CHILD MALTREATMENT AND PREADOLESCENT PSYCHOPATHOLOGY: INDIRECT EFFECTS VIA PHYSIOLOGICAL STRESS REACTIVITY

by

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(Under the Direction of Assaf Oshri)

ABSTRACT

Child maltreatment is an adverse childhood experience that increases the risk for developing internalizing and externalizing problems in preadolescence and adolescence. The chronic stress that results from child maltreatment may also alter functioning of physiological stress response systems (i.e., autonomic nervous system and hypothalamic-pituitary-adrenal [HPA] axis). Recent research has linked deficits in physiological stress response function to elevated internalizing and externalizing symptomology in youth. Consequently, the goal of the present study is to examine three measures of physiological stress response function as mediators in the association between childhood maltreatment and preadolescent internalizing and externalizing symptomology. To investigate these aims, I utilized data from the UGA Youth Development Institute (YDI) Parenting and Decision Making Project, which includes physiological and self-report assessments from youth and their primary caregiver ($N = 101$) and follow-up data obtained approximately one year later (Wave 2; $N = 71$). Structural equation models were constructed to test the study's hypotheses. Results showed that child maltreatment severity, and particularly the

severity of emotional abuse, was associated with heightened cortisol reactivity and blunted heart rate variability reactivity. Heightened cortisol reactivity was associated with more depressive symptoms at Wave 2, providing support for mediation between child maltreatment and youth internalizing symptoms via HPA-axis reactivity. The results of the present study provide novel information on the multi-level etiology of preadolescent psychopathology. Child maltreatment may cause changes to stress response systems, leading to elevated risk for affective symptomology. Clinical and prevention programs for maltreated youth may benefit by including intervention components that help youth regulate their acute stress response reactions.

INDEX WORDS: Child maltreatment; Early life stress; Preadolescence; Adolescence; Internalizing symptoms; Externalizing symptoms; stress reactivity

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DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA

2019

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May 2019

ACKNOWLEDGEMENTS

First and foremost, I would like to acknowledge the generous support of the University of Georgia Graduate School and the Dissertation Completion Award.

I am indebted to my primary advisor, Dr. Assaf Oshri, for his continued mentorship. Through working with Dr. Oshri, I have learned the ins and outs of research and academic life and have gained the confidence and skills to become an independent scientist. Most importantly, Dr. Oshri has engrained in me a passion for developmental science. I would also like to extend my immense gratitude to my committee members, Dr. Steven Kogan and Dr. Margaret Caughy. I am thankful for the *many* hours they have spent giving me advice, writing recommendation letters, and for their unending support.

My success can also be attributed to the many faculty and staff in the Department of Human Development and Family Science who have taught and supported me through the years. Additionally, I am extremely grateful to Dr. Laura McKee for nurturing my growth as a researcher. I am also thankful for the friendship, collaboration, and support from my cohort and fellow students in HDFS and in YDI. I have learned so much from working beside you all.

Last but certainly not least, I would not be here without the love and support from my family and friends. I am extremely grateful to my parents, who have always been my biggest fans. My husband, Brian, has been by my side through the highs and lows of doctoral studies, and his love and support is unwavering.

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CHAPTER 1

INTRODUCTION

Childhood maltreatment is a form of significant early life adversity that includes experiences of physical abuse, sexual abuse, neglect, and emotional abuse (Manly, 2005). Youth reared in environments characterized by abuse and neglect are at risk for the development of psychopathology throughout the lifespan (Cicchetti & Banny, 2014; Lindert et al., 2014; Norman et al., 2012). During adolescence, youth who have been exposed to childhood maltreatment have a heightened occurrence of internalizing (i.e., depressive and anxious) and externalizing (i.e., disruptive and aggressive) symptomology (e.g., Bolger & Patterson, 2001; Li & Godinet, 2014; Manly, Oshri, Lynch, Herzog, & Wortel, 2013). Although the research literature has established the link between childhood maltreatment and adolescent psychopathology, fewer studies have investigated the psychophysiological mechanisms underlying this association.

The developmental mechanisms that underlie the emergence of psychopathology in adolescence has been described by the developmental psychopathology perspective (Cicchetti & Rogosch, 2002). According to this perspective, experiences of childhood maltreatment are linked with future psychopathology due to disruptions in normative developmental processes that are necessary for subsequent adaptive development (Cicchetti & Banny, 2014). Simultaneously, evidence from psychophysiological research suggests that chronic stress such as child maltreatment can cause disruptions in the

development of stress response systems, resulting in physiological "wear and tear" and dysregulation in these stress-related systems. This cumulative burden of chronic stress on the body has been named *allostatic load* (McEwen, 2017). Taken together, the developmental psychopathology perspective and allostatic load theory suggest that the chronic stress induced by childhood maltreatment can disrupt the development of stress regulation systems, and in turn these disruptions can lead to cascading maladaptive effects on neurocognitive development and related psychopathology. Indeed, there is empirical support for the mediating role of stress response dysregulation in the association between early life stress and later life psychopathology (Koss & Gunnar, 2018; Stroud, Chen, Doane & Granger, 2018). However, there remains a lack of knowledge on the physiological mechanisms linking childhood maltreatment to internalizing and externalizing symptomology among preadolescents. Preadolescence is an important developmental transition period that typically occurs between the ages of 9 and 12, which is characterized by significant biological and social changes, and in which the onset of psychopathology is likely to occur (Kessler et al., 2005). Further, there is scant literature that examines stress response dysregulation in both the HPA axis and autonomic nervous system (the two primary stress response systems) as intermediaries in the link between childhood maltreatment and youth psychopathology. Thus, the present study investigates biomarkers of stress dysregulation in the HPA axis, the sympathetic nervous system (SNS), and the parasympathetic nervous system (PNS), as three separate mediating mechanisms linking childhood maltreatment with prospective internalizing and externalizing outcomes in preadolescence (See Figure 1.1).

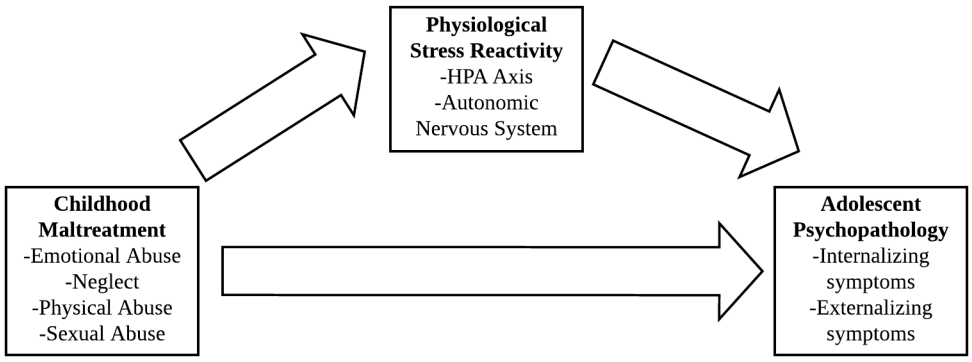

Figure 1.1. Conceptual mechanistic framework guiding study's hypotheses.

Significance and Overview of Study

The incidence of childhood maltreatment has been increasing since the year 2012 in the United States (US Department of Health & Human Services, 2018). A recent report from the U.S. Department of Health and Human Services (2018) indicated that in 2016, there were 3.47 million children who were part of an investigation or who received aid from child protective services (CPS). Out of these CPS-involved children, there were 692,235 substantiated cases of child maltreatment (US Department of Health & Human Services, 2018). However, these statistics are likely biased, as many maltreatment incidents are not reported to CPS agencies. Failure to report maltreatment incidents can stem from errors by mandated reporters, lack of knowledge on the warning signs for child abuse and neglect, or an absence of detectable signs of maltreatment (Finkelhor, 2005). Due to the underreporting of childhood maltreatment, it is likely that most official statistics on maltreatment are underestimates. Indeed, in a study using a nationally representative sample, nearly one in four adults reported that they experienced some form of childhood maltreatment in their lifetime (Finkelhor, Turner, Shattuck & Hamby, 2013). Youth who have been exposed to childhood maltreatment are at an increased risk

for exhibiting both internalizing and externalizing problem behaviors during adolescence (Lindert et al., 2014; Norman et al., 2012; Oshri et al., 2013). Thus, child maltreatment presents a significant public health issue due in part to the various mental health consequences it poses for adolescents.

Research on the developmental mechanisms that link childhood maltreatment and preadolescent psychopathology can inform prevention efforts that target maltreated youth. Specifically, malleable factors that are identified as mediators between child maltreatment and youth psychopathology can be integrated into programs that aim to prevent poor mental health outcomes among at-risk youth. However, several significant gaps remain in the literature on the indirect association between child maltreatment and preadolescent psychopathology. First, recent approaches in developmental psychopathology recommend that investigators consider multiple levels of analysis (e.g., biological and behavioral; Cicchetti, 2016). Indeed, investigators utilizing multiple levels of analysis to study the effects of chronic stress have found that stressors such as child maltreatment can disrupt stress physiology, resulting in dysregulations that are associated with psychopathology (Cicchetti & Handley, 2019; Koss & Gunnar, 2018; McLaughlin, Sheridan, Alves, & Mendes, 2014). A number of studies document the link between chronic stress and HPA axis dysregulation, and on the link between HPA axis dysregulation and psychopathology (Nestler et al., 2002; Tarullo & Gunnar, 2006; Wismer Fries, Shirtcliff & Pollak, 2008; Zorn et al., 2017). Few studies, however, have examined HPA axis dysregulation as a mediator in the prospective link between childhood maltreatment and youth psychopathology. Additionally, researchers have seldom examined the role of physiological stress reactivity in the autonomic nervous

system (ANS) in the link between childhood maltreatment and youth psychopathology. There is also ambiguity in the current literature on the direction of associations between chronic stress and psychopathology with specific measures of stress system dysregulation. For instance, both blunted and excessive cortisol reactivity (a measure of HPA axis dysregulation) have been linked to chronic stress and mental health symptomology (e.g., Bunea, Szentágotai-Tătar, & Miu, 2017; McLaughlin et al., 2014; Zorn et al., 2017). Thus, there is a need for more research to clarify the associations between chronic stress, stress response reactivity, and psychopathology, and to advance knowledge on the role of ANS reactivity in the developmental pathway from child maltreatment to youth psychopathology.

Second, there is a lack of knowledge on the psychophysiological mechanisms linking childhood maltreatment to psychopathology during the preadolescent developmental period. Preadolescent youth experience various psychosocial and psychobiological transitions that may engender vulnerability for the development of psychopathology (Mesman & Koot, 2000; Obradović & Hipwell, 2010). Preadolescence is also a period of heightened neurocognitive plasticity, affording prevention programs the opportunity for delivering effective interventions aimed at reducing mental health and behavioral problems (Fuhrmann, Knoll & Blakemore, 2015). Thus, preadolescence may be a sensitive point in development wherein prevention programs can disrupt the onset (or lessen the severity) of psychopathology for youth who have been exposed to chronic stress.

Last, non-urban low-SES youth remain an understudied population, despite the rising rates of mental health problems and the heightened frequency of adverse childhood

experiences in rural populations (Fontanella et al., 2015; Sedlack et al., 2010). Investigations on the psychobiological mechanisms linking child maltreatment with psychopathology that utilize non-urban and low-SES samples can inform prevention efforts for at-risk youth. Thus, the present study fills a needed gap in the literature on the developmental etiology of psychopathology among non-urban low-SES youth.

In order to address these gaps in the literature and to inform future prevention and intervention programs for maltreated youth, the present study utilizes a longitudinal sample of low-SES preadolescent youth aged 9-12 (N = 101). The specific aims of the study are to (a) investigate the indirect associations between childhood maltreatment and internalizing and externalizing psychopathology via cortisol (HPA-axis) reactivity, and (b) investigate the indirect associations between childhood maltreatment and internalizing and externalizing psychopathology via ANS (SNS and PNS) system reactivity. The goal of the present study is to augment the literature on early life stress and psychopathology in youth by using a sample of non-urban low-SES preadolescents and by investigating three separate stress response systems, thereby informing prevention programs for reducing psychological health disparities in non-urban populations and improving mental health outcomes for at-risk adolescents.

CHAPTER 2

THEORETICAL PERSPECTIVES AND LITERATURE REVIEW

In the present chapter, I will first review the theoretical perspectives that guide this research project, including the developmental psychopathology framework and theories on the biological embedding of stress, such as allostatic load theory. Next, I will review and discuss definitions of chronic and acute stress, childhood maltreatment, and the physiology of human stress response systems. Finally, I will review and synthesize the literature relating to the associations between childhood maltreatment, physiological stress reactivity, and youth internalizing and externalizing psychopathology.

Developmental Psychopathology Perspective

Developmental psychopathology is an overarching theoretical framework describing the development of typical and atypical behavior. This theoretical approach is often contrasted with the "medical model" of psychopathology, in which psychopathology is viewed in terms of discrete clinical criteria and is thought to emerge from mainly endogenous (internal or biological) factors, such as genetics (Sroufe, 1997, 2013). A core assumption of the developmental psychopathology perspective is that each case of psychopathology has a unique etiology resulting from the product of deviations in development that occur over time and across multiple systems, including both within individuals and with their environment (Cicchetti, 2016; Sroufe, 2013). In other words, psychopathology is an outcome of development that arises from complex interactions

between endogenous and exogenous factors (i.e., genetics and environment) *and* the individual's "history of adaptation" up to that point (Sroufe, 1997, p. 252). In contrast, the medical model views psychopathology as an illness, thereby having a discrete and distinct set of symptoms that arise from fixed organic etiological factors (Sroufe, 1997). Hence, researchers who adhere to the developmental psychopathology perspective prioritize the study of symptomology as an outcome rather than clinical diagnoses. Additionally, the focus on the developmental etiology of psychopathology has led developmental psychopathology scholars to examine how early life experiences (e.g., child maltreatment) influence psychopathology over the life course.

Organizational Theory of Development

The organizational theory of child development is often utilized in the developmental psychopathology approach. Organizational theory purports that child development consists of an iterating series of psychological, cognitive, and physiological system reorganizations (Cicchetti & Banny, 2014). These system reorganizations are often the basis of *stage-salient tasks*, which are the developmental goalposts that are necessary for competence throughout a child's growth. Stage-salient developmental tasks include socioemotional tasks such as developing a secure attachment relationship with a caregiver, cognitive tasks such as developing working memory functions, and physical tasks such as sitting up and walking. According to organizational theory, each of these stage-salient developmental tasks requires the successful reorganization of multiple subsystems in the overall developing system, and maladaptation arises when there are disruptions in this system reorganization (Cicchetti & Banny, 2014). In the case of child maltreatment, the presence of an abusive or neglectful caregiver may disrupt the child's

attainment of stage salient tasks, such as secure attachment, and consequently jeopardize future socioemotional competence. Empirical studies have provided support for the organizational theory of development as it pertains to the consequences of child maltreatment (e.g., Flynn, Cicchetti & Rogosch, 2014). For example, in a study using prospective data, adolescents who had been exposed to childhood maltreatment were more likely than their non-maltreated peers to have low self-worth and low-quality relationships with their parent, which in turn predicted increased internalizing psychopathology (Flynn, Cicchetti & Rogosch, 2014).

Multilevel Systems Perspective

Systems perspectives have informed many aspects of the developmental psychopathology framework (Cicchetti, 2006). This includes the conceptualization of the developing individual as an open, as opposed to a closed, system (Cicchetti & Rogosch, 1996). As an open system, the development of both adaptive and maladaptive behavior is influenced by internal subsystems (e.g., stress physiology) as well as external systems (e.g., the family system). In the developmental systems viewpoint, the concept of *emergence* refers to the process by which interactions among subsystems result in the materialization of qualitatively different functions of the system, such as behaviors (Gottlieb & Halpern, 2002). For example, factors in the family system (e.g., child maltreatment) and socioemotional systems (e.g., self-regulation abilities) may interact and result in new behaviors (e.g., externalizing behaviors). Additionally, *transactions* can occur between different subsystems within the individual. Transactions are different from simple interactions, in that both components of the system are changed because of the interplay between them (Sameroff, 2009).

