there was not a significant relationship between the primary study variables, Hypotheses 3 was not supported; however, variables were examined for bi-

variate correlations between each primary variable and each of the secondary variables independently.

The relationship between heart rate and aggression has been supported in the literature. For example, as previously discussed, Scarpa and Ollendick (2003) found that aggression is related to decreased baseline heart rate (BHR). Similarly, others have found decreased heart rate has been associated with aggression in adolescence and criminal activity in young adults, making it a risk factor even in the absence of social risk (Raine, 2002). This study did not support the relationship between lower baseline heart rate and exposure to violence or aggression. As discussed, factors related to the primary variable measures may have contributed to the negative findings.

A relationship between exposure to violence and posttraumatic stress symptoms has also been consistently found across literature (McDonald & Richmond, 2008). The impact of exposure to violence has been directly tied to the development of trauma related symptoms, Post-Traumatic Stress (PTS), and Post-Traumatic Stress Disorder (PTSD) (McDonald & Richmond; Solomon & Heide, 2005). It has been found that children from low-income communities who witnessed violent acts reported biopsychosocial reactions, which included stomachache, anger, confusion, fatigue, and nervousness. In one study nearly 95% of the children recruited witnessed acts of violence, which had positively correlated with self-reported stress symptoms (Skybo, 2005).

When a bi-variate correlation was employed to the variables of exposure to violence and the self-report measure of post-traumatic symptoms, a significant relationship was found. Given the limitations of the current study, a moderating

relationship could not be examined or speculated; however, when a bi-variate correlation analysis was performed, results were consistent with literature. Higher endorsements of exposure were related to an increase in self-reported symptoms related to post-traumatic stress.

While the results of Hypothesis 3 must be viewed within the context of previously mentioned experimental confounds of the primary study variable measures, bi-variate correlation relationships were not present between the individual factors of heart rate, aggression, and exposure to violence, but a significant relationship did exist between endorsed exposure and the child's reported post-traumatic symptoms. As discussed, factors related to the primary variable measures may have contributed to the lack of significant findings and may have underrepresented the significance between exposure and post-traumatic symptoms.

Hypothesis 4

Hypothesis 4 posited that the effect of exposure on aggression would be moderated by neurodevelopment where higher verbal-perceptual discrepancy, lower executive functioning scores, and increased perseveration scores will be related to greater aggression in children exposed to violence. Since there was not a significant relationship between the primary study variables, Hypotheses 4 was not supported; however, variables were examined for bi-variate correlations between each primary variable and each of the secondary variables independently.

Hypothesis 4 was aimed to examine the possible moderating relationship that neurocognitive factors have on the exposure to violence and aggression. As previously

discussed, Hypothesis 4 was not examined as a moderating factor between exposure to violence and aggression, but instead by a bi-variate correlation. This analysis showed a significant relationship between Verbal-Perceptual discrepancy and the amount of aggression reported. This finding is consistent with previous findings, as research has shown that under-developed verbal reasoning may be a contributory factor to aggressive behavior. It is suggested that more developed perceptual skills and less developed verbal skills may create a situation where a child has a heightened attention to non-verbal cues but may not have the skills to verbally negotiate during conflict (Kikas et al., 2009).

In numerous studies, executive function irregularities have been present in children that are more aggressive. Specifically, children who exhibit aggressive behaviors were found to be more perseverative and possessed less developed problem solving skill, suggesting that they may have difficulty generating alternative perspectives (Sequin et al., 2002). Furthermore, young adults that presented with more disruptive behaviors showed less developed organizational and planning abilities (Villemarette-Pittman et al., 2002). While a relationship did not exist between the executive functioning factors proposed in this study, within the affect recognition task, a significant relationship did exist between exposure to violence and disgust errors as well as between aggression and fear errors. These relationships are supported by the literature as children who were exposed to violence were found to show a tendency toward misperceiving affective cues (Teisl & Cicchetti, 2008).

The results of Hypothesis 4 revealed that, while secondary relationships exist, there was an overall lack of significant findings between primary variables and individual factors initially proposed as moderators. While this is contrary to expectation, the overall

lack of significant findings may be a result of primary variable measurement limitations.

As such, results must be interpreted within this context.

Hypothesis 5

Hypothesis 5 stated that the effects of exposure on aggression would be moderated by social support where lower social support, higher scores of perceptions of aggression in social scenes, and higher normative values of aggression will be related to greater aggression in children exposed to violence. Since there was not a significant relationship between the primary study variables, Hypotheses 5 was not supported, however, variables were examined for bi-variate correlations between each primary variable and each of the secondary variables independently.