Internal subsystems include a developing child's physiology and psychology, whereas external systems include a child's family, school, community, and culture. Components of different systems at the same level, or of different systems at different levels, may transact to influence the child's probabilistic (as opposed to deterministic) developmental pathway (Gottlieb & Lickliter, 2007). This is illustrated by research showing that characteristics of the family system (e.g., child maltreatment) and characteristics of the youth (e.g., self-regulation or genetic polymorphisms) interact to predict the emergence of internalizing and externalizing disorders (Bolger & Patterson, 2001; Cicchetti & Rogosch, 2014; McLaughlin, Rith-Najarian, Dirks & Sheridan, 2015). Thus, according to the developmental psychopathology view, the influence of multiple systems should be considered when studying the development of youth adaptation and maladaptation (Cicchetti, 2006). The study of the mechanisms linking child maltreatment with preadolescent internalizing and externalizing psychopathology will benefit by incorporating measures from multiple levels of analysis (e.g., physiological and behavioral).

Multifinality. Stressors such as childhood maltreatment may place the developing child on a probabilistic pathway towards maladaptation. Indeed, there is evidence to suggest that experiences of childhood maltreatment are associated with various clinically distinct outcomes, including both internalizing and externalizing psychopathology (Bolger & Patterson, 2001; Cicchetti & Handley, 2019). However, many youth who experience maltreatment do not develop psychopathology. According to the concept of *multifinality*, multiple developmental pathways can result from a common source of disturbance to the developing system, such as child maltreatment (Cicchetti & Rogosch,

1996). For example, given two children who both experience high levels of physical abuse, it is possible that one child will exhibit academic problems and disruptive behavior during adolescence, while the other child will develop in a more adaptive fashion and avoid the aforementioned difficulties. Multifinality is also evident in the development of psychopathology following chronic stress such as maltreatment (Oshri et al., 2013). Some youth who experience maltreatment will develop internalizing disorders, while others may be more likely to exhibit externalizing symptoms (See Figure 2.1).

Risk and protective factors influence the emergence of multifinality in developmental outcomes following a system disturbance (e.g., early life stress). Risk factors are intrapersonal or contextual factors that are probabilistically associated with poor developmental outcomes (Sameroff, 2006). Youth who are maltreated may be exposed to additional risk factors such as poverty, which is often considered a risk factor for poor academic and cognitive outcomes (Hildyard & Wolfe, 2002; McLoyd et al., 2009). On the other hand, protective factors are intrapersonal or external influences that can enhance a child's developmental trajectory towards adaptive outcomes. For example, good self-regulation and the presence of supportive adults are both protective factors that are related to socioemotional competence in the presence of risk (Masten, 2014). Risk and protective factors are often dynamic during child development, that is, their presence or absence can fluctuate. A recent study found that a constellation of risk and protective factors influenced the development of future orientation during adolescence, and the effect of these risk and protective factors varied over time (Oshri, Duprey, Kogan, Carlson & Liu, 2018). Thus, the constellation of risk and protective factors in a child's life can change and influence the result of a common system disturbance, resulting in

different developmental trajectories.

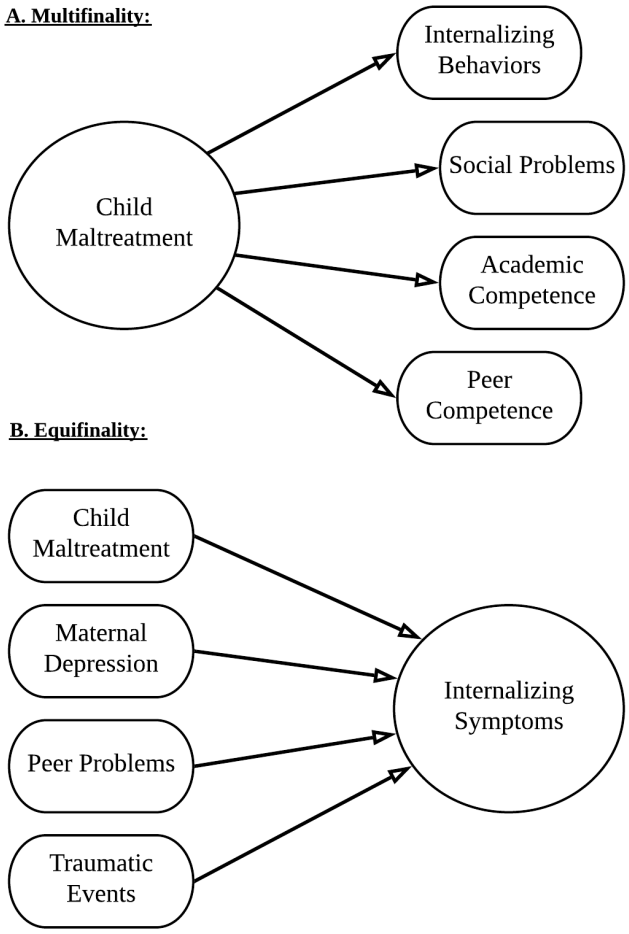

Figure 2.1. An example of multifinality and equifinality.

Equifinality. The concept of equifinality contrasts with the concept of multifinality, as it refers to the possibility that multiple distinct developmental pathways can result in a similar outcome (Cicchetti & Rogosch, 1996). In the case of adolescent internalizing symptoms, for example, there are numerous possible etiologies (See Figure 2.1). Developmental processes that stem from chronic child abuse and neglect, as well as

proximal experiences during adolescence such as isolation and peer problems, can each trigger the onset of youth internalizing symptoms and disorders (Li & Godinet, 2014; Turner, Finkelhor, Shattuck & Hamby, 2012). Similarly, experiences of physical abuse as well as experiences of emotional abuse (two qualitatively different forms of maltreatment) have been found to predict internalizing (Norman et al., 2012). In accordance with the developmental psychopathology perspective, the specific developmental pathway toward internalizing psychopathology will be different for each individual. Thus, there are a variety of developmental processes that result in the emergence of internalizing behaviors and other psychopathology outcomes.

Chronic and Acute Stress: The Case of Child Maltreatment

Acute Stress

Stress is defined as the physiological and psychological reaction to a stressor (i.e., a stimulus that threatens ones' well-being; Gunnar & Quevedo, 2007). Stressors can present either physical or psychosocial threat. The perception of danger to one's physical wellbeing, such as seeing a predator nearby, prompts an acute stress reaction for most species. However, for many mammals including humans, psychosocial stress can spark the same stress reaction as a stressor that poses a threat to one's physical wellbeing (Lovallo, 2015). Thus, both psychosocial and physical threats can produce the same acute physiological stress reaction in humans (Lovallo, 2015). Stressors also differ in their aversiveness. Exercise is an example of a positive stressor that is typically non-aversive (Lovallo, 2015). Further, *anticipatory stress* is the physiological stress reaction that occurs after thinking about an imagined or perceived stressor (Lovallo, 2015). Thinking about or ruminating about a potential stressor (e.g., a child who fears the possibility of a

parent hitting them) can provoke the same physiological reaction as an actual stressor (e.g., a parent inflicting physical harm on a child). In the present dissertation, stress will refer to psychosocial stress that is aversive in nature (e.g., unpredictable, uncontrollable, and/or accompanied by negative emotions) and which includes responses to actual and anticipatory threats.

In order to study the physiological impacts of acute psychosocial stress, researchers often administer a mildly adverse stressor in a laboratory setting. A common acute stressor in laboratory settings is the mental arithmetic task, in which participants are asked to complete a series of mental math problems, often in front of a group of researchers (Kirschbaum, Pirke & Hellhammer, 1993; Lovallo, 2015). The mental arithmetic task is derived from the Trier Social Stress Test (TSST), a widely validated stress-induction test that consists of a mental arithmetic component and a public speaking component (Kirschbaum et al., 1993). The mental arithmetic task is an example of a psychosocial stressor: Although there is no danger of *physical* harm to the individual completing the task, the task is aversive due to the effortful cognitive processes associated with mental math and the social-evaluative threat that is induced by the task (Lovallo, 2015). This task reliably produces an autonomic and endocrine stress response, which I will refer to as *stress reactivity* in the following discussion (Berntson, Cacioppo & Fieldstone, 1996).

Child Maltreatment as Chronic Stress

Childhood maltreatment is a psychosocial and/or physical stressor that confers significant threats to the developing child's wellbeing. Isolated experiences of child abuse and neglect elicit acute stress response reactions. However, the rearing environment in

which child abuse and neglect occurs is apt to elicit chronic psychosocial stress due to both anticipatory and real danger to the child. Chronic stress is defined as "multiple, frequent exposure to stressors and/or long term constant exposure to stressors" (Cyr & Romero, 2009).

Child maltreatment as toxic stress. In accordance with the National Scientific Council in the Developing Child's taxonomy of stressors, childhood maltreatment may also be defined as a *toxic* stressor (Shonkoff, 2010). In this taxonomy, childhood stress is categorized into three general categories: *positive*, *tolerable*, and *toxic* (Shonkoff, 2010). Positive stressors include normative life challenges that are often beneficial for the child and promote learning and attainment of stage-salient developmental tasks. To illustrate, a child walking into their classroom on the first day of kindergarten may exhibit a sympathetic reaction characterized by sweaty palms and an elevated heartbeat. This normative response enables the child to complete an important stage-salient task (developing school-related skills and attaining independence from the primary caregiver). Tolerable stress in this taxonomy includes more significant and negative stressors, such as witnessing violence or the death of a loved one, which can be potentially harmful to a child. Stressors that are defined as tolerable occur in the context of a supportive and nurturing adult figure, which buffers the possible negative impact of the stressor. Toxic stress is similar to tolerable stress in that it involves the occurrence of negative and significant stressors. However, toxic stress occurs when there is not supportive adult figures to help buffer the child's stress response. Consequently, toxic stress involves the chronic activation of a child's stress response systems. This prolonged stress system activation in the absence of a stress-buffering caregiver is harmful to the developing

child, and can result in various negative developmental outcomes (Shonkoff et al., 2012). Child maltreatment is a negative and significant stressor that often occurs in the absence of a supportive rearing environment, and thus is an example of a toxic stressor.

Child Maltreatment Dimensions

Experiences of maltreatment are heterogeneous, and accordingly, maltreatment is a multidimensional concept that is often categorized into types of maltreatment, timing of maltreatment, and severity of maltreatment. These characteristics of childhood maltreatment are often measured via self-report (by the child or parent) or by using case files from Child Protective Services. Below I will discuss the nomenclature and classification of child maltreatment in the literature.

Child maltreatment types. Maltreatment is often classified into four *types*, including physical abuse, sexual abuse, neglect, and emotional/psychological abuse (English, Bangdiwala, & Runyan, 2005). *Physical abuse* is defined as the purposeful affliction of physical harm upon a child and includes actions such as hitting, kicking, and burning. *Sexual abuse* is defined as the actual or attempted sexual contact between the child and a family member or adult caregiver. *Neglect* is defined as a caregiver's failure to provide for their child's basic needs and/or a failure to adequately supervise the child. Finally, *emotional* or *psychological abuse* is defined as a failure to provide a child with basic emotional needs, and may include acts of degrading the child, isolating the child, and/or threatening the child (Paul & Eckenrode, 2015; Toth & Cicchetti, 2013). According to the Centers for Disease Control and Prevention, child maltreatment types can be categorized into acts of *commission* (i.e., inappropriate and harmful actions towards the child) and acts of *omission* (i.e., failure to give appropriate caregiving to a

child; Leeb, Paulozzi, Melanson, Simon & Arias, 2008). Physical and sexual abuse are both acts of commission, while neglect is an act of omission. Emotional abuse can either be an act of commission (e.g., name-calling) or omission (e.g., failing to provide emotional support). Maltreatment types can also be further categorized into subtypes depending on the categorization system that one is using. For example, according to the Maltreatment Classification System (MCS), neglect can be separated into several subtypes such as educational neglect and physical neglect (English et al., 2005).

Child maltreatment severity. Childhood maltreatment differs not only by the type of abuse or neglect but also by its severity. For instance, physical abuse ranges from mild abuse such as spanking to severe abuse that can cause child fatalities. Research indicates that child outcomes differ depending on the severity of maltreatment they experience (Manly, Kim, Rogosch & Cicchetti, 2001; Yoon, 2017). In a longitudinal study of developmental trajectories of internalizing symptoms following maltreatment, Yoon (2017) found that more severe maltreatment uniquely predicted class membership in an elevated internalizing symptom trajectory. Other authors have similarly found that maltreatment severity predicts a range of youth outcomes, including internalizing and externalizing psychopathology (Oshri, Rogosch, Burnette & Cicchetti, 2011; Yoon, 2017).

One common way to measure child maltreatment severity is with the MCS, which ranks maltreatment severity from one to six (English & LONGSCAN Investigators, 1997). According to the MCS measure, a less severe form of neglect (specifically, failure-to-provide) includes a child who occasionally misses meals or has to fix his/her own meals due to parental negligence, whereas the most severe form of

neglect (failure-to-provide) includes a child who is severely malnourished due to the parent's negligence. Likewise, according to the MCS, a less severe form of emotional abuse includes a child whose parent makes derogatory comments towards them, while a more severe form of emotional abuse includes a child who is placed in confinement for an extended period. Researchers have also operationalized severity of child maltreatment by utilizing a continuous score on self-report measures such as the Conflict Tactics Scale or the Childhood Trauma Questionnaire (Bernstein, Ahluvalia, Pogge & Handelsman, 1997; Straus, Hamby, Finkelhor, Moore & Runyan, 1998).

Child maltreatment timing. Developmental theory and research provide ample evidence on the significance of developmental timing in which maltreatment occurs (Dunn, McLaughlin, Slopen, Rosand & Smoller, 2013; Manly et al., 2001). Maltreatment timing can refer to (a) the age of a child when maltreatment occurs, or (b) chronicity, the length of time that the maltreatment experiences occur during a child's life (Manly et al., 2001). Research has shown that maltreatment occurring in different developmental periods may incur distinct developmental consequences. Maltreatment that occurs in early childhood may be particularly pernicious, as this is a period of rapid socioemotional, cognitive, and physiological growth (Dunn et al., 2013; Gomez et al., 2017). Timing of maltreatment may also interact with other aspects of maltreatment, such as type. It is possible that one type of maltreatment is particularly harmful during early childhood, while another type is particularly harmful during preadolescence. Indeed, Manly et al. (2001) found that physical abuse occurring during the preschool years and neglect that occurred during infancy both significantly predicted youth externalizing symptoms, after controlling for other maltreatment types and timing.

Physiology of Stress Response Systems

Stressors such as childhood maltreatment require a child's psychological *and* physiological systems to respond in order to adequately cope with the threat. There are two primary biological systems that are responsible for responding to threats and stressors in the environment: the autonomic nervous system (ANS) and the hypothalamic-pituitary-adrenal (HPA) axis (Gunnar & Quevedo, 2007). The ANS is responsible for the fight-or-flight reaction, while the HPA axis, broadly, supports and regulates the fight-or-flight reaction. During a stress reaction, the ANS and the HPA axis are both activated. The following section will briefly introduce the underlying physiology of each stress response system along with common measurement approaches for assessing stress responses in each system.

Autonomic nervous system. The autonomic nervous system consists of the sympathetic and the parasympathetic nervous systems. The former responds to external demands with immediate "fight or flight" reactions, and the latter works to down-regulate the individual and maintain homeostasis (Gunnar & Quevedo, 2007).

Sympathetic nervous system. The sympathetic nervous system supports various life-supporting homeostatic functions, including the stress response (Lovallo, 2016). Specifically, the sympathetic adreno-medullary (SAM) system is a component of the sympathetic nervous system which is primarily responsible for the fight-or-flight stress response (Gunnar et al., 2015). After an individual perceives a stressor, the adrenal medulla (a part of the adrenal gland) signals the release of epinephrine (i.e., adrenaline) and nerve terminals signal the release of norepinephrine. The widespread effects of these catecholamines form the physiological basis of the fight or flight response. These

catecholamines play various roles in the sympathetic stress response, such as increasing heart rate, increasing serum glucose levels in order to provide energy to the organism, and prompting vasodilation in blood vessels supplying to muscles (Gunnar & Quevedo, 2007). The physiological actions of epinephrine and norepinephrine work to increase arousal and vigilance, helping the body sustain the fight-or flight response.