Literature suggests that when children possess distortions within social information processes, there is a stronger likelihood of aggressive behavior. Specifically, when a child inaccurately perceives, interprets, and bases conclusions about social exchanges, they may exhibit more aggression (Nader, 2008). Research has shown that social and family support can be a moderator in exposure to violence as children that identified positive social support were less likely to experience posttraumatic stress symptoms and aggression (Pollak, 2004). Lastly, normative beliefs of aggression were been viewed as the vital component of a child's development of aggressive behavior after exposure to violence (Guerra, Huesmann, & Spindler, 2003).

Results of bi-variate correlations between exposure to violence, measures of social support and aggressive themes embedded in stories, were not significant; however, exposure to violence was found to be related to children's normative beliefs about

aggressive behaviors which is consistent with previously discussed literature. While a relationship was not seen between exposure and normative beliefs, the already suggested under-endorsement of exposure may have contributed to the insignificant findings.

Limitations

This study was based on the premise that there would be a significant relationship between children's endorsed exposure and a teacher report of aggression. As previously described, a significant relationship did not exist between these two variables, thereby negating the possibility of a moderating effect by the physiological, neurocognitive, and social factors. External factors may have contributed to this lack of significance.

Specifically, underreporting of exposure by the children was a concern as children were observed to not endorse any exposure at home and school despite investigator knowledge of the contrary. Exposure in the community may have also been underreported given the deleterious crime rates of the city in which the participants live. Given the lack of caregiver involvement and agreement to participate, those that were not captured in this study may have more exposure and/or aggressive behaviors.

Another limiting factor was low participation, secondary to the lack of parental response and amenability to have their children participate. Despite numerous methods of contact and attempts to recruit subjects, caregivers did not respond to solicitation. Some of those who did respond, refused participation stating that "it was nobody's business to know." Fear of being reported to child protective services was also raised as a concern by caregivers as was a general sense of distrust to have the child participate in a study being conducted by an examiner from outside of the community.

Culture may have been an underlying cause of the lack of willingness to participate. Cultural differences have been shown to have an impact of research (Scott-Jones, 1994). One of the most debated topics in conducting research with children is reporting suspected abuse and/or harm. While ethics guidelines serve to protect the child's welfare, reporting can be equally as damaging. Ethnic minority groups may view reporting as an extension of harm experienced from participating in past research, which can perpetuate fear and distrust (Scott-Jones). Despite having an already established rapport and presence in the school, the investigator's race may have contributed to the lack of trust by the caretakers, thus serving as deterrent for participation. One study showed that parents of children in urban schools did not trust outside help of any ethnic group. These parents went on to detail incidences where they believed that these individuals violated trust or even caused serious injury to loved ones (Horowitz, McKay, & Marshall, 2005). In an effort to counteract the effects of mistrust, the parent liaison and trusted members of the school staff were used to bridge the communication gap between caretakers and the this investigator. This gap is said to be the result of distrust and skepticism toward outside "experts" (Atkins Frazier, Leathers, Graczyk, Talbott, Jakobsons, Adil, Marinez-Lora, Demirtas, Gibbons, & Bell, 2008). Despite parent report that they have been able to develop relationships with mental health professionals that they have known for some time and that have been reliable and trustworthy (Horowitz, McKay, & Marshall). This was the case with some of the caretakers at the school as they were familiar with this examiner. As such, some still refused participation while others would not even come in to hear about the project.

Primary variable measurement limitations may have also contributed to the lack

of significant findings. Specifically, teachers' report of aggression may not have been objective as they may have been influenced by the child's environment and level of perceived aggression based on other children in the classroom.

Suggestions for Clinical Practice

The process and results of this research underscore critical issues that impact clinical work within this setting. Clinicians must be aware of cultural factors when working within organizations and communities. These factors must be considered in intervention planning in effort to maximize treatment. Dually, clinicians must acknowledge systemic challenges that may interfere in program development and interventions and incorporate appropriate measures to maximize efficacy.

When providing direct clinical services, one must recognize the potential that children in urban communities may be exposed to violence at a higher rate then reported. Screening for PTSD in a community setting where violence is prevalent should be considered as well as developing skill-building programs for children to develop prosocial behavior. Additionally, psychoeducation should become an integral part of programming to increase awareness of the potential impact that exposure can have of children.