Measurement approaches. There are several common psychophysiological measures that researchers utilize to examine sympathetic nervous system function in the context of stress. Pre-ejection period, skin conductance level, and alpha amylase are three measures that are often assessed before, during, and after a stressor is administered in order to measure stress reactivity in the sympathetic nervous system (Beauchaine, 2012). The pre-ejection period is an index of the amount of the stress hormone epinephrine that is produced during a sympathetic nervous system reaction, and is measured by the amount of time it takes between a heartbeat and the ejection of blood into the aortic valve (Beauchaine, 2012). The measurement of pre-ejection period is obtained via an electrocardiogram. Another measure of the sympathetic response to stress is skin conductance level, also known as the galvanic skin response. Skin conductance is an electrodermal measure, meaning that it indexes the skin's electrical properties (Dawson, Schell & Courtney, 2011). During fight-or-flight reactions, norepinephrine causes the sweat glands to become more active, and consequently the electrical conductivity of the skin increases. Thus, skin conductance level indirectly indexes the amount of norepinephrine that is released during the stress response. Lastly, alpha amylase can be utilized to measure the sympathetic nervous system reaction, as this is an enzyme regulated by norepinephrine (Beauchaine, 2012; Gunnar & Quevedo, 2007). Shortly

following threats, norepinephrine is released into bodily tissues and glands such as the salivary glands. However, researchers have been unable to directly measure salivary norepinephrine in relation to stress response. Thus, alpha-amylase levels measured via saliva provide an indirect measurement of norepinephrine response and, consequently, sympathetic response (Granger, Kivlighan, El-Sheikh, Gordis & Stroud, 2007).

Both hypo- and hyper-reactive sympathetic responses to stress may mark dysregulation. A hypo-reactive (blunted) sympathetic response occurs when there is an inadequate increase of norepinephrine and epinephrine following a stressor, while a hyper-reactive sympathetic response occurs when there is an excessive fight-or-flight reaction. Blunted alpha-amylase reactivity to an acute stressor was associated with suicide risk in a sample of adults, and reduced pre-ejection period reactivity was associated with experiences of child maltreatment in a sample of adolescents (McGirr et al., 2010; McLaughlin et al., 2014). On the other hand, women with experiences of sexual abuse displayed a heightened sympathetic reaction to an acute stressor compared to non-abused women (Heim et al., 2000).

Parasympathetic nervous system. The parasympathetic system works to return the body to baseline after the fight-or-flight reaction occurs. The parasympathetic nervous system also plays a key role in the acute stress response, as it is responsible for withdrawing its regulatory effect in order to enable the sympathetic fight or flight response. Together, the sympathetic and parasympathetic nervous systems have opposing but complementary roles in the physiological acute stress response (Thayer & Lane, 2000).

Measurement approaches. To measure parasympathetic nervous system activity, a common index is high-frequency heart rate variability (HF HRV; Appelhans & Luecken, 2006). Heart rate variability is a measure of the difference in inter-beat intervals between aspirations (i.e., breathing in) and expirations (i.e., breathing out). It should be noted that HF HRV and *respiratory sinus arrhythmia* are often used synonymously in the research literature (Laborde, Mosley & Thayer, 2017). The high frequency components of heart rate variability can be isolated via power spectrum analysis in order to operationalize parasympathetic nervous system activity (Appelhans & Luecken, 2006). HF HRV is thought to specifically measure vagal tone, which refers to the activity of the *vagus nerve*. The vagus nerve is the tenth cranial nerve originating from the nucleus accumbens, and serves as one of the primary nerves in the parasympathetic system. During times when there is not an immediate threat present, the vagus nerve applies a consistent "braking" effect on cardiac function via its efferent neural pathway to the heart. This has the effect of maintaining homeostasis and inhibiting fight or flight reactions (Laborde et al., 2017). When there is a stressor present, the parasympathetic nervous system quickly withdraws the suppressing influence of the vagal nerve, thus increasing heart rate and allowing the fight or flight reaction to take over. This immediate vagal withdrawal helps the organism to quickly and flexibly respond to environmental threats (Appelhans & Luecken, 2006). A lack of vagal withdrawal during a stressful situation (i.e., blunted HRV reactivity) is often viewed as a form of stress system dysregulation, and has been linked with early life stress and depression (Hamilton & Alloy, 2016; McLaughlin et al., 2015). Thus, vagal tone is an indirect index of the parasympathetic nervous system's ability to respond quickly and flexibly to environmental demands. There are several current theories that

conceptualize vagal tone as a measure of both socioemotional regulation (e.g., Porges, 2001) and cognitive-behavioral regulation (e.g., Thayer, Hansen, Saus-Rose, & Johnsen, 2009).

The polyvagal theory. According to Porges' polyvagal theory, the vagus nerve evolved in humans and other mammals to enable specialized responding to external social demands (Porges, 2001). The vagus nerve is a core physiological component of social behavior. For example, it is linked to other myelinated cranial nerves that control facial expressions and vocalization, key aspects of emotional and social responding (Porges, 2001). Accordingly, the polyvagal theory suggests that HF HRV, or respiratory sinus arrhythmia, provides a measure of emotion regulation during social situations.

The neurovisceral integration theory. The neurovisceral integration perspective offers another theoretical approach to the study of HRV. According to the neurovisceral integration theory, HRV is implicated not only in emotion regulation but also cognitive self-regulation tasks related to executive function, such as inhibitory control (Thayer et al., 2009). Central to this theory is the role of the prefrontal cortex, which provides inhibitory control related to the parasympathetic system via several subcortical circuits (Thayer et al., 2009). Accordingly, the neurovisceral integration model states there is a "flexible neural network associated with self- regulation and adaptability" (Thayer et al., 2009, p. 145). Due to this role of the vagal nerve, HF HRV can be utilized to index self-regulatory function. Thus, supported by both neurovisceral integration theory and polyvagal theory, vagal tone, as measured by HF HRV, is a marker of an individual's ability to regulate physiologically under conditions of stress.

Hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis is responsible for the hormonal, or endocrine, stress response. The actions of the HPA axis are slower and take place over a longer period of time compared to the sympathetic nervous system response (Gunnar & Quevedo, 2007). Cortisol is the primary glucocorticoid that the HPA system produces and circulates in response to stressors (Tarullo & Gunnar, 2006). Upon experiencing a stressor, the hypothalamus is activated and signals for the secretion of corticotropin releasing factor (CRH). CRH, in turn, stimulates the anterior pituitary gland to secrete adrenocorticotropic hormone (ACTH), which enters general circulation and subsequently prompts the secretion of cortisol from the adrenal cortex (Gunnar & Quevedo, 2007). Cortisol then acts upon both mineralocorticoid and glucocorticoid receptor sites, which are widespread throughout the body.

The HPA axis regulates the stress response via the actions of cortisol (Lovallo, 2015). Glucocorticoids have a variety of regulatory actions in the body during the stress response, including the suppression of the immune and inflammatory response (Irwin & Cole, 2011), regulation of an individual's energy via glucose metabolism, and the inhibition of short-term fight or flight responses (Gunnar & Quevedo, 2007). Glucocorticoid receptor sites specifically mediate the stress-related outputs of cortisol such as increased energy production. The distribution of cortisol typically peaks approximately twenty minutes after a stressor occurs, and its effects may last for days (Gunnar et al., 2015). Unlike stress-related neurotransmitters such as epinephrine and norepinephrine, cortisol can cross the blood-brain barrier to exert influences on the brain (Gunnar, Doom, & Esposito, 2015). One of the important actions of cortisol in the brain is the down-regulation of glucocorticoids (i.e., the negative inhibitory feedback cycle).

Notably, chronic stress has been shown to modify glucocorticoid receptors, thus affecting the negative feedback regulation of the HPA axis and resulting in either hyper- or hypo-secretion of cortisol. For instance, individuals with reduced negative feedback regulation of the HPA axis are likely to exhibit hypercortisolism in response to stress (Gunnar & Quevedo, 2007).

In terms of development, the HPA axis is not in place at birth but instead develops throughout childhood and is influenced by environmental experiences, including the early rearing environment (Tarullo & Gunnar, 2006). Cortisol is an essential and healthy part of the body's physiological response to stress, and it also plays a role in non-stress-related functions such as responding to metabolic needs (Lovallo, 2015). However, chronic stress can cause dysregulated cortisol patterns that have adverse effects on physical and mental health. Dysregulation of the HPA axis is often exhibited via a flattened diurnal (i.e., daily) pattern of cortisol, a blunted cortisol response to acute stressors, or a hyper-active cortisol response to acute stressors. Cortisol release also differs depending on mood states. Stressors that are emotionally aversive (e.g., a mental math task) have been shown to prompt more cortisol release as compared to stressors that are positive in nature such as exercise (Lovallo, 2015).

Measurement approaches. There are two standard approaches to measuring cortisol in psychophysiological research: (1) the diurnal cortisol approach, and (2) the cortisol reactivity approach. Both approaches analyze cortisol most often using salivary samples, although urine samples can also be used to obtain cortisol. In most humans, cortisol follows a daily (diurnal) pattern that peaks shortly after waking in the morning and slowly decreases throughout the day. The cortisol awakening response (CAR) refers

to the sharp peak of cortisol that occurs 20-30 minutes after awakening. Individuals can be said to have abnormal diurnal cortisol profiles if their cortisol levels are elevated at nighttime or if they have suppressed levels in the morning. Additionally, a blunted CAR is a signal of HPA dysregulation and has been linked with childhood adversity among preadolescents (Quevedo, Johnson, Loman, LaFavor & Gunnar, 2012). Thus, researchers studying the HPA axis can take several salivary cortisol measurements throughout the day to assess whether an individual's diurnal cortisol rhythm is flattened, indicating dysregulation (e.g., Bernard, Butzin-Dozier, Rittenhouse & Dozier, 2010).

In order to examine the HPA response to an acute stressor, researchers can also measure cortisol reactivity. In this approach, stress is induced via an experimental lab task and salivary cortisol is measured before the stressor, 20 minutes post-stressor, and 40 minutes post-stressor (Gunnar et al., 2015). Chronic experiences of stress can lead to a blunted *or* hyper-active pattern of cortisol reactivity, which are both signals of HPA axis dysregulation. Youth with a history of chronic deprivation are more likely to exhibit a lower cortisol reaction to stressors (McLaughlin et al., 2015), while other studies have found associations between child maltreatment and cortisol hyper-reactivity to stress (Heim et al., 2000). Recently, researchers have begun to study cortisol by measuring levels in hair. This approach avoids the complications that are posed by the diurnal changing rhythms of cortisol, and is able to measure long term cortisol production (Gunnar et al., 2015). Hair cortisol levels are a measure of the amount of exposure to stress and cannot be used to measure regulation of the HPA axis (Sauvé, Koren, Walsh, Tokmakejian & Van Uum, 2007). Specifically, a short hair sample (2-3 cm.) can show

the amount of exposure to cortisol in the two to three months prior to collection (Sauvé et al., 2007). Thus, hair cortisol is a measure of exposure to chronic stress.

Theories on the Biological Embedding of Stress

In the child development literature, there is a breadth of findings regarding the association between early life stress and poor health outcomes (Miller, Chen & Parker, 2011). As a result, recent attention has been given to the biological mechanisms that underlie the association between chronic early life stress, such as childhood maltreatment, and future maladaptive health outcomes. Consequently, multiple theories have emerged on the biological embedding of chronic childhood stress (Miller, Chen, & Parker, 2011; Repetti, Taylor, & Seeman, 2002; Shonkoff, 2010). The biological embedding of stress refers to the processes whereby chronic stress influences functioning of various neurological, endocrine, and immune systems, subsequently leading to poor health and mental health outcomes (Berens, Jensen & Nelson, 2017). Theories on the biological embedding of stress are pertinent not only to health outcomes, but also to investigations on the multi-level etiology of psychopathology and the psychophysiological mechanisms linking chronic stress to mental health outcomes. Thus, the present study is influenced by current theoretical perspectives and recent literature on the biological embedding of stress, which will be reviewed below.

Allostatic Load Theory

Allostasis refers to the physiological processes that are associated with the body's maintenance of stability despite both internal and external stressors (Juster, McEwen, & Lupien, 2010). This concept is related to homeostasis but differs in that allostasis is a constantly changing and adaptive process. According to the allostatic load model, the

experience of chronic stressors requires constant activation of the body's allostatic stress response systems, resulting in a "wear and tear" on the body (Juster et al., 2010; McEwen, 2017). The wear and tear that follows chronic stress is referred to as *allostatic load.* The chronic activation of stress-response systems and the resulting allostatic load may result in future dysregulated responses to acute stress, which can be exhibited as either hyper- or hypoactive response profiles (Juster et al., 2010). The allostatic model purports that chronic stress "gets beneath the skin" as a result of the constant activation of allostatic processes in the two primary physiological stress response systems (i.e., sympathetic-adrenal-medullary [SAM] axis and the HPA axis) and the resulting dysregulation, or allostatic load (McEwen, 2017). In other words, individuals who experience chronic stress may eventually exhibit dysregulated acute stress responses, such as blunted cortisol reactivity. Allostatic overload is traditionally operationalized by creating a sum score of biomarkers that represent both primary mediators of the stress reaction (e.g., cortisol reactivity) and secondary outcomes of allostatic overload (e.g., blood pressure; metabolic markers such as cholesterol; immune markers such as interleukin-6).

Much of the research on allostatic load in human populations has examined the impact of adverse childhood experiences or poverty on allostatic load indices (for examples, see Blair, Raver, Granger, Mills-Koonce, & Hibel, 2011). However, there is also emerging research on the specific role of child maltreatment in predicting allostatic overload (Danese & McEwen, 2012). In a large prospective study using a sample of individuals who had been exposed to child abuse and neglect and demographically matched non-maltreated controls, Horan & Widom (2015) found evidence for a causal

role of childhood maltreatment in predicting an adult allostatic load composite. Specifically, in a path model, child abuse and neglect (measured using official child protective services reports) was associated with increased allostatic load at age 41. There is also evidence for the association between childhood maltreatment and specific allostatic load indices in youth, including cortisol regulation. These studies will be expanded upon below in the section on childhood maltreatment and stress response system outcomes. Although the present study does not include a sum score of allostatic overload, the allostatic load theory is used to theorize why child maltreatment may result in dysregulated reactions to acute stress.

Allostatic load and psychopathology. The allostatic load model also has utility in theorizing the association between early life stress and mood disorders, as mediated by biological factors. According to McEwen (2003), the chronic activation of the autonomic, immune, and endocrine systems in response to stress may result in damage to one's brain. Experimental evidence from animal models shows that chronic stress predicts suppressed neurogenesis, hippocampal remodeling, and atrophying of the amygdala (McEwen, 2003). Specifically, dysregulation of the HPA axis may impact the primary brain areas in the limbic system such as the hippocampus and amygdala, which play an important role in emotion regulation (McEwen, 2003). In turn, structural and functional deviations in the hippocampus and amygdala have been associated with depression and other mood disorders (McEwen, 2003). Thus, in accordance with the allostatic load theory, chronic stress such as child maltreatment may lead to mood and behavioral disorders as a result of stress system dysregulation that influences the structure and function in key brain areas.

Empirical evidence supports the allostatic load theory in relation to childhood maltreatment experiences and psychopathology outcomes. For example, Rogosch, Dackis, and Cicchetti (2011) investigated allostatic load indices of maltreated and non-maltreated 8-10 year old youth who attended a summer day camp. All youth were from families that were considered low-income. An allostatic load composite was created that included six individual risk factors related to allostatic load: blood pressure, body mass index, waist-hip ratio, and three measures of diurnal cortisol regulation. Their results showed that allostatic load and maltreatment status, together, predicted higher levels of youth psychopathology. In another study that investigated childhood abuse and allostatic load in a clinical sample of adults with depressive disorders, researchers found that allostatic load mediated the association between childhood physical abuse and severity of depressive symptoms in adulthood (Scheuer et al., 2018). Thus, there is evidence that supports the role of allostatic load in the link between childhood maltreatment and psychopathology outcomes in both childhood and adulthood.

Ecobiodevelopmental (EBD) Framework

Shonkoff's EBD framework extends allostatic load theory by offering an integrative model of the effects of a child's environment on future health outcomes (Shonkoff, 2010). This framework incorporates allostatic load theory by asserting that chronic stress partly gets "beneath the skin" via dysregulated allostatic responses to stressors. The EBD framework extends upon allostatic load theory by proposing a hierarchical categorization of three types of stress: positive stress, tolerable stress, and toxic stress (Shonkoff et al., 2012). Per the EBD framework, toxic stress is more apt to cause physiological dysregulation and the resulting allostatic load (Shonkoff, 2010).

Further, influenced by the science of pediatrics, the EBD framework considers normative brain development throughout childhood and proposes that toxic stress impairs normative development via the excessive release of stress hormones (Shonkoff et al., 2012). However, supportive and nurturing caregiving, or the presence of a stable adult figure, is hypothesized to buffer the child from the consequences of stress on brain development and stress physiology. Thus, the EBD framework is useful for conceptualizing the role of chronic stress in child development within the family context.

Literature Review

In the remaining section of this chapter, I will review current findings and issues in research on the associations between child maltreatment, acute stress response reactivity, and psychopathology during preadolescence.

Child Maltreatment and Preadolescent Psychopathology

Individuals who experience child maltreatment are at a heightened risk for psychopathology, including internalizing and externalizing disorders, during late childhood and adolescence (Duprey, Oshri, & Caughy, 2017; Rogosch, Oshri, & Cicchetti, 2010; Thompson, English, & White, 2016). Internalizing symptomology includes depressive, anxious, and withdrawn symptoms, whereas externalizing symptomology includes disruptive and aggressive behaviors (Achenbach, 1991). The terms ‘internalizing’ and ‘externalizing’ typically refers to symptomology but may also refer to disorders. For example, major depression can be categorized as a type of internalizing disorder, while conduct disorder can be categorized as a type of externalizing disorder. However, there is a preference for researchers in the field of developmental psychopathology to operationalize psychopathology as a continuous

gradient of symptoms instead of as discrete disorders. This is because the developmental psychopathology perspective moves away from the medical diagnostic model of psychopathology and the taxometric study of disorders by giving more attention to the developmental course of symptomology. Thus, the current study utilizes a continuous measurement of internalizing and externalizing symptoms instead of referring to diagnostic criteria.

In developmental psychopathology-informed investigations of child maltreatment and adolescent psychopathology, it is important to consider the developmental timing in which symptoms emerge. This includes particular attention to *transition* periods, which are defined as phases in development characterized by change rather than stability, and which often include social role changes (Rutter, 1996). Further, developmental transitions such as adolescence are characterized by enhanced brain plasticity (Crone & Dahl, 2012). Preadolescence marks an important developmental transition period from childhood to adolescence, which typically occurs between the ages of 9 to 12. It is pertinent to study the links between childhood maltreatment and internalizing and externalizing psychopathology during the transition from childhood to adolescence: First, preventive interventions may be more effective when targeted to youth during key transitional periods such as preadolescence, due to the neurocognitive flexibility and plasticity characteristic of these developmental periods (Crone & Dahl, 2012). Thus, the study of childhood maltreatment and the emergence of psychopathology during preadolescence offers insight into potential intervention and prevention targets for this age group. Second, the average age of onset of several internalizing and externalizing disorders occurs during preadolescence. For example, a recent study showed that the average age-

of-onset was 11 for both anxiety disorders and impulsive-control disorders (e.g., conduct disorder, oppositional-defiant disorder; Kessler et al., 2005).

Child maltreatment and internalizing symptoms. The research literature shows a robust association between child maltreatment experiences and internalizing symptomology in late childhood and adolescence. Several large-scale longitudinal studies show that exposure to child maltreatment increases youths' risk for depressive and anxious symptoms (Collishaw et al., 2007; Thompson et al., 2016). In the longitudinal Isle of Weight study, youth who reported being maltreated were 15.5 times more likely to experience minor depression and 8.11 times more likely to experience an anxiety disorder, compared to non-abused youth (Collishaw et al., 2007). Further, in a study utilizing the Longitudinal Studies of Child Abuse and Neglect dataset, investigators found that maltreated youth were more likely to have an elevated internalizing symptom trajectory from early childhood through late adolescence, as compared to non-maltreated youth (Thompson et al., 2016). As noted previously, maltreatment is a multidimensional construct. Accordingly, there have been different findings regarding the relations among different dimensions of maltreatment with internalizing symptomology. For instance, recent findings indicated that youth exposed to sexual abuse or neglect were 3.75 and 2.83 times more likely to have internalizing symptoms that increased throughout childhood, respectively, as compared to youth who did not experience maltreatment (Yoon, 2017).

Child maltreatment and externalizing symptoms. There is also robust evidence for the association between child maltreatment and externalizing symptomology during preadolescence (Heleniak, Jenness, Vander Stoep, McCauley & McLaughlin, 2016; Oshri

et al., 2011; Thompson et al., 2016). The organizational theory of development can be used to conceptualize this association. In support of this theory, research shows that maltreatment causes developmental disruptions in the attainment of self-regulation abilities during childhood (Kim-Spoon, Cicchetti & Rogosch, 2013). In turn, a lack of age-appropriate self-regulation capacities can put youth at risk for externalizing behaviors such as disruptiveness and aggression (Eisenberg et al., 2009).

There is evidence from large-scale prospective studies of the association between maltreatment and externalizing behaviors. In a study that utilized the Longitudinal Studies of Child Abuse and Neglect sample, maltreatment history was associated with externalizing symptoms that increased over time from early childhood to age-16 (Thompson et al., 2016). Further, a study found that childhood maltreatment was significantly related to both internalizing and externalizing symptomology, and this link was mediated by parental report of the child's emotional reactivity (Heleniak et al., 2016). Some studies have also looked at associations between specific dimensions of childhood maltreatment and externalizing outcomes. For example, in a study using a sample of low-income urban youth, child protective services reports of neglect severity was significantly associated with prospective externalizing symptoms when children were age nine (Manly et al., 2013). In another study, researchers utilized a sample of pre-school aged foster children, and found that children who experienced a combination of physical abuse, sexual abuse, emotional abuse, and neglect, were significantly more likely to exhibit externalizing symptomology as compared to youth who experienced fewer maltreatment types (Pears, Kim & Fisher, 2008).

Child Maltreatment and Psychopathology: The Role of Stress Response Systems

It is clear from the research literature that childhood maltreatment is associated with preadolescent internalizing and externalizing psychopathology. However, more research is needed on the developmental mechanisms that underlie this association. As aforementioned, the developmental psychopathology perspective and the organizational theory of development suggest that experiences of maltreatment are linked with future psychopathology due to disruptions in the attainment of stage-salient tasks, such as the development of self-regulation (Cicchetti & Banny, 2014). Furthermore, evidence from the allostatic load theory suggests that early life chronic stress, such as child maltreatment, can cause disruptions in the development of stress response systems, resulting in dysregulation of allostatic processes (McEwen, 2017). The developmental psychopathology perspective and allostatic load theory can therefore be integrated to make hypotheses about the physiological mechanisms connecting early life chronic stress with adolescent psychopathology. Specifically, I hypothesize in the present study that disruptions in the development of psychobiological systems of stress-response regulation (i.e., HPA axis and the autonomic nervous system) can occur as a result of early life stress. Further, these disruptions in the development of stress-response systems can lead to cascading maladaptive effects on neurocognitive development, thereby increasing the risk for the development of psychopathology. Thus, the present study investigates stress reactivity in both the HPA axis and the autonomic nervous system as two possible mediating mechanisms linking childhood maltreatment with internalizing and externalizing outcomes in preadolescence. In the following section, I will review literature that supports the hypothesis that stress reactivity mediates the link between

childhood maltreatment and preadolescent internalizing and externalizing psychopathology.

Child maltreatment and HPA-axis outcomes. There is robust empirical support for the association between early life stress that stems from adverse rearing environments and HPA axis dysregulation. In the following subsections I will expand on this literature, including studies using animal models and with human subjects.

Animal models. Much of the early research on early life stress and physiological outcomes utilized rodent models, enabling researchers to perform experimental paradigms that randomized test subjects into stress and non-stress conditions. Seymour Levine and his research group are noted for their experimental studies that established the impact of early deprivation on stress physiology and anxious behaviors in rats (e.g., Levine, 1960). Specifically, a series of replicated experiments showed that rat pups who were not handled or groomed were significantly more likely to display an exaggerated endocrine response to stress (i.e., heightened stress reactivity) and increased fearful behaviors later in life, compared to rats who were regularly handled (Levine, 1960; Levine, 2005). Similarly, rat pups who experience long-term maternal separation (MS) are more likely to exhibit hyper-reactivity to stress, as compared to non-MS rat pups (Levine, 2005).

Cross-fostering studies with rat pups have also yielded useful information on the biological impacts of early life stress. This approach enables researchers to disentangle the influences of genetics and environment on fearfulness or stress reactivity in rat pups. For example, in a study using the early handling paradigm with rat pups, researchers operationalized early deprivation as pups who received low licking-grooming [LG]

behaviors by either the maternal or foster mother and no handling by researchers. They found that early deprivation in pups was associated with future lack of exploration, and this was mediated through having fewer glucocorticoid receptors in the hippocampus (Francis, Diorio, Liu & Meaney, 1999). Because hippocampal glucocorticoid receptors play a large part in the negative feedback regulation of cortisol, a lack of glucocorticoid receptors relates to heightened cortisol reactivity. Notably, rat pups with biological high LG behaviors that were placed with low LG foster mothers exhibited the same increased fearfulness and cortisol changes that the rat pups with biological low LG mothers exhibited – emphasizing the importance of the rearing environment in HPA axis regulation (Liu et al., 1997). Thus, experimental animal research on the effects of deprivation has repeatedly shown that deprivation causes alterations to HPA axis functioning.

Human subjects. While animal models have consistently shown that early life stress is related to heightened cortisol reactivity, findings with human subjects have uncovered mixed findings. Some researchers have investigated the link between early life stress and HPA axis dysregulation by investigating maternal responsivity and cortisol (Blair et al., 2008; Loman & Gunnar, 2010). For example, Megan Gunnar and colleagues conducted a study in which cortisol levels were measured during regular "well-baby" exams and vaccination visits at the child's 18 months and were compared to observer ratings of maternal responsivity at the child's 6 months. This study found that low maternal responsivity was associated with elevated cortisol reactivity during the child well visit (Loman & Gunnar, 2010). In another study utilizing diurnal cortisol as an outcome, experiences with child maltreatment were associated with blunted diurnal

cortisol levels in infancy and early childhood, a sign of HPA axis dysregulation (Bernard et al., 2010). Research also documents the relation between childhood maltreatment and increased cortisol reactivity in adulthood. In one study, women who experienced childhood abuse and who were diagnosed with depression showed an elevated cortisol response to a psychosocial stressor, as compared to controls with depression and no abuse, and controls with no depression and with abuse (Heim, Newport, Mletzko, Miller & Nemeroff, 2008).

Studies with older children and adolescents are not as abundant in the literature, but several have indeed established a relation between early life stress and HPA dysregulation for adolescents (Cicchetti, Rogosch, & Oshri, 2011; Mclaughlin et al., 2015). Many of these studies have related early life stress with a blunted, instead of hyper-reactive, cortisol response. In the Bucharest Early Intervention Project, one of the only experimental studies of early deprivation in humans, researchers found causal evidence of the effect of early life stress on physiological dysregulation and psychopathology (McLaughlin et al., 2015). In this study of institutionalized children in Bucharest, children were randomized into a foster care condition and a control condition (in which children remained in the institution's care). This large-scale experiment yielded several studies that reported the detrimental impacts of child deprivation. Notably, researchers found that children who remained institutionalized had a blunted cortisol response to a stressor when they were 12 years of age (McLaughlin et al., 2015). This study also presented evidence that the timing of early life stress has a significant impact on outcomes. Children who were adopted before 24 months of age showed a normal HPA

axis reactivity pattern, while children who were adopted after 24 months of age showed a more blunted and dysregulated pattern (McLaughlin et al., 2015).

Child maltreatment and autonomic nervous system outcomes. There is scarce research on the association between early life stress and autonomic nervous system function. However, emerging research suggests that early life stress and child maltreatment can impact autonomic nervous system function, leading to sympathetic hyper-reactivity to stress (El-Sheikh & Erath, 2011; McLaughlin et al., 2014). For example, researchers found that women with experiences of physical and sexual abuse had significantly higher heart rate reactions (a measure of sympathetic nervous system activity) as compared to non-abused controls (Heim et al., 2000). Further, in a sample of adolescents, McLaughlin and colleagues found that youth who experienced maltreatment were more likely to have a dysregulated autonomic response to a social stress task compared to youth who had not experienced physical, sexual, or emotional abuse (McLaughlin et al., 2014). Additionally, El-Sheikh utilized skin conductance level reactivity (SCLR), a measure of sympathetic reactivity, to test the association between family conflict, stress system regulation, and youth outcomes (2005). Findings showed that higher levels of SCLR (e.g., hyper-reactivity of the sympathetic nervous system) mediated the association between family conflict and internalizing problems for boys, but not for girls.

Fewer studies have investigated the direct effect of early life stress on biomarkers of parasympathetic nervous system function, typically measured with high-frequency heart rate variability (often called *respiratory sinus arrhythmia* or *vagal tone* in the literature). In a sample of adolescents, McLaughlin and colleagues found a modest but

significant correlation between child abuse and higher parasympathetic suppression (i.e., a blunted parasympathetic response) during a mental math task (Mclaughlin, Alves & Sheridan, 2014). Additionally, findings from the Bucharest Early Intervention Project provide evidence for the role of early life stress in predicting blunted parasympathetic responses to a stressor. Adolescents who spent more time in institutional care as children were more likely to exhibit lower heart rate variability reactivity, as compared to adolescents who had been placed earlier in foster homes (McLaughlin et al., 2015).

Some findings provide support for a moderating role of parasympathetic nervous system variables in the link between childhood adversity and youth psychopathology. In a recent study, McLaughlin, Alves, and Sheridan (2014) found that respiratory sinus arrhythmia (a measure of parasympathetic nervous system activity), but not pre-ejection period (a measure of sympathetic nervous system activity) moderated the association between childhood adversity and adolescent internalizing symptoms. Accordingly, it is unclear whether the autonomic nervous system serves as a mediating or moderating factor in the association between childhood maltreatment and adolescent psychopathology. It is possible that the role of the autonomic nervous system differs depending on developmental timing, due to the growth and plasticity of brain areas associated with the ANS throughout childhood. For example, the ANS may be more likely to serve as a mediator during childhood and early adolescence and as a moderator during late adolescence and adulthood. Thus, there is a need for more research investigating the role of both sympathetic and parasympathetic nervous system responses to stress in the association between early life stress, specifically childhood maltreatment, and youth psychopathology outcomes.

Stress response systems and internalizing and externalizing psychopathology. There is evidence linking maladaptive stress response system functioning with both internalizing and externalizing psychopathology in children and adolescents (Alink et al., 2008; Nestler et al., 2002). In particular, there is strong evidence for the link between HPA axis dysregulation (in the form of hyper-reactivity) and internalizing psychopathology, specifically depression (Heim et al., 2008). In fact, altered HPA axis responsivity to stress is one of the most prominent neurobiological risk factors for depression in adult samples (Nestler et al., 2002). These findings have also been replicated for children and adolescents with depression, with depressed adolescents more likely to exhibit hyper-reactivity to a psychosocial stressor (Guerry & Hastings, 2011). It is possible that the relation between HPA axis regulation and internalizing symptoms differs by gender. In a sample of adolescents, heightened cortisol reactivity predicted more internalizing symptoms for girls but not for boys (Natsuaki et al., 2009).