Suggestions for Future Research

While the findings of the current study were, as a whole, statically insignificant, they illustrated larger, more critical issues for conducting research of this nature in urban communities. Caregiver involvement was a major barrier to performing research in this

community that warrants investigation into the impact that it has on child behavior and performance. In addition, methods of eliciting involvement need to be examined. This alone would have implications for educational, clinical, and research settings. The underreporting of exposure by some of the children highlights the need for a multimodal method of obtaining this type of information in an effort to more accurately capture the children's experiences. Specifically, qualitative components may capture children's experiences more accurately. Inconsistencies seen in earlier findings may have been influenced by these methodological factors more than previously considered.

Conclusions

Despite the prevalence and severe impact of childhood exposure to violence, this still remains a much under studied area that requires more attention and research.

Although many postulates exist about the magnitude of the lifelong effects of trauma and children, pediatric assessment and research techniques need to continue to evolve.

Cultural factors must be examined and better understood in order to conduct valid research that truly reflects the dynamics and experiences faced by urban children. As researchers gain a better understanding of the neurophysiological, neurocognitive, psychosocial changes that occur in the child that is faced with severe trauma, therapists, along with service providers, will be better equipped to provide effective intervention.

References

- American Psychiatric Association (2000). Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR). Washington, DC, American Psychiatric Association.
- Anisman, H., Griffiths, J., Matheson, K., Ravindran, A. V., & Merali, Z. (2001).

 Posttraumatic stress symptoms and salivary cortisol levels. *American Journal of Psychiatry*, 158, 1509-1511.
- Atkins, M. S., Frazier, S. L., Leathers, S. L., Graczyk, P. A., Talbott, E., Jakobsons, L. Adil, J. A., Marinez-Lora, A., Demirtas, H. Gibbons, R. B., & Bell, C. C. (2008).
 Teacher key opinion leaders and mental health consultation in low-income urban schools. *Journal of Consulting & Clinical Psychology*, 56 (5), 905-908.
- Bandura, A. (1977). Social learning theory. New York: General Learning Press.
- 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 (6), 1173-1182.
- Berger, K. S. (2008). The developing person through the lifespan (7th ed.). NY: Worth.
- Bradshaw, C. P., Rodgers, C. R., Ghandour, L. A., & Garbarino, J. (2009). Social-cognitive mediators of the association between community violence exposure and aggressive behavior. *School Psychology Quarterly*, 24 (3), 199-210.
- Bremner, J. D. (2002). Neuroimaging of childhood trauma. Seminar in Clinical Neuropsychiatry, 7 (2), 104-12.
- Buka, S. L., Stichick, T. L., Birdthistle, I., & Earls, F. J. (2001). Youth exposure to

- violence: Prevalence, risks, and consequences. *American Journal of Orthopsychiatry*, 71 (3), 298-310.
- Carlson, N. R. (2007). Physiology of behavior (9th ed.). Boston: Pearson.
- Carion, V. G., Weems, C. F., & Reiss, A. L. (2007). Stress predicts brain changes in children: A pilot longitudinal study on youth stress, posttraumatic stress disorder, and the hippocampus. *Pediatrics*, 119 (3), 509-516.
- Ceballo, R., Dahl, T. A., Aretakis, M. T., & Ramirez, C. (2001). Inner-city children's exposure to community violence: How much do parents know? *Journal of Marriage and Family*, 63, 927-940.
- City Rating. (2002). Newark crime report. Retrieved on June 27, 2010 http://www.cityrating.com/citycrime.asp?city=Newark&state=NJ.
- Crick, N. R., & Dodge, K. A. (1996). Social information processing mechanisms in reactive and proactive aggression. *Child Development*, 67, 993-1002.
- Critchley, H. D., Mathias, C. J., Josephs, O., O'Doherty, J., Zanini, S., Dewar, B., Cipolotti, L., Shallice, T., & Dolan, R. J. (2003). Human cingulate cortex and

autonomic

- control: Converging neuroimaging and clinical evidence. Brain, 126, 2139-2152.
- Dahlberg, L. L. (1998). Youth violence in the United States: Major trends, risk factors, and prevention approaches. *American Journal of Preventative Medicine*, 14 (4), 259-272.
- DeBellis, M. D., Hooper, S. R., Spratt, E. G., & Woolley, D. P. (2009).

 Neuropsychological findings in childhood neglect and their relationships to pediatric PTSD. Journal of the International Neuropsychological Society, 15,

- 868-878.
- DePrince, A. P., Weinzierl, K. M., & Combs, M. D. (2009). Executive function performance and trauma exposure in a community sample of children. *Child Abuse and Neglect*, 33, 353-361.
- Dodge, K. A., & Coie, J. D. (1987). Social-information-processing factors in reactive and proactive aggression in children's peer group. *Journal of Personality and Social Psychology*, 53 (6), 1146-1158.
- Dodge, K. A., & Pettit, G. S. (2003). A biopsycholosocial model of the development of chronic conduct problems in adolescence. *Developmental Psychology*, 39 (2), 349-371.
- Eckes, A., & Radunovich, H. L. (2007). *Trauma and adolescents*. (Document No. FCS2280). Retrieved on August 21, 2010 from http://ifsrvvedis.ifas.ufl.edu/pdffiles/FY/FY100400.pdf.
- Faul, F., Erdfelder, E., Buchner, A., & Lang, A. G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*, 41, 1149-1160.
- Frazier, P. A., Tix, A. P., & Barron, K. E. (2004). Testing moderator and mediator effects in counseling psychology research. *Journal of Counseling Psychology*, 51 (1), 115-134.
- Gilbert, P. (2002). Understanding the biopsychosocial approach: Conceptualization.

 Clinical Psychology, 14, 13-17.
- Green, R. G. (1981). Behavioral and physiological reactions to observed violence: Effects of prior exposure to aggressive stimuli. *Journal of Personality and Social*

- Psychology, 40 (5), 868-875.
- Greenwald, R. (1997). Child Report of Post Traumatic Symptoms Scale (CROPS).
- Greenwald, R., & Rubin, A. (1999). Assessment of posttraumatic symptoms in children:

 Development and preliminary validation of parent and child scales. *Research on Social Work Practice*, 9 (1), 61-75.
- Groth-Marnat, G. (2009). *Handbook of psychological assessment* (5th ed.). Hoboken, NJ: Wiley.
- Guerra, N. G., Huesmann, R., & Spindler, A. (2003). Community violence exposure, social cognition, and aggression among urban elementary school children. *Child Development*, 74 (5), 1561-1576.
- Hamill, S. K. (n.d.). Resilience & self efficacy: The importance of efficacy beliefs and coping mechanisms in resilient children. *Colgate University Journal of the Sciences*, 115-146.
- Hanson, R., Borntrager, C., Self-Brown, S., Kilpatrick, D., Saunders, B., Resnick, H., & Amstadter, A. (2008). Relations among gender, violence exposure, and mental health: The National Survey of Adolescents. *American Journal of Orthopsychiatry*, 78(3): 313–321.
- Hastings, T. L. (1996). Screen for adolescent violence exposure (SAVE).
- Hastings, T. L., & Kelley, M. L. (1997). Development and validation of the screen for adolescent violence exposure (SAVE). *Journal of Abnormal Child Psychology*, 25 (6), 511-520.
- Hay, D. F. (2005). The beginnings of aggression on infancy. In R. Trembly, W. Hartup, J. & Archer (Eds.), *Developmental origins of aggression* (pp.107-132). NY:

- Guilford Press.
- Henry, J., Sloane, M., & Black-Pond, C. (2007). Neurobiology and neurodevelopmental impact of childhood traumatic stress and prenatal alcohol exposure. *Language*, *Speech*, & *Hearing Services in Schools*, 38, 99-108.
- Hoaken, P. N., Allaby, D. B., & Earle, J. (2007). Executive cognitive functioning and the recognition of facial expressions of emotion in incarcerated violent offenders, non-violent offenders, and controls. *Aggressive Behavior*, 33, 412-421.
- Horowitz, K., McKay, M., & Marshall, R. (2005). Community violence & urban families: Experiences, effects, & directions for intervention. *American Journal of Orthopsychiatry*, 75 (3), 356-368.
- Huesmann, L. R., Guerra, N. G. Miller, L. & Zelli. A. (1989). Normative Beliefs About Aggression Scale. Author.
- Jackowski, A. P., de Araújo, C. M, Tavares de Lacerda, A. L., Mari, J. & Kaufman, J.
 (2009). Neurostructural imaging findings in children with post-traumatic stress
 disorder: Brief review. *Psychiatry and Clinical Neurosciences*, 63 (1), 1-8.
- Jensen, E. (2009). Teaching with poverty in mind: What being poor does to kids' brains and what schools can do about it. Alexandria, Virginia: ASCD.
- Johnson, M. H. (2005). Sensitive periods in functional brain development: Problems and prospects. Centre for Brain and Cognitive Development. Hoboken, NJ: Wiley Periodicals.
- Johnson, M. H., Grossmann, T., & Kadosh, K.C. (2009). Mapping functional brain development: Building a social brain through interactive specialization. *Developmental Psychology*, 45 (1), 151-159.