Conversely, there is also evidence for hypo-cortisolism in the etiology of depression, particularly in samples of children and adolescents who have experienced adversity (Koss & Gunnar, 2018). In a sample of low-income girls, cortisol hypo-reactivity to a cold pressor task (a physical stressor) was associated with higher levels of depression (Keenan et al., 2013). These associations may also differ depending on pubertal stage. For example, blunted cortisol reactivity was associated with major depressive disorder in girls who were earlier in puberty, while heightened cortisol reactivity was associated with major depressive disorder in girls who were more advanced in puberty (Colich, Kircanski, Foland-Ross & Gotlib, 2015).

There is mixed evidence of the role of the HPA axis in predicting externalizing disorders. In a recent meta-analysis on the associations between cortisol and externalizing symptoms in youth, the authors found there was not a significant relation between cortisol reactivity and externalizing although there was a small effect for the association between lower basal cortisol levels and higher externalizing symptoms (Alink et al., 2008). Recent findings using a sample of preadolescents from a Dutch longitudinal cohort study indicated that externalizing symptomology was related to elevated morning and evening levels of cortisol, and that this association was moderated by gender. Specifically, girls showed the strongest relation between heightened cortisol levels and externalizing (Marsman et al., 2008). Thus, it is possible that the mixed findings in this area of research are due to gender differences.

There is less research on the association between autonomic nervous system function and internalizing and externalizing symptomology. However, some evidence does support the link between autonomic nervous system functioning and internalizing symptomology. Basal, or resting state, heart rate variability is one indicator of parasympathetic system functioning that shows strong links with internalizing symptomology (Beauchaine & Thayer, 2015; Koenig, Kemp, Beauchaine, Thayer & Kaess, 2016). Specifically, lower basal HF HRV is linked to higher levels of both depression and anxiety disorders (Beauchaine & Thayer, 2015). There is also evidence for the role of autonomic nervous system reactivity (i.e., response to a stressor) in the etiology of internalizing symptomology. For example, in a sample of preadolescents, low levels of skin conductance reactivity paired with high levels of RSA reactivity were associated with higher levels of internalizing symptoms (Benito-Gomez, Fletcher &

Buehler, 2018). Additionally, El-Sheikh and colleagues have investigated the role of autonomic nervous system function and family conflict in the etiology of child behavior problems, including both internalizing and externalizing symptomology. In one of their recent studies, results showed that, for boys, lower baseline RSA was associated with more delinquency symptoms when family conflict was present (El-Sheikh, Hinnant & Erath, 2011).

The indirect link between child maltreatment and psychopathology via stress response systems. Although empirical research shows a robust link between childhood maltreatment and stress system dysregulation, and between stress system dysregulation and psychopathology, very few studies have been conducted that investigate physiological stress response function as a mediator in the association between childhood maltreatment and youth psychopathology. Further, the studies that have been conducted have largely focused on HPA axis function (Koss & Gunnar, 2018). Research has supported the hypothesized mediation between early adversity and youth psychopathology via HPA axis function (Gunnar & Quevedo, 2007; Koss & Gunnar, 2018). In one recent study using a sample of early-adolescent girls, latent trait cortisol (i.e., a latent measure of ones' diurnal cortisol patterns) mediated the association between a broad measure of early adversity and internalizing symptoms at a second time point (Stroud et al., 2018). Specifically, experiences of early adversity were associated with higher internalizing symptoms via a lower, or blunted, latent trait cortisol. However, this study utilized a sample of girls from middle- to upper-class families that were racially homogenous (86% White). Thus, there remain gaps in the literature on early adversity,

HPA axis function, and internalizing and externalizing symptomology among youth who are low-SES and/or from racial or ethnic minority groups.

Aims and Hypotheses

The aims of the present study were to investigate the indirect associations between child maltreatment and internalizing and externalizing psychopathology via cortisol reactivity and two biomarkers of autonomic nervous system reactivity. There are several hypotheses for the current study (see Figure 2.2). I hypothesized that (H1) childhood maltreatment and internalizing symptoms would be associated indirectly via blunted cortisol reactivity, and (H2) childhood maltreatment and externalizing symptoms would be associated indirectly via blunted cortisol reactivity. I also hypothesized that (H3) childhood maltreatment and internalizing symptoms would be associated indirectly via increased sympathetic stress reactivity (i.e., higher PEP-R), and (H4) childhood maltreatment and externalizing symptoms would be associated indirectly via increased sympathetic stress reactivity (i.e., higher PEP-R). Lastly, I hypothesized (H5) that childhood maltreatment and internalizing symptoms would be indirectly associated via blunted parasympathetic stress reactivity (i.e., lower HF HRV-R), and (H6) childhood maltreatment and externalizing symptoms would be associated indirectly via blunted parasympathetic stress reactivity (i.e., lower HF-HRV-R).

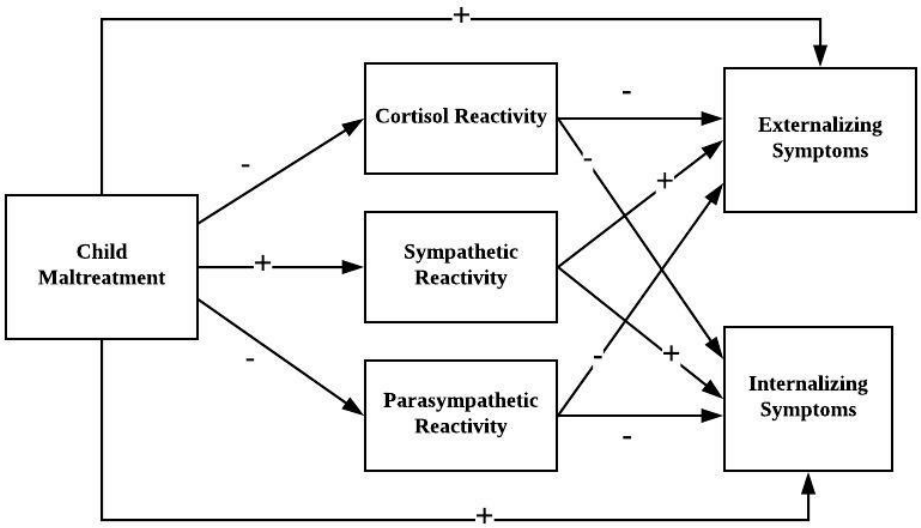

Figure 2.2. Study hypotheses.

CHAPTER 3

METHODS

In the present study, I utilized longitudinal data from the Parenting and Decision-Making Project, which were collected at the University of Georgia at the Center for Clinical and Translational Research Unit. Two waves of data were collected from Spring 2017 to Spring 2018, with approximately one year between assessments.

Participants and Procedures

Participants (N = 101) were youth aged 9-12 years old and one of their caregivers who were recruited into a larger study that examined the family and community context of youth decision making. Families were recruited from a non-metropolitan region of the Southeastern United States in an area immediately surrounding the university community. In order to be eligible for the study, youth must have been (a) between the ages of 9-12, (b) English speakers, and (c) able to read and answer questions at an elementary reading level. Additionally, families who participated must have had a household income at or below 200% of the federal poverty level, which at the time (2017) was indicated by an annual income of $48,600 for a family of four. Participants were ineligible for the study if (a) the parent was pregnant, or (b) the youth had type II diabetes or significant developmental disabilities. Of the full sample, there were approximately 8.8% (n = 8) and 16.5% (n = 16) of families who had an open or closed case with child protective services, respectively. The majority of primary caregivers

recruited into the study were the youth's biological mother (n = 91). For demographic characteristics, see Table 3.1.

Table 3.1. **Demographic characteristics of the study sample (N = 101)**

	n	%
Child's race/ethnicity		
African American	76	75.2
White	11	10.9
Latino/a	9	8.9
Native American	1	1.0
Other	4	4.0
Child's sex		
Female	53	52.5
Male	48	47.5
Primary caregiver's race/ethnicity		
African American	79	78.2
Caucasian/ White	14	13.9
Hispanic/Latino(a)	7	6.9
Other	1	1.0
Primary caregiver's sex		
Female	96	95.0
Male	5	5.0
Primary caregiver's marital status		
Married	34	33.7
Widowed	2	2.0
Divorced	18	17.8
Separated	5	5
Never married	42	41.6
Primary caregiver's education level		
Some High School	21	20.8
Completed High School	25	24.8
Some College	38	37.6
Completed College	16	15.8
Advanced Degree	1	1.0

Procedures

Recruitment. Before the data collection took place, study procedures were approved by the University of Georgia Institutional Review Board for ethical conduct in

research. During Wave 1, community recruiters were hired in order to refer participants to the study. Community recruiters were active members of their community and were paid $100 per family they recruited to the study. Participants were also recruited via online and in-person advertisements. Participants were compensated $100 for their time in the study. At Wave 2, trained research assistants re-contacted families who had consented in Wave 1 to participant in the follow-up study. Families were contacted first by phone and then by e-mail to schedule a follow-up appointment, and 71 families completed the follow-up.

Wave 1. The first wave of data collection took place at the University of Georgia's Center for Translational and Clinical Research Unit (CTRU). The data collection began in January 2017 and was completed by June 2017. Trained research staff and licensed pediatric nurses implemented all study procedures. Before any study procedures took place, parents provided their written informed consent and youth provided their informed assent. After informed consent took place, researchers collected saliva via the passive drool method in order to obtain measures of cortisol. Then, research staff connected the youth and their caregiver each to a mobile electrocardiogram (ECG) pack. The ECG was attached using seven dermal ECG electrodes which were attached on the clavicle (both sides) and lower rib cage (both sides), on the sternum, and the upper and lower spine. There were also two galvanic skin conductance electrodes placed on the palm of the participant's non-dominant hand. Electrodes were secured using medical tape to prevent movement of electrodes during data collection.

After electrodes were attached to the parent and youth, a baseline measure of autonomic nervous system activity was obtained via the ECG. Researchers instructed

participants to close their eyes and listen to a five-minute video of nature sounds (e.g., waterfalls or rainforest sounds). This baseline procedure was informed by recommendations for using HRV in behavioral research (Malik et al., 1996). After the baseline was established, researchers implemented a social stress task for the youth in order to obtain measures of autonomic nervous system reactivity. The procedures were modified from the commonly administered Trier Social Stress Test (Kirschbaum et al., 1993). Youth were instructed to answer a series of arithmetic problems aloud in front of a group of researchers and their parent. For example, youth were told to "Start at 100, and subtract by 2". The difficulty of subtraction problems increased as the task went on, and the difficulty was adjusted accordingly to the participant's ability. For example, if a participant quickly answered three questions correctly in a row, the researcher was instructed to increase the difficulty of the next set of questions. Researchers were trained to abstain from giving any feedback to the participants and to maintain a neutral facial expression throughout the task. The total task took five minutes to complete.

After the mental arithmetic task, families completed a discussion task that was intended to induce stress. Youth and their caregiver were given index cards that contained common topics of disagreement (e.g., completing homework, chores). Researchers then instructed dyads to choose three topics that they had the most disagreement on and to discuss these disagreements. The full task spanned ten minutes. The task was developed as part of the Early Head Start 5th Grade Follow-up Study (Vogel, Xue, Moiduddin, Carlson & Kisker, 2010). Following, a second collection of saliva took place approximately 20-minutes after the first stress task began in order to assess cortisol

reactivity. All autonomic nervous system parameters were collected during the first stress task (mental arithmetic).

Following the mental math and discussion tasks, youth and their parents completed a battery of survey measures. Youth completed their surveys with the assistance of a trained research assistant who read each item and the corresponding answer choices to the youth. Parents completed surveys independently on a laptop computer in a room apart from the child.

Wave 2. The second wave of data collection took place in January through June of 2018, approximately one year after the first wave. There were 71 families who completed the follow-up assessment. Two trained research assistants visited families at their home to conduct the follow-up appointment. Several families (n = 16) opted to have their appointment at the on-campus laboratory instead of their home. A graduate research assistant obtained informed consent from the parent and assent from the youth. Then, parents and youth completed assessments separately using a handheld device. The follow-up visit took approximately one hour to complete. Parents received $50 in cash and a university-branded coffee mug for completing the follow-up study.

Measures

Childhood Maltreatment

Childhood maltreatment was measured at Wave 1 utilizing subscales from the Parent-Child Conflict Tactics Scale (CTS-PC; Straus, Hamby, Finkelhor, Moore, & Runyan, 1998). Parents were asked to indicate the frequency of specific behaviors towards their child in the past year, and answer choices ranged from "0" (*this has not happened in the past year*) to "6" (*more than 20 times in the past year*). Physical abuse

was measured using six items from the Corporal Punishment subscale (α = .83). An example item was "slapped your child on the hand, arm, or leg." Neglect was measured using four items from the Neglect subscale (α = .70). An example item was "had to leave your child home alone, even when you thought some other adult should be with him/her." Emotional abuse was measured using five items from the Psychological Aggression subscale (α = .72). An example item was "swore or cursed at him/her." A maltreatment composite was also created by summing the corporal punishment, neglect, and psychological aggression subscales.

Stress System Reactivity

All physiological measures of stress reactivity were collected during the first wave of data collection.

Cortisol reactivity. Peak cortisol reactivity was measured via salivary cortisol that was collected immediately before and 20-minutes after the stress task protocol. Saliva samples were collected via the passive drool method for collecting whole saliva with SalivaBio's 2 mL cryovials and the Saliva Collection Aid (exclusively from Salimetrics, State College, PA). Immediately after collection, samples were frozen at -80°C. Samples were assayed twice using enzyme-linked immunosorbent assay (ELISA), and the average value in μg/dL was utilized for the present analysis. Peak reactivity was calculated by subtracting the baseline level of cortisol from the 20-minutes post-stress cortisol level. Thus, higher peak reactivity scores (i.e., larger difference score from baseline to peak, or larger Δ cortisol) represent a greater increase in cortisol levels from baseline to the stress task.

SNS reactivity. Pre-ejection period (PEP) reactivity was utilized to measure the function of the parasympathetic nervous system in response to an acute stressor. Cardiac impedance was measured by utilizing ECG data using the BioNex system from MindWare Technologies (Gahanna, OH), and the MindWare HRV 3.1.4 Software module. Impedance cardiography isolates the sympathetic influence on the heart by measuring the opposition to a small constant electrical current, which is generated through four electrodes that are placed on the front and back of the participant's thorax. This current is modulated, or opposed, by the amount of blood in the chest. The pre-ejection period (PEP) indicates the time interval between the initial electrical stimulation of the heart (onset of the R peak) and the opening of the aortic valve (B point of the dZ/dT wave; Lozana et al., 2007). Impedance data were ensemble-averaged in 120s epochs, in combination with R waves that were obtained from the electrocardiogram. Then, trained research assistants cross-inspected the data to correct abnormal R-R intervals, inadvertent cardiac fluctuations, and ectopic beats due to physical movement or breathing.

To measure PEP reactivity (PEP-R), a residualized change score was created using the mean level of PEP during the rest period and during the stress task:

$$\Delta PEP = \frac{PEP_{\,Stress} - PEP_{\,Baseline}}{SD(PEP_{\,Baseline}) \times \sqrt{1 - r(PEP_{\,Stress},\ PEP_{\,Baseline})}}$$

PNS reactivity. High-frequency heart rate variability (HF HRV) reactivity was utilized to measure the function of the parasympathetic nervous system in response to an acute stressor. All procedures were in accordance with current standards for measuring HF HRV in psychophysiological research (Berntson et al., 1997). In order to obtain and digitize HRV data, the BioNex system from MindWare Technologies (Gahanna, OH),

and the MindWare HRV 3.1.4 Software module was utilized. Respiration was derived using spectral analysis of thoracic impedance (Ernst, Litvack, Lozano, Cacioppo, & Berntson, 1999). First, ECG data was filtered using a .5-45 Hz bandpass, in order to remove noise related to movement and baseline drift. Then, inter-beat intervals (IBIs) were converted into 120s segments using an interpolation algorithm provided by the MindWare HRV software. Then, the Fast Fourier Transformation was used to convert the time-series domain to frequency-domain. Finally, high-frequency components of HRV were captured via power spectrum analysis, to isolate parasympathetic nervous system activity. According to previous research, high-frequency components of HRV are often used to measure parasympathetic activity (Akselrod et al., 1981; Appelhans & Luecken, 2006). The high-frequency bandpass was set at .12 to .40, with a sample rate set of 1000 Hz. In order to detect and remove noise from the heart rate data that was not previously filtered by the autonomic bandpass filter, trained researchers inspected the ECG data for artifacts (e.g., extra beats, double beats), and subsequently corrected the time series.