- Kaplan, D. M., & Coogan, S. L. (2005). The next advancement in counseling: The biopsychosocial model. The Counseling Outfitters: Article 3. Retrieved on August 1, 2010 from www.counselingoutfitters.com.
- Kikas, E., Peets, K., Tropp, K., & Hinn, M. (2009). Associations between verbal reasoning, normative beliefs about aggression, and different forms of aggression.

 Journal of Research on Adolescence, 19 (1), 137-149.
- Kilpatrick, K. L., Litt, M., & Williams L. M. (1997). Post-Traumatic Stress Disorder in child witness to domestic violence. *American Journal of Orthopsychiatry*, 67 (4), 639-644.
- Korkman, M. Kirk, U., & Kemp, S. (2007). NEPSY-II. Administration Manual. San Antonio: PsychCorp.
- Korn, M. L. (n.d.). Recent developments in the science and treatments of PTSD. CME Series, posted for Medscape. Retrieved from http://www.medscape.com.
- Lee, P. N., & Hoaken, P. N. (2007). Cognition, emotion, and neurobiological development: Mediating the relation between maltreatment and aggression. *Child Maltreatment*, 12, 281-298.
- Lipina, S. J., & Colombo, J. A. (2009). Poverty and brain development during childhood:

 An approach from cognitive psychology and neuroscience. Washington, DC:

 American Psychological Association.
- Luu, P., & Posner, M. I. (2003). Anterior cingulate cortex regulation of sympathetic activity. *Brain, 126*, 2119-2120.
- Lynch, M., & Cicchetti, D. (1998). An ecological-transactional analysis of children and contexts: The longitudinal interplay among child maltreatment, community

- violence, and child's symptomatology. *Development and Psychopathology*, 10 (2), 235-257.
- Manly, J. J. (2005). Advantages and disadvantages of separate norms for African Americans. *The Clinical Neuropsychologist*, 19, 270-275.
- Margolin, G., & Gordis, E. B. (2004). Children's exposure to violence in the family and community. *Current Direction in Psychological Science*, 13 (4), 152-155.
- Margolin, G., & Vickerman, K. A. (2007). Posttraumatic stress in children and adolescents exposed to family violence: I. Overview and issues. *Professional Psychology: Research and Practice*, 38 (6), 613-619.
- Markham, J. A., & Greenough, W. T. (2004). Experience-driven brain plasticity: Beyond the synapse. *Neuron Glia Biology*, 1 (4), 351-363.
- McCabe, K. M., Lucchini, S. E., Hough, R. L., Yeh, M., & Hazen, A. (2005). The relation between violence exposure and conduct problems among adolescents: A prospective study. *American Journal of Orthopsychiatry*, 75 (4), 575-584.
- McCollum, D. (2006). Child Maltreatment and Brain Development. Minnesota Medical Association. Retrieved on August 1, 2010 from http://www.minnesotamedicine.com.
- McDonald, C. C., & Richmond, T. R. (2008). The relationship between community violence exposure and mental heath symptoms in urban adolescents. *Journal of Psychiatric and Mental Health Nursing*, 15, 833-849.
- McNally, R. J. (2003). Progress and controversy in the study of Posttraumatic Stress Disorder. *Annual Review of Psychology*, 54, 229-52.
- Mrug, S., Loosier, P., & Windle, M. (2008). Violence exposure across multiple contexts:

- Individual and joint effects on adjustment. American Journal of Orthopsychiatry, 78 (1), 70-84.
- Murali, R., & Chen, E. (2005). Exposure to violence and cardiovascular and neuroendocrine measures in adolescents. *Annals of Behavioral Medicine*, 30 (2), 155-163.
- Nader, K. (2008). Understanding & assessing trauma in children & adolescence:

 Measures, methods, and youth in context. NY: Routledge.
- Neighborhood Scout. (2012). Crime reports. Retrieved on May 1, 2012from www.neighborhoodscout.com.
- Nestor, P. G., Kubicki, M., Nakamura, M., Niznikiewcz, M., McCarley, R. W., & Shenton, M. E. (2010). Comparing prefrontal gray and white matter contributions to intelligence and decision making in schizophrenia and healthy controls.