To calculate HF HRV reactivity (HF HRV-R), a residualized difference score was calculated using the youth's mean HF HRV during the rest period and the stress task. This type of calculation allows for the adjustment of the typical variation in baseline HF HRV (Berntson et al., 1997). Lower HRV-R residualized change scores indicate a decrease in parasympathetic influence from baseline to the stress task, which is indicative of higher levels of HF HRV-R and more self-regulation. Alternatively, higher ΔHF HRV scores represent a lack of vagal withdrawal during stress, which is indicative of dysregulation. The equation to calculate ΔHF HRV is below:

$$\Delta HF\ HRV = \frac{HRV_{\ Stress} - HRV_{\ Baseline}}{SD(HRV_{\ Baseline}) \times \sqrt{1 - r(HRV_{\ Stress},\ HRV_{\ Baseline})}}$$

Internalizing Symptoms

Youth self-reported on their depressive symptoms using the Center for Epidemiological Studies Depression Scale for children (CES-DC; Faulstich, Carey, Ruggiero, Enyart, & Gresham, 1986) at Wave 1 and Wave 2. A total sum score was created from eleven items. All items were on a Likert scale that ranged from "0" (*not at all*) to "3" (*nearly every day*), and youth reported on how often they experienced symptoms in the past two weeks. An example item is "I felt sad". The scale exhibited good internal consistency in the present sample at Wave 1 (α = .70) and Wave 2 (α = .65). The CES-DC is a modified version of the Center for Epidemiological Studies Depression Scale, and was developed specifically for use with youth aged 7 to 17.

Externalizing Symptoms

Caregivers reported on youth externalizing symptoms at Wave 1 and Wave 2 using the Child Behavior Checklist (Achenbach, 1991). Parents were instructed to report whether items described their child's behavior in the last six months. All items were assessed on a Likert scale that ranged from "1" (*not true, as far as you know*) to "2" (*very true or often true)*. The Externalizing subscale score was calculated by summing the total score of the Aggressive Behavior and Delinquent Behavior scales. The Aggressive Behavior scale consisted of 20 items, such as "gets in many fights", and the Delinquent Behavior scale consisted of 13 items, such as "doesn't seem to feel guilty after misbehaving". The Externalizing subscale score showed excellent internal reliability at Time 1 and Time 2 (α_{T1} = .90, α_{T2} = .88).

Controls

Control variables included youths' age, sex, race, pubertal stage, and their body-mass index (BMI). BMI was obtained by a licensed pediatric nurse, and youth reported on their age and sex. Primary caregivers reported on child's race and ethnicity. Youth pubertal stage was measured via three self-report questions on the Pubertal Development Scale (Petersen, Crockett, Richards & Boxer, 1988). Time 1 reports of youth internalizing and externalizing were also included in the model as a covariate.

Data Analysis Plan

Exploratory Analyses

Before testing hypotheses, descriptive statistics and bivariate correlations were examined, and data were inspected for missing data, non-normality, and distributional issues, using IBM SPSS Statistics Version 25. To test the assumption of data normality, I examined data skewness, kurtosis, and histograms for all study variables. Variance inflation factor (VIF) values were also obtained prior to hypothesis testing in order to diagnose multicollinearity.

Power analysis. I conducted a post hoc power analysis using the program GPower (Erdfelder, Faul, Buchner & Lang, 2009) in order to determine the power obtained using a sample size of $N = 101$. The program computed the achieved power for an anticipated small effect size (.15) at the significance level of $\alpha = .05$ in a multiple regression framework with eight predictors.

Missing data. Before testing hypotheses, missing data were evaluated in order to determine whether missingness was at random (MAR), completely at random (MCAR), or not at random (MNAR). It should be noted that while these tests support our

assumptions about patterns of missingness, there is not a test to *prove* the pattern of missing data. Missing data analysis is conducted in order to refute either the MCAR or the MAR missing data hypothesis (Little & Rubin, 2002). In order to test if data were MCAR, I conducted the Little's MCAR test using SPSS. Significant values of the Little's test indicate that the data are *not* missing completely at random and call for subsequent analysis. Following, I investigated correlations between missingness on study variables and the observed data utilized in the study. If the probability of missingness was related to the observed data, then MAR was supported (Schafer & Graham, 2002). Lastly, ANOVA was utilized to determine if the final sample was biased due to attrition.

Hypothesis Testing

Structural equation modeling (SEM) was used to test study hypotheses using Mplus version 7.4 with maximum likelihood estimation (Muthén & Muthén, 2010). A series of path models were constructed that evaluated the association between childhood maltreatment and internalizing and externalizing symptomology as mediated by youths' stress response reactivity. Models 1-3 tested the indirect effect between a sum score for childhood maltreatment and behavioral symptomology through physiological stress reactivity (i.e., cortisol reactivity, PEP reactivity, HF HRV reactivity). Note that mediators were tested separately in each model. Then, follow up analyses examined the indirect effect between separate types of childhood maltreatment (i.e., emotional abuse, physical abuse, and neglect) and behavioral problems, through physiological stress reactivity. For all models, control variables included sex, age, pubertal stage, race, and BMI. Nonsignificant covariates were trimmed from the final models in accordance with recommendations (Kline, 2015).

Fit of SEM models. SEM models were evaluated based on absolute and relative fit indices. Absolute fit indices include the root mean squared residual (SRMR) and the root mean square error of approximation (RMSEA); these fit indices can be used to assess how well the structural equation model replicates the actual sample data. On the other hand, relative fit indices include the Tucker-Lewis Index (TLI) and Bentler's Comparative Fit Index (CFI), which assess the fit of the model in comparison to a more restrictive baseline model. According to published standards, model fit was determined to be adequate if CFI and TLI values were at or above .95, RMSEA was at or below .06, and SRMR was at or below .08.

Indirect effects. An indirect effect model is often used to describe mediation models, in which "one variable has an effect on another variable through its influence on some intermediate variable" (Selig & Preacher, 2009). In the present study, the significance of the indirect effect was determined using the joint significance test. The joint significance test of mediation states that if path α is statistically significant, and if path β is statistically significant, then one can reject the null hypothesis of there being no mediational effect (Fairchild & MacKinnon, 2009).

CHAPTER 4

RESULTS

In this chapter, I will present findings from the present study in two sections. I will first review the descriptive analyses that were performed to better understand characteristics of study variables and evaluate distributional assumptions. In this section, I will also detail results from the power analysis, missing data analysis, and attrition analysis. The second section of this chapter will present findings from several path analysis models that were utilized to test study hypotheses. Three separate models were constructed to investigate the association between a maltreatment composite and Wave 2 internalizing and externalizing symptomology. Cortisol reactivity was tested as a mediator in the first model, heart rate variability reactivity was tested as a mediator in the second model, and pre-ejection period reactivity was tested as a mediator in the third model. Lastly, I will present results from a follow-up analysis which investigated the role of separate maltreatment types in the pathway towards stress reactivity and youth psychopathology.

Preliminary Analyses

Descriptive Statistics

The mean, standard deviation, minimum and maximum values, skewness, and kurtosis for each continuous study variable are displayed in Table 4.1. The normality of study variables was assessed by examining the skewness and kurtosis statistics and by

examining a histogram with a normal curve for each variable. Corporal punishment and neglect at Wave 1 were positively skewed at 3.39 and 4.14, and were kurtotic at 13.17 and 17.80, respectively. Thus, both variables were transformed before further analysis by taking their logarithmic value (e.g., log[x+1]). Cortisol peak reactivity was negatively skewed at -3.78 and kurtotic at 32.17 and subsequently was transformed as recommended for values with negative skewness (e.g., log[1-x]). The transformed cortisol peak reactivity score was more normal, with a skewness of .56 and kurtosis of 18.99.

Descriptive statistics were also examined for the continuous control variables (age, BMI, and pubertal stage) and dichotomous control variables (race and sex). Note that full sample descriptive statistics are in Chapter 3, Table 3.1. Race was dummy coded so that 1 = African-American and 0 = Other, and sex was coded as 1 = female and 0 = male. At Wave 1, 51.5% of youth were female (n = 52), and 75.2% of youth were African-American or Black (n = 76). Normality was assessed for all control variables by examining the skewness and kurtosis and by examining histograms with normal curves. All control variables were determined to follow a normal curve and thus were not transformed before analysis.

In order to examine the assumption of linearity, a normal p-plot of regression standardized residual values was examined, which depicted the observed cumulative distribution function (CDF) of the standardized residuals compared to the expected CDF of the normal distribution. The plot showed a linear trend, supporting the assumption of linearity in the data. In addition, multicollinearity between study variables was examined. Variance inflation factor (VIF) values for all predictor variables fell between 1.046 and

3.716. VIF values above >10 typically indicate multicollinearity (Alin, 2010). Thus, the present data did not present any multicollinearity issues among the predictor variables.

Table 4.1. *Descriptive Statistics for Study Variables*

	N	*M*	*n (%)*[1]	*SD*	Minimum	Maximum	Skewness	Kurtosis
1. Age	100	10.28		1.19	8.00	12.00	0.10	-1.21
2. BMI	101	21.96		6.41	13.59	47.00	1.46	1.46
3. Race (African-American)	101	0.75	76(75.2)	0.43	0.00	1.00	-1.19	-0.60
4. Sex (Female)	100	0.52	52(51.5)	0.50	0.00	1.00	-0.08	-2.03
5. Pubertal stage	100	6.89		1.95	3.00	12.00	1.46	2.46
6. W1 Corporal punishment	100	8.16		14.79	0.00	90.00	3.39	13.17
7. W1 Psychological aggression	100	21.74		22.39	0.00	104.00	1.07	0.70
8. W1 Neglect	100	3.66		10.43	0.00	60.00	4.14	17.80
9. W1 Maltreatment sum	101	33.56		36.86	0.00	225.00	2.10	6.92
10. W1 CESD	98	6.29		5.08	0.00	29.00	1.44	3.61
11. W1 CBCL Externalizing	101	5.07		6.49	0.00	33.00	1.99	4.26
12. W1 Cortisol reactivity	99	-0.03		0.13	-0.96	0.43	-3.78	32.17
13. W1 ΔPEP reactivity	96	-0.06		1.39	-5.18	4.36	-0.38	4.58
14. W1 ΔHF HRV reactivity	96	-0.10		1.38	-3.81	4.47	-0.05	0.94
15. W2 CESD	71	5.17		4.11	0.00	17.00	1.03	0.58
16. W2 CBCL Externalizing	71	4.61		5.37	0.00	22.00	1.27	0.78

Note. Race is coded as 1 = African-American, 0 = Other; Sex is coded as 1 = female, 0 = male. CESD = Center for Epidemiological Studies Depression Inventory for Children; CBCL = Child Behavior Checklist; ΔPEP-R = pre-ejection period reactivity change score; ΔHF HRV-R = heart rate variability reactivity change score.
[1]Indicates the number and percentage of participants coded as African-American and/or female.

Attrition Analysis

Analyses were conducted to examine the influence of attrition in the sample. T-tests were used to examine group differences between participants who participated in Wave 2 and those who dropped out of the study. Full results from this analysis are presented in Table 4.2. There was a significance group difference by age such that youth who dropped out of the study were younger than youth who completed both waves, t (98) = 2.49, $p < .05$. Additionally, chi-square tests of significance were used to determine whether Wave 2 participants differed from drop-out participants on dichotomous study variables (sex and race). Results showed no significant group differences by gender, χ^2 (1) = 1.836, $p = .174$, African-American race, χ^2 (1) = .046, $p = .830$, Hispanic/Latino

ethnicity, χ^2 (1) = .265, p = .607, or other race, χ^2 (1) = .044, p = .834.

Table 4.2. *Attrition Analyses T-Tests*

	Follow-up (n = 71)		Drop out (n = 30)			
	M	*SD*	*M*	*SD*	*t (df)*	*p*
Age	10.46	1.18	9.83	1.10	2.49 (98)	.01*
BMI	22.63	6.79	20.37	5.16	1.62 (99)	.11
Pubertal stage	6.30	1.94	6.79	2.01	0.32 (98)	.75
Income	22.50	13.38	20.36	11.87	0.61 (57)	.54
W1 Corporal punishment	7.06	12.32	10.86	19.56	-1.17 (98)	.25
W1 Psychological aggression	21.41	20.45	22.55	26.93	-0.23 (98)	.82
W1 Neglect	2.76	7.56	5.86	15.33	-1.04 (98)	.18
W1 Maltreatment sum	31.22	29.32	39.28	51.06	-0.99 (98)	.32
W1 CESD	5.79	4.49	7.54	6.23	-1.55 (96)	.12
W1 CBCL Externalizing	4.94	6.17	5.37	7.28	-0.30 (99)	.77
W1 Cortisol reactivity	-0.03	0.14	-0.01	0.07	-0.99 (97)	.33
W1 Δ PEP-R	-0.08	1.49	-0.00	1.10	-0.25 (94)	.80
W1 Δ HF HRV-R	-0.03	1.26	-0.28	1.65	0.83 (94)	.41

Note. CESD = Center for Epidemiological Studies Depression Inventory for Children; CBCL = Child Behavior Checklist; ΔPEP-R = pre-ejection period reactivity change score; ΔHF HRV-R = heart rate variability reactivity change score. Income is coded in $1000s.

Power Analysis

The program G*Power was used to perform a post hoc power analysis before hypothesis testing. A power analysis was calculated for a linear multiple regression with two tails, an anticipated small effect size of .15, an alpha set at .05 (i.e., chance of Type 1 error), 8 predictors, and a total sample size of N = 101. Results showed that the model achieved 97.07% power (1-β error probability = .9707), a value that is larger than the standard 80% (Suresh & Chandrashekara, 2012). Thus, I determined that the present study sample (N = 101) was adequately powered for the intended analyses.

Missing Data Analysis

At Wave 1, the percentage of missing data ranged from zero to 4.95%. Missing data at Wave 1 were due to participant non-response and errors in attaining physiological data. PEP reactivity and HF HRV reactivity were each missing 5 cases due to errors with

the electrocardiogram equipment, the presence of too many artifacts in the ECG data, or administration error (e.g., data not saved correctly). At Wave 2, there were missing data for 30 participants (29.7%). All missing data at Wave 2 were due to participant attrition.

Data missingness was tested using Little's Missing Completely at Random (MCAR) test. The test was non-significant, indicating that data were MCAR, and thus missingness was ignorable, χ^2 (113) = 80.252, p = .993. In data that are MCAR, participants' missing values do not depend on their responses to other study variables. An additional requirement for MCAR is that participant dropout does not depend on measured study variables (Schafer & Graham, 2002). As seen above in the attrition analysis (Table 4.2.), participant dropout was significantly related to youth age. Thus, the missing data pattern in the present study was assumed to be missing at random (MAR). Missing data is categorized as MAR when data missingness is dependent on observed data. Full information maximum likelihood (FIML) estimation is recommended for use with MAR data (Enders & Bandalos, 2001). Thus, subsequent hypothesis testing utilized FIML to fully estimate missing data. The FIML technique utilizes all cases when estimating parameters, resulting in more efficient and less biased parameter estimates compared to techniques such as listwise or pairwise deletion (Enders & Bandalos, 2001).

Bivariate Correlations

Pearson's correlations were computed for all continuous study variables (See Table 4.3.). Point-biserial correlation coefficients were calculated in order to examine the bivariate associations between dichotomous and continuous study variables, and phi

coefficients were calculated in order to investigate the bivariate associations between two dichotomous study variables (i.e., sex and race).