 *Neuropsychology, 24 (1), 121-129.
- Overstreet, S. (2000). Exposure to community violence: Defining the problem and understanding the consequences. *Journal of Child and Family Studies*, 9 (1) 7-25.
- Overstreet, S., Dempsey, M., Graham, D. C., & Moely, B. (1999). Availability of family support as a moderator of exposure to community violence. *Journal of Clinical Child Psychology*, 28, 151-159.
- Palmer, L., Farrar, A. R., & Ghahary, N. (2002). A bio-psychosocial approach to play therapy with maltreated children: The prescriptive elements in Erik Erikson's theory. In R. R. Massey & S. D. Massey (Eds.) & F. Kaslow (Series Ed.), Comprehensive handbook of psychotherapy, Vol. 3. Interpersonal, humanistic, existential (pp. 109 130). New York: Wiley.

- Palmer, L. K., Frantz, C., Armsworth, M., Swak, P., Copley, J., & Bush, G. (1999).
 Neuropsychological sequelae of chronically psychologically traumatized children:
 Specific findings in memory and higher cognitive functions. In L. Williams & V.
 Banyard (Eds.), Trauma and memory (pp. 229-244). Thousand Oaks, CA: Sage.
- Papalia, D. E., Olds, S. W., & Feldman, R. D. (2007). *Human development* (10th ed.).

 Boston: McGraw-Hill.
- Paschall, M. J., & Fishbein, D. H. (2002). Executive functioning and aggression: A public health perspective. *Aggression and Violent Behavior*, 215-235.
- Perry, B. D. (1999). Post-traumatic stress disorders in children and adolescents. *Current Options in Pediatrics*, 11 (4).
- Perry, B. D. (2001). The neurodevelopmental impact of violence in childhood. In D. Schetky & E. P. Benedek (Eds.), *Textbook of child and adolescent forensic psychiatry*. Washington, DC: American Psychiatric Press, pp. 221-238.
- Perry, B. D. (2005). Violence and childhood: How persisting fear can alter the developing child's brain. Retrieved on August 1, 2010 from http://www.terrylarimore.com/Painandviolence.html.
- Pollak, S. (2004). The impact of child maltreatment on the psychosocial development of young children. In R. E. Tremblay & R. D. Peters, (Eds). *Encyclopedia on Early Childhood Development* [online]. Montreal, Quebec: Centre of Excellence for Early Childhood Development; 1-6. Retrieved on August 1, 2010 from http://www.child-encyclopedia.com.
- Pollak, S. D., & Kistler, D. J. (2002). Early experience is associated with the development of categorical representations for facial expressions of emotions.

- Proceedings of the National Academy of Sciences, 99 (13). Retrieved on August 10, 2010 from www.pnas.org.
- Prince-Embury, S. (2005). Resiliency Scales for Adolescents: A Profile of Personal Strengths. San Antonio, TX: Harcourt Assessment.
- Prince-Embury, S. (2008). Measurement invariance of the Resiliency Scales for Children and Adolescents with respect to sex and age cohorts. *Canadian Journal of School Psychology*, 23, 26-40.
- Prince-Embury, S. (2010). Assessment for integrated screening and prevention using the Resiliency Scales for Children and Adolescents. In B. Doll, W. Pfohl, & J. Yoon (Eds.). (2010). Handbook of Youth Prevention Science. New York: Routledge.
- PsychCorp (1999). Wechsler Abbreviated Scale of Intelligence: Manual. San Antonio: Harcourt Assessment.
- Raine, A. (2002). Annotation: The role of prefrontal deficits, low autonomic arousal, and early health factors in the development of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry*, 43 (4), 417-434.
- Raine, A., Reynolds, C., Venables. P. H., Mednick, S. A, & Farrington, D. P. (1998). Fearlessness, stimulation-seeking, and large body size at age 3 years as early predispositions to childhood aggression at age 11 years. *Archives of General Psychiatry*, 55, 7454-751.
- Resick, P. A. (2001). Stress and trauma. NY: Psychology Press.
- Reynolds, C. R., & Kamphaus, R. W. (2004). Behavior Assessment System for Children, Second Edition. Minneapolis: Pearson.
- Richters, J. E., & Martinez, P. E. (1993). The NIMH community violence project: I.

- Children as victims of and witnesses to violence. Psychiatry, 56, 7-21.
- Roberts, G. E., & Gruber, C. (2005). *Roberts-2*. Los Angeles. Western Psychological Services.
- Rosenthal, B. S. (2000). Exposure to community violence in adolescence: Trauma symptoms. *Adolescence*, *35* (138), 271-284.
- Rothschild, B. (2000). The body remembers: The psychophysiology of trauma and trauma treatment. NY: W. M. Norton & Company.
- Saltzman, W. R., Pynoos, R. S., Layne, C. M., Steinberg, A., & Aisenberg, E. (2001).
 Trauma/grief-focused intervention for adolescents exposed to community
 violence: Results of a school-based screening and group treatment protocol.
 Group Dynamics: Theory, Research, and Practice, 5, 291-303.
- Sattler, J. M. (2001). Assessment for children: Cognitive applications (4th ed.). San Diego: Jerome M. Sattler.
- Scarpa, A., & Ollendick, T. H. (2003). Community violence exposure in a young adult sample: III. Psychophysiology and victimization interact to affect risk for aggression. *Journal of Community Psychology*, 31 (4), 321-338.
- Scarpa, A. S., & Raine, A. (1997). Psychophysiology of anger and violent behavior.

 *Psychiatric Clinics of North America, 20 (2) 375-394.
- Scarpa, A. S., & Raine, A. (2000). Violence associated with anger and impulsivity, In J. Borod (Ed). The Neuropsychology of emotion (pp.320-339). New York: Oxford University Press.
- Scarpa, A., Tanaka, A. and Chiara Haden, S. (2008). Biosocial bases of reactive and proactive aggression: the roles of community violence exposure and heart

- rate. Journal of Community Psychology, 36, 969-988.
- Scott-Jones, D. (1994). Ethical issues in reporting and referring in research with low-income minority children. *Ethics & Behavior*, 4 (2), 97-108.
- Selye, H. (1984). The stress of life: Revised edition. NY: McGraw-Hill.
- Séguin, J. R., Arseneault, L., Boulerice, B., Harden, P. W., & Tremblay, R. E. (2002).
 Response perseveration in adolescent boys with stable and unstable histories of physical aggression: The role of underlying processes. *Journal of Child Psychology and Psychiatry*, 43 (4), 481-494.
- Séguin, J. R., Pihl, R. O., Harden, P. W., Tremblay, R. E., & Boulerice, B. (1995).
 Cognitive and neuropsychological characteristics of physically aggressive boys.
 Journal of Abnormal Psychology, 104 (4), 614-624.
- Séguin, J. R., & Zelazo, P. D. (2005). Executive function in early physical aggression. In R. Trembly, W. Hartup, & J. Archer (Eds.), Developmental origins of aggression (pp. 307-329). NY: Guilford Press
- Shahinfar, A., Kupersmith, J. B., & Matza, L. S. (2001). The relation between exposure to violence and social information processing among incarcerated adolescents.

 **Journal of Abnormal Psychology, 110 (1), 136-141.
- Skybo, T. (2005). Witnessing violence: Biopsychosocial impact on children. *Pediatric Nursing*, 31 (4), 263-270.
- Solomon, E. P., & Heide, K. M. (2005). The biology of trauma: Implications for treatment. *Journal of Interpersonal Violence*, 20 (1), 51-60.
- Spear, L. P. (2000a). The adolescent brain and age-related behavioral manifestations.

 Neuroscience and Biobehavioral Review, 24 (4), 417-463.

- Spear, L. P. (2000b). Neurobehavioral changes in adolescence. Current Directions in Psychological Science, 9 (4), 11-114.
- State of New Jersey (2009). State of New Jersey: Department of Education.

 Retrieved on April 1, 2011 from http://education.state.nj.us.
- Teisl, M., & Cicchetti, D. (2008). Physical abuse, cognitive and emotional process, and aggressive/disruptive behavior problems. *Social Development*, 17 (1), 1-23.
- Thomas, M. S., & Johnson, M. H. (2008). New advances in understanding sensitive periods in brain development. *Current Directions in Psychological Science*, 17 (1), 1-5.
- Tremblay, R. E., & Nagin, D. S. (2005). The developmental origins of physical aggression in humans. In R. Trembly, W. Hartup, & J. Archer (Eds.), Developmental origins of aggression (pp. 83-106). NY: Guilford Press.
- Tremblay, R. E., Hartup, W. W., & Archer, J. (Eds.). (2005). Developmental origins of aggression. NY: Guilford Press.
- Twardosz, S., & Lutzker, J. R. (2010). Child maltreatment and the developing brain: A review of neuroscience perspectives. Aggression and Violent Behavior, 15, 59-68.
- US Census Bureau. (2002). *Economic census*. Retrieved on April 1, 2010 from http://www.census.gov/econ/census02.
- Van Voorhees, E., & Scarpa, A. (2004). The effects of child maltreatment on the hypothalamic-pituitary-adrenal axis. *Trauma*, *Violence*, and Abuse, 5 (4), 333-352.
- Villemarette-Pittman, N. R., Stanford, M. S., & Greve, K. W. (2002). Language and