Bivariate correlations with maltreatment. As expected, associations between maltreatment types were significant. Corporal punishment was significantly correlated with psychological aggression, $r = .51$, $p < .001$, 95% *CI* [.340, .680], and neglect, $r = .22$, $p < .05$, 95% *CI* [.027, .413], and psychological aggression was significantly correlated with neglect, $r = .36$, $p < .001$, 95% *CI* [.175, .545]. There were also several significant associations between the maltreatment predictor variables and psychopathology outcomes. Corporal punishment was significantly associated with youth externalizing symptoms at Wave 1, $r = .27$, $p < .01$, 95% *CI* [.079, .461], and at Wave 2, $r = .32$, $p < .01$, 95% *CI* [.096, .543]. Psychological aggression was also significantly associated with youth externalizing symptoms at Wave 1, $r = .40$, $p < .001$, 95% *CI* [.219, .581], and Wave 2, $r = .40$, $p < .01$, 95% *CI* [.184, .616]. Additionally, neglect was significantly associated with youth externalizing symptoms at Wave 1, $r = .25$, $p < .01$, 95% *CI* [.058, .442], and was marginally associated with youth depressive symptoms at Wave 2, $r = .23$, $p < .10$, 95% *CI* [.000, .460].

Bivariate correlations with stress response variables. Bivariate correlations were also inspected between physiological stress response variables (Cortisol reactivity, ΔPEP reactivity, ΔHF HRV reactivity) and other study variables. Cortisol peak reactivity was significantly associated with ΔHF HRV reactivity, $r = .26$, $p < .05$, 95% *CI* [.067, .453]. Cortisol reactivity was also significantly associated with psychological aggression, $r = .21$, $p < .05$, 95% *CI* [.014, .406], and marginally associated with both neglect, $r = .15$, $p < .10$, 95% *CI* [-.048, .348], and the maltreatment sum score, $r = .20$, $p < .10$, 95%

CI [.004, .396]. There were also marginally significant associations between cortisol reactivity with youth depressive symptoms at Wave 2, $r = .22$, $p < .10$, 95% *CI* [-.010, .450], and externalizing symptoms at Wave 2, $r = .23$, $p < .10$, 95% *CI* [.000, .460]. Further, ΔHF HRV reactivity was significantly associated with corporal punishment, $r = .24$, $p < .05$, 95% *CI* [.046, .434], psychological aggression, $r = .27$, $p < .01$, 95% *CI* [.077, .463], and the maltreatment sum score, $r = .28$, $p < .01$, 95% *CI* [.088, .472]. ΔHF HRV reactivity was also significantly associated with youth externalizing at Wave 2, $r = .24$, $p < .05$, 95% *CI* [.011, .470].

Bivariate correlations with psychopathology variables. As expected, there was a significant correlation between youth externalizing symptoms at Wave 1 and at Wave 2, $r = .52$, $p < .001$, 95% *CI* [.318, .722]. There was a marginal association between youth depression symptoms at Wave 1 and at Wave 2, $r = .23$, $p < .10$, 95% *CI* [.000, .470].

Bivariate correlations with control variables. There were several significant associations between control variables. For example, age was significantly associated with BMI, $r = .30$, $p < .01$, 95% *CI* [.111, .489], and pubertal stage, $r = .52$, $p < .001$, 95% *CI* [.351, .689], and sex was associated with pubertal stage such that girls were more likely to be advanced in puberty, $r = .39$, $p < .001$, 95% *CI* [.208, .572]. Further, there were significant associations between control variables with both predictor and outcome variables. Youth age was significantly associated with reports of psychological aggression, $r = .24$, $p < .05$, 95% *CI* [.069, .451], externalizing symptoms at Wave 1, $r = .26$, $p < .01$, 95% *CI* [.069, .451], and depression symptoms at Wave 2, $r = .32$, $p < .01$, 95% *CI* [.097, .543]. Race was associated with neglect such that African-American youth were less likely to experience neglect, $r = -.25$, $p < .01$, 95% *CI* [-.442, -.058]. Lastly,

pubertal stage was associated with psychological aggression, such that youth who were more advanced in puberty were likely to experience more psychological aggression, r = .20, $p < .05$, 95% *CI* [.006, .394].

Models Testing Study Hypotheses

Direct Effect Model

A preliminary path analysis model was examined to determine the significance of associations between childhood maltreatment at Wave 1 with internalizing and externalizing psychopathology at Wave 2. The model exhibited excellent fit: χ^2 (1) = .872, p =.350, RMSEA = .000, CFI = 1.000, TLI = 1.024, SRMR = .025. The maltreatment sum variable significantly predicted internalizing symptoms at Wave 2 after controlling for internalizing symptoms at Wave 1, β = .258, *SE* = .132, p = .05, 95% *CI* [-.001, .517]. Further, the maltreatment sum variable significantly predicted externalizing symptoms at Wave 2 after controlling for externalizing at Wave 1, β = .303, *SE* = .126, $p < .05$, 95% *CI* [.056, .550]. Internalizing and externalizing symptoms at Wave 2 were significantly associated with each other, β = .300, *SE* = .087, $p < .01$, 95% *CI* [.087, .513].

Mediation Models

After examining descriptive statistics and bivariate correlations, mediational hypotheses were tested using path analysis within a structural equation modeling framework. See Table 4.4. for full results from these models. In all models, control variables were trimmed from the final model if they were not significantly related to study variables as per recommendations in the structural equation modeling literature (Kline, 2015).

Model 1 testing cortisol reactivity as a mediator. In Model 1, cortisol peak reactivity (20-minute post stress - baseline) was tested as a mediator in the pathway between childhood maltreatment and preadolescent internalizing and externalizing symptoms (see Figure 4.1). The model displayed excellent fit: χ^2 (6) = 7.313, p =.293, RMSEA = .048, CFI = .974, TLI = .935, SRMR = .046. Higher levels of child maltreatment were associated significantly with youth heightened cortisol reactivity, β = .205, *SE* = .098, p < .05, 95% *CI* [.013, .397], and heightened cortisol reactivity in turn predicted higher levels of youth depressive symptoms at Wave 2, β = .200, *SE* = .097, p < .05, 95% *CI* [.010, .390]. Additionally, there was a significant direct effect from child maltreatment to youth externalizing symptoms at Wave 2, β = .263, *SE* = .131, p < .05, 95% *CI* [.005, .521]. Age had a significant association with youth depressive symptoms at Wave 2, such that older youth were more likely to have elevated depressive symptoms, β = .375, *SE* = .097, p < .001, 95% *CI* [.186, .565]. The indirect effect from childhood maltreatment to youth depressive symptoms at Wave 2 was nonsignificant, α*β = .041, *SE* = .028, p = .145, 95% *CI* [-.014, .096], and the indirect effect from child maltreatment to youth externalizing symptoms at Wave 2 was also nonsignificant, α*β = .017, *SE* = .021, p = .420, 95% *CI* [-.136, .385]. Using the joint test of significance (Fairchild & MacKinnon, 2009), results indicated that the indirect effect between childhood maltreatment and youth internalizing symptoms via cortisol reactivity was significant. The model explained 34.8% of variance in youth externalizing symptoms and 27.3% of variance in youth depressive symptoms at Wave 2.

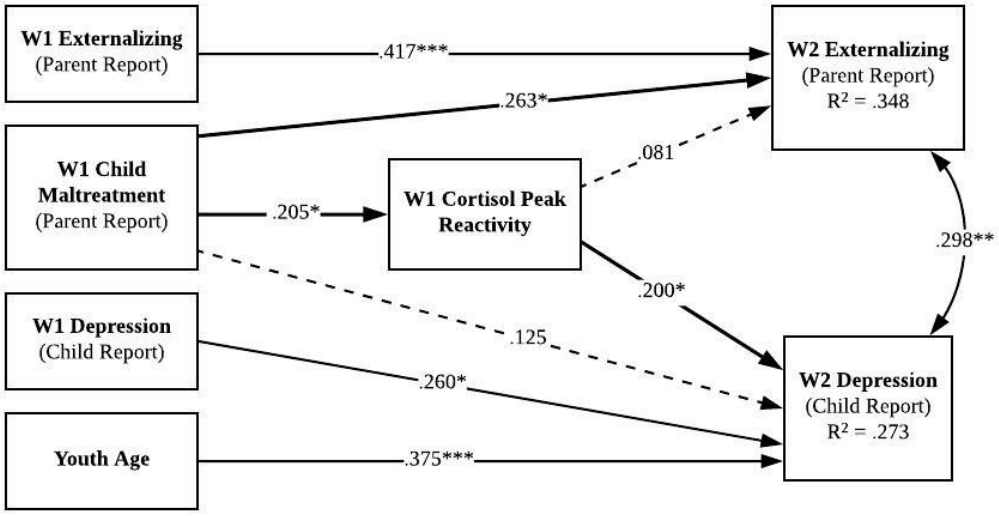

Figure 4.1. Mediation model testing the role of cortisol peak reactivity as a mediator between child maltreatment and youth internalizing and externalizing symptoms.

Note. Standardized model coefficients are presented in the model.

Model 2 testing heart rate variability reactivity as a mediator. In Model 2, the parasympathetic stress response was tested as a mediator in the pathway between child maltreatment and youth internalizing and externalizing symptoms. The model exhibited excellent fit: χ^2 (6) = 4.740, p =.577, RMSEA = .000, CFI = 1.000, TLI = 1.064, RMR = .044. Child maltreatment was associated significantly with elevated ΔHF HRV-R scores, β = .274, *SE* = .095, $p < .01$, 95% *CI* [.088, .461]. Elevated ΔHF HRV-R scores indicate lower HF HRV reactivity. Thus, as expected, child maltreatment was associated with blunted HF HRV reactivity which is indicative of stress system dysregulation. There were no significant associations between ΔHF HRV-R and the outcome variables, and the indirect effects tested in Model 2 were nonsignificant. Similar to Model 1, there was a significant association between child maltreatment and youth externalizing symptoms at

Wave 2, β = .263, *SE* = .129, $p < .05$, 95% *CI* [.011, .516], such that higher levels of maltreatment predicted higher levels of externalizing. Additionally, older youth were more likely than younger youth to exhibit depressive symptoms at Wave 2, β = .345, SE = .099, $p < .001$, 95% *CI* [.151, .539]. Overall, the model explained 37.2% variance in youth externalizing symptoms and 24.7% variance in youth internalizing symptoms at Wave 2.

Model 3 testing pre-ejection period reactivity as a mediator. Model 3 tested pre-ejection period reactivity, a measure of the sympathetic nervous system response to stress, as a mediator in the pathway from child maltreatment to youth internalizing and externalizing symptoms. The model exhibited excellent fit: χ^2 (6) = 3.314, p =.768, RMSEA = .000, CFI = 1.000, TLI = 1.173, RMR = .036. There were no significant direct effects from child maltreatment to ΔPEP-R nor from ΔPEP-R to youth internalizing or externalizing symptoms. Child maltreatment significantly predicted youth externalizing symptoms at Wave 2, β = .284, *SE* = .131, $p < .05$, 95% *CI* [.028, .540], and older youth were more likely to exhibit higher levels of depressive symptoms at Wave 2 compared to younger youth, β = .348, *SE* = .100, $p < .001$, 95% *CI* [.152, .543]. There were no significant indirect effects. Overall, the model explained 35.0% variance in youth externalizing symptoms and 23.4% variance in youth depressive symptoms at Wave 2.

Follow-up Analyses

Model 4 testing maltreatment type, cortisol reactivity, and internalizing symptoms. An additional model was tested to determine whether maltreatment type mattered in the indirect association between child maltreatment and youth internalizing symptoms via cortisol reactivity. Three maltreatment types were tested as competing

predictors in the same model: physical abuse, emotional abuse, and neglect. The resulting model exhibited poor model fit, χ^2 (5) = 35.306, p = .002, CFI = .613, TLI = .458, RMSEA = .118, SRMR = .105. The association between cortisol reactivity and youth internalizing symptoms approached conventional significance levels, β = .180, *SE* = .098, p = .067, 95% *CI* [-.012, .373], and the association between emotional abuse and cortisol youth reactivity also approached conventional significance levels, β = .224, *SE* = .118, p = .058, 95% *CI* [-.008, .457].

In accordance with recommendations in the structural equation modeling literature (Kline, 2015), the model was trimmed by removing nonsignificant paths. The trimmed model exhibited improved fit, χ^2 (2) = .563, p = .755, CFI = 1.000, TLI = 1.231, RMSEA = .000, SRMR = 018. See Figure 4.2. The indirect effect from Wave 1 emotional abuse to Wave 2 depressive symptoms via cortisol reactivity trended towards significance, α*β = .060, *SE* = .034, p = .082, 95% *CI* [-.007, .127]. The association between emotional abuse and cortisol reactivity was significant, β = .268, *SE* = .098, p < .001, 95% *CI* [.077, .459], and the association between cortisol reactivity and depressive symptoms was significant, β = .222, *SE* = .096, p < .05, 95% *CI* [.035, .410]. Thus, using the joint test of significance, the results indicate that there is a significant indirect effect from childhood emotional abuse to adolescent depressive symptoms via elevated cortisol reactivity. Overall, the model explained 26.5% of the variance in youth depressive symptoms at Wave 2.

Table 4.3. *Bivariate Correlations* ($N = 101$)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. Age	—															
2. BMI	.30**	—														
3. Race	-.20†	-.05	—													
4. Sex (Female)	.14	.04	-.05	—												
5. Pubertal stage	.52***	.19†	-.13	.39***	—											
6. W1 COP	.06	.05	-.01	.13	.05	—										
7. W1 PAG	.24*	.14	-.00	.02	.20*	.51***	—									
8. W1 Neglect	.03	.04	-.25*	.05	.06	.22*	.36***	—								
9. W1 Mltx Sum	.18†	.10	-.08	.03	.16	.69***	.85***	.57**	—							
10. W1 CESD	-.12	.15	.05	-.03	-.04	-.10	.01	.02	.01	—						
11. W1 CBCL Ext	.26**	.16	-.01	.01	.17†	.27**	.40***	.25*	.35***	.04	—					
12. W1 Cortisol-R	-.12	-.06	-.12	-.07	-.13	.06	.21*	.15†	.20†	.02	.19	—				
13. W1 ΔPEP-R	-.01	-.03	-.01	-.04	-.07	-.07	.05	-.08	-.04	-.06	-.00	.03	—			
14. W1 ΔHF HRV-R	.11	.05	-.16	.16	-.15	.24*	.27**	.18†	.28**	.13	.15	.26*	.04	—		
15. W2 CESD	.32**	.12	-.20	-.01	.11	-.04	.19	.23†	.20†	.23†	.20	.22†	-.08	.14	—	
16. W2 CBCL Ext	.16	-.07	-.01	.10	.08	.32**	.40**	.15	.42***	.16	.52***	.23†	-.00	.24*	.39**	—

Note. Race is coded as 1 = African-American, 0 = Other; Sex is coded as 1 = female, 0 = male. COP = corporal punishment; PAG = psychological aggression; Mltx = Maltreatment; CESD = Center for Epidemiological Studies Depression Inventory for Children; CBCL Ext = Child Behavior Checklist Externalizing raw score; ΔPEP = pre-ejection period reactivity change score; ΔHF HRV = heart rate variability reactivity change score. COP, Neglect, and Cortisol reactivity were transformed prior to correlation analysis.

$^{\dagger}p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$

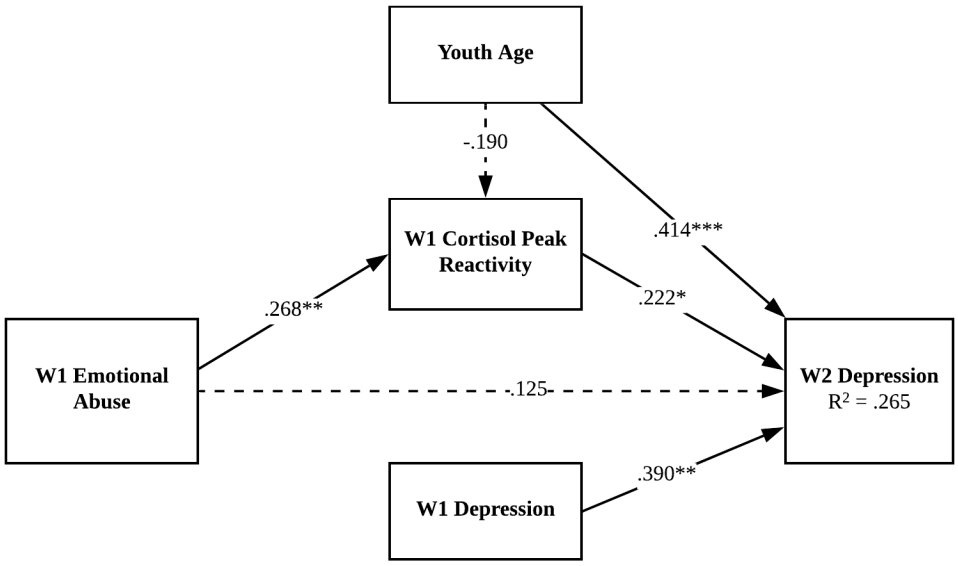

Figure 4.2. Mediation model testing the role of peak cortisol reactivity as a mediator between emotional abuse and youth depressive symptoms.

Note. Standardized model coefficients are presented in the model.

Table 4.4. Model Parameters for Path Analysis Models with Maltreatment Sum Predictor (N = 101)

Model 1	*B*	*SE*	β	*p*	95% *CI*
Maltreatment → W2 INT	.014	.094	.125	.348	[-.136, .385]
Maltreatment → W2 EXT	.039	.020	.263	.046*	[.005, .521]
Maltreatment → Cort-R	<.001	<.001	.205	.036*	[.013, .397]
Cort-R → W2 INT	16.797	8.235	.200	.039*	[.010, 390]
Cort-R → W2 EXT	8.996	10.195	.081	.380	[-.100, .261]
Age → W2 INT	1.284	.356	.260	<.001***	[.186, .565]
W1 INT → W2 INT	.210	.094	.375	.021*	[.039, .481]
W2 EXT → W2 EXT	.357	.094	.417	<.001***	[.212, .623]
Indirect Effects	α*β	*SE*	*p*	95% *CI*	
Malt → Cort-R → W2 INT	.041	.028	.145	[-.014, .096]	
Malt → Cort-R → W2 EXT	.017	.021	.420	[-.024, .057]	
Model 2	*B*	*SE*	β	*p*	95% *CI*
Maltreatment → W2 INT	.019	.015	.169	.202	[-.090, .428]
Maltreatment → W2 EXT	.039	.020	.263	.041*	[.011, .516]
Maltreatment → ΔHF HRV-R	.010	.004	.274	.004**	[.088, .461]
ΔHF HRV-R → W2 INT	.305	.347	.101	.376	[-.123, .325]
ΔHF HRV-R → W2 EXT	.605	.426	.150	.151	[-.055, .356]
Age → W2 INT	1.187	.360	.345	<.001***	[.151, .539]
W1 INT → W2 INT	.225	.096	.278	.015*	[.055, .500]
W2 EXT → W2 EXT	.347	.093	.404	<.001***	[.199, .610]
Indirect Effects	α*β	*SE*	*p*	95% *CI*	
Malt → ΔHF HRV-R → W2 INT	.028	.033	.399	[-.037, .092]	
Malt → ΔHF HRV-R → W2 EXT	.041	.032	.201	[-.022, .104]	
Model 3	*B*	*SE*	β	*p*	95% *CI*
Maltreatment → W2 INT	.021	.015	.188	.155	[-.071, .446]
Maltreatment → W2 EXT	.042	.020	.284	.030*	[.028, .540]
Maltreatment → ΔPEP-R	-.001	.004	-.033	.751	[-.235, .170]
ΔPEP-R → W2 INT	-.082	.297	-.028	.782	[-.223, .168]
ΔPEP-R → W2 EXT	-.029	.364	-.007	.937	[-.187, 172]
Age → W2 INT	1.187	.361	.348	<.001***	[.152, .543]
W1 INT → W2 INT	.224	.096	.277	.016*	[.052, .501]
W2 EXT → W2 EXT	.359	.093	.421	<.001***	[216, .626]
Indirect Effects	α*β	*SE*	*p*	95% *CI*	
Malt → ΔPEP-R → W2 INT	.001	.004	.835	[-.008, .009]	
Malt → ΔPEP-R → W2 EXT	<.001	.003	.939	[-.006, .006]	

Notes. INT = Internalizing, EXT = Externalizing, Cort-R = Cortisol peak reactivity, ΔHF HRV-R = Heart rate variability reactivity change score, ΔPEP-R = Pre-ejection period reactivity change score, W1 = Wave 1, W2 = Wave 2, Malt = Maltreatment.

*p < .05, **p < .01, ***p < .001

Model 1: χ^2 = 7.313 (df = 6), p =.293, RMSEA = .048, CFI = .974, TLI = .935, RMR = .046

Model 2: χ^2 = 4.740 (df = 6), p =.577, RMSEA = .000, CFI = 1.000, TLI = 1.064, RMR = .044

Model 3: χ^2 = 3.314 (df = 6), p =.768, RMSEA = .000, CFI = 1.000, TLI = 1.173, RMR = .036

CHAPTER 5

DISCUSSION

Childhood maltreatment has profound adverse effects on youth socioemotional adjustment and has been shown to predict the development of both internalizing and externalizing symptomology. In the present study, I investigated the underlying mechanisms in this established link. Recent research informed by allostatic load theory states that the chronic stress maltreated youth experience can disrupt physiological stress-response systems, often resulting in dysregulated (e.g., hyper- or hypo-reactive) responses to future acute stressors (Juster et al., 2011). Consistent with this theory, I hypothesized that experiences of child maltreatment would be linked with heightened internalizing and externalizing psychopathology in early adolescence and that this would be mediated by dysregulated physiological responses to acute stress. The findings partially supported my hypotheses, and inform theory on the mechanisms linking childhood maltreatment with psychopathology during early adolescence. Results suggested that dysregulation of the HPA axis, indicated by cortisol hyper-reactivity, mediated the association between childhood maltreatment in general, and emotional abuse in particular, and prospective internalizing symptomology. Additionally, the present study provided evidence for an association between childhood maltreatment and blunted parasympathetic responses to acute stress, as measured by heart rate variability reactivity. There were no significant findings regarding the mediational role of

sympathetic nervous system reactivity in the association between child maltreatment and preadolescent psychopathology. Further, although childhood maltreatment was significantly associated with youth externalizing symptoms, there was no evidence that this association was mediated through acute stress response reactivity in the HPA, sympathetic, or parasympathetic systems. Overall, the results of the present study show that the chronic stress of child maltreatment can alter youths' ability to respond adaptively to acute stress, and this self-dysregulation can predict internalizing symptoms in preadolescence.

Childhood Maltreatment and Preadolescent Psychopathology

Study findings are consistent with developmental psychopathology perspectives which suggest that experiences of child maltreatment have significant and lasting impacts on youth mental health (for a review, see Jaffee, 2017). In the present study, reported experiences of childhood maltreatment at Wave 1 were associated significantly with both internalizing and externalizing psychopathology at Wave 2 after controlling for symptomology at the previous timepoint. These results also corroborate a recent meta-analysis that investigated the association between child maltreatment and long-term mental health outcomes (Norman et al., 2012). The meta-analysis showed that different types of child maltreatment (i.e., physical abuse, emotional abuse, and neglect) were associated with a 54% to 177% increase in risk for depressive disorders as compared to non-abused individuals. Further, the meta-analysis showed a significant association between child maltreatment and childhood behavioral and conduct disorders (Norman et al., 2012).

Notably, the association between childhood maltreatment and Wave 2 externalizing symptoms remained significant after introducing the proposed physiological mediators into the model. It is likely there are other mechanisms in the pathway between childhood maltreatment and youth externalizing psychopathology. Indeed, in a prospective study by Heleniak et al. (2016), findings showed that youths' behavioral responses to distress and emotional reactivity each mediated the association between child maltreatment and adolescent externalizing psychopathology symptoms. There is also evidence that personality characteristics mediate the association between child maltreatment and youth externalizing symptoms. For example, one study found that ego under-control (a personality trait characterized by disinhibition) mediated the association between maltreatment severity and externalizing symptoms in a longitudinal sample of youth (Oshri et al., 2011). Future research will benefit from considering the interplay between behavioral *and* physiological factors in the pathway from early life stress to adolescent externalizing psychopathology.

Mediation through Acute Stress Response Reactivity

HPA Axis

Findings showed that childhood maltreatment was indirectly associated with internalizing symptomology via a heightened cortisol response to acute stress. This result partially supported study hypotheses regarding acute stress reactivity in the HPA axis as a mediator of the association between child maltreatment and internalizing symptomology at Wave 2. However, findings were contrary to my prediction that mediation would be via *hypo*-reactivity to acute stress. In this sample of low-SES preadolescents, experiences of childhood maltreatment were associated with a heightened, or hyper-reactive, cortisol

response to an acute psychosocial stressor. This heightened cortisol response was, in turn, associated with elevated internalizing symptomology.

The finding that child maltreatment was associated with cortisol hyper-reactivity is consistent with Miller, Chen, and Parker's biological embedding of childhood adversity model (2011). According to their theory, adverse childhood experiences such as maltreatment result in the biological embedding of stress *and* behavioral tendencies such as hyper-vigilance to threat. Hyper-vigilance to threat may be reflected physiologically by cortisol hyper-reactivity. This theory and the present study's findings are also consistent with other research in animal and clinical samples. The work of Michael Meaney's research group has shown that experimentally inducing early life stress predicts cortisol hyper-reactivity in rats, and this hyper-reactivity is associated with fearful behaviors (Francis et al., 1999). Furthermore, in a study of healthy women, researchers found that childhood experiences of physical and sexual abuse were associated with increased cortisol reactivity to a social stressor, particularly among women who also were diagnosed with major depression (Heim et al., 2000). Similarly, in a sample of adolescents, youth with maltreatment experiences exhibited significantly higher cortisol reactivity to social stress. However, this effect only existed for youth with minimal or moderate depressive symptoms, as youth with severe depression exhibited blunted cortisol reactivity (Harkness, Stewart & Wynne-Edwards, 2011). It is possible that hyper-cortisolism is a unique attribute of individuals who experienced early life stress such as child maltreatment and who simultaneously exhibit mild to moderate severity mood disorders. Thus, hyper-cortisolism may be one indicator in a unique etiological pathway for depression shared by youth who experience early life trauma.

Contrary to the findings of the present study, several studies have shown an association between early life stress and cortisol hypo-reactivity (e.g., blunted reactivity) in adolescence and adulthood. However, many of the studies linking early life adversity and blunted cortisol reactivity have defined early life adversity as neglect or deprivation, as opposed to abuse. For example, there is strong evidence of the link between early life stress and cortisol hypo-reactivity from the Bucharest Early Intervention Project (McLaughlin et al., 2015; Zeanah et al., 2003). In one of the few randomized experiments with humans in the field of developmental psychology, researchers randomized institutionalized children in Romania into two groups. The control group consisted of children who remained in institutional care, while the experimental group consisted of children who were placed into high-quality foster homes (Zeanah et al., 2003). Researchers then collected follow-up data until youth reached adolescence. In one study utilizing follow-up data when participants were aged 12, researchers tested cortisol and autonomic reactivity to a psychosocial stressor and then compared results from the institutionalized and foster-care groups, along with a group of adolescents who were never institutionalized. Results revealed that youth who were institutionalized had a significantly more blunted cortisol response to a social stressor, as compared to the foster care and never-institutionalized group (McLaughlin, Sheridan et al., 2015). However, results may have been different in samples of youth who were abused as opposed to deprived via institutionalization. Indeed, the recent *threat and deprivation model* purports that threatening conditions (e.g., physical or emotional abuse) and depriving conditions (e.g., neglect) have distinct impacts on youth neurocognitive and psychological development (Sheridan, Peverill, Finn & McLaughlin, 2017).

It is also possible that the psychosocial stress task used in the current study influenced the results and contributed to the significant association between reports of child maltreatment and youth hyper-cortisolism. Most studies examining cortisol and autonomic reactivity, including the present study, have utilized the Trier Social Stress Test or a mental arithmetic task to produce a physiological stress response. In these tasks, researchers instruct individuals to perform either a speech task or an arithmetic task in front of an evaluative group of researchers (Kirschbaum et al., 1993; Lovallo, 2015). The present study utilized a modified version of the Trier Social Stress Test. In order to induce stress, we instructed youth to answer a series of arithmetic problems out-loud and without a pencil and paper, similar to traditional mental arithmetic stress manipulations. However, in contrast to typical stress tasks, the primary caregiver sat beside their child during the stress task in order to enhance the contextual validity of the task. This may have altered the results of the present study. The presence of a supportive parental figure can buffer the physiological effect of stress for youth, particularly in regard to HPA axis reactivity (Koss & Gunnar, 2018). For example, Hostinar and colleagues (2015) found that the presence of a parent significantly reduced children's physiological reaction to a psychosocial stressor (Trier Social Stress Test). In fact, amongst children aged 9 to 10, there was no difference in their pre-stress and 20-minute peak cortisol level when the stress task was performed in a parental support condition. This is in contrast to children who performed the task with strangers, as these children exhibited a significant and expected cortisol increase after the stress task was administered (Hostinar et al., 2015). Thus, for the 9- to 12-year old children in the present sample, the presence of a parent during the stress task may have had stress-buffering effects.

Relatedly, the stress induced by the mental arithmetic task may have been increased for youth who were with non-supportive or maltreating parents during the stress task. It is likely that abusive or neglectful parents do not carry the same stress-buffering effects that supportive parents do, hence increasing the child's physiological reaction to the mental arithmetic stress task (Koss & Gunnar, 2018). More so, it is possible that the presence of a maltreating caregiver induces even more stress than intended from the mental arithmetic task. Thus, in the present study, youth who were maltreated may have exhibited an elevated cortisol response to the mental arithmetic stress task as compared to their non-maltreated peers, due in part to the presence of their caregiver during the task.

Another key finding in the present study was that youths' cortisol hyper-reactivity to a psychosocial stressor was related to elevated depressive symptoms. This finding is contrary to the hypothesis that blunted cortisol reactivity would be linked to internalizing symptomology. However, there is some support in the literature for a link between cortisol hyper-reactivity and depressive symptoms among adolescents. In one study of at-risk adolescent girls, an exaggerated cortisol response to a psychosocial stressor was related to increased rates of suicidal ideation (Giletta et al., 2015). Girls who were rated as having a hyper-responsive cortisol reaction to psychosocial stress were 4.98 times more likely to report suicidal ideation at the time of the study, and 16.23 times more likely to report suicidal ideation at a three-month follow-up study, as compared to girls who exhibited a normative cortisol response (Giletta et al., 2015). Another study found that adolescents with major depression exhibited an exaggerated cortisol response to the Trier Social Stress Test, compared to adolescents with no depression diagnosis (Rao,

Hammen, Ortiz, Chen & Poland, 2008). The results of the present study corroborates the literature suggesting a link between cortisol hyper-reactivity and internalizing symptomology.

Parasympathetic Nervous System

There was not any evidence in the present study for mediation between childhood maltreatment and youth internalizing or externalizing symptoms via parasympathetic functioning. However, some support was evident that severity of childhood maltreatment was associated with a blunted parasympathetic response to the psychosocial stressor. In this study, high-frequency heart rate variability was utilized as a measure of the parasympathetic system's response to acute stress. Heart rate variability is a measure of the amount of vagal withdrawal that occurs during acute stress. After a stressor occurs, the withdrawal of the vagal nerve allows the sympathetic nervous system to elicit an adaptive fight-or-flight response (Porges, 1995). In the present study, child maltreatment was linked with less vagal withdrawal and consequently a less adaptive response to the acute stressor. This was expected and consistent with previous research (e.g., McLaughlin et al., 2015). For example, McLaughlin and colleagues utilized data from the Bucharest Early Intervention Project and found that institutionalization, a severe form of early life adversity, was linked with a blunted parasympathetic response to acute stress (McLaughlin et al., 2015).

Unexpectedly, parasympathetic nervous system reactivity was not associated with depressive nor externalizing symptoms in the sample of adolescents utilized for the present study. It is possible that parasympathetic function in general, and heart rate variability in particular, serves as a moderator of early life adversity and adolescent

psychopathology as opposed to a direct risk factor (McLaughlin et al., 2014). For example, in