- executive function in self-reported impulsive aggression. *Personality and Individual Differences*, 34, 1533-1544.
- Watts-English, T. Fortson, B. L., Gibler, N. Hooper, S. R., & De Bellis, M. D. (2006).

 The psychobiology of maltreatment in childhood. *Journal of Social Issues*, 62 (4), 717-736.
- White, K. S., Bruce, S. E., Farrell, A. D., & Kliewer, W. (2004). Impact of community violence on anxiety: A longitudinal study of family social support as a protective factor for urban children. *Journal of Child and Family Studies*, 7 (2), 187-203.
- Wright, R. J. (1998). Exposure to violence. Research network on socioeconomic status and health. Retrieved on April 1, 2010 from www.macses.ucsf.edu/research/psychosocial/violence.php.
- Zillmer, E. A., Spiers, M. V., & Culbertson, W. C. (2008). *Principles of neuropsychology* (2nd ed.). Belmont, CA: Thompson Wadworth.

Appendix A

Caregiver Demographic Form

Background Information

Child Participant ID:	Date:
Please fill in the information below:	
DEMOGRAPHICS Caregiver Age: (in years)	Child's Date of Birth:
Caregiver Gender:MaleFemale	
Native Hav	
	glish oanish her
Do you practice or identify with a reli	gion?YesNo
If Yes, which religion?	
Relationship Status: MarriedSingleSpringer	_In a relationship Living with Partner pouse/ Partner Deceased
Current Employment Status: employed full timeemployed part timenot employedother	full time studentpart time studentretired
Approximate household income: 10- 20K	0K over 100K
Highest level of education completed: Grammar School Col	lege Degree

Middle School Graduate Degree
High School Post-graduate Degree
High School Equivalent
Does your child have any learning disabilities?
If yes, describe:
Does your child have any significant medical and/or psychological problems?
If yes, describe:
Has your child ever fell or hit his head and lost consciousness for even a short
period of time?
If yes, describe:
Has your child ever had a concussion?
If yes, describe:
Has your child ever been retained in a grade?
If yes, which grade and why:

Appendix B

Child Demographic Form

RESEARCH ID						
BIRTH DATE:						
GRADE:	GEN	DER:				
WHAT HAND DO	YOU WRITE WITH	H: LEFT	RIGHT	*		
☐ MOTHER ☐ GRANDMOTHE ☐ AUNT ☐ STEPMOTHER ☐ FOSTER MOTH ☐ ADOPTIVE MOT ☐ FATHER ☐ GRANDFATHER ☐ UNCLE ☐ STEPFATHER ☐ FOSTER FATHE ☐ ADOPTIVE FAT ☐ SISTER: ☐ STEP SISTER: ☐ BROTHER: ☐ STEP BROTHER ☐ COUSINS: ☐ FRIENDS: ☐ OTHER: ☐ PETS:	ER THER R ER THER HOW MANY?	HOW OLD? HOW OLD? HOW OLD? HOW OLD? HOW OLD? HOW OLD? HOW OLD?				
WHAT TIME DO	YOU GO TO BED?					
(CHECK ALL THA ☐ AFTERSCHOOL ☐ SPORTS ☐ CLASSES/INST! ☐ PLAY WITH MY ☐ PLAY OUTSIDE ☐ PLAY WITH MY ☐ WATCH TV ☐ PLAY ON THE G ☐ HOMEWORK	. PROGRAM RUCTION (MUSIC, I Y FRIENDS E Y BROTHERS/SISTE	DANCE, KARA RS/COUSINS	TE, ETC.)			

□ SLEEP	
☐ I GET INTO TROUBLE AFTER SCHOOL	
□NOTHING	
□ OTHER	
WHO IS YOUR ROLE MODEL (SOMEONE YOU WOULD LIKE TO BE WHEN YOU GROW UP?)?	LIKE
WHO UNDERSTANDS YOU THE BEST?	
WHO WOULD YOU TELL YOUR WORRIES TO?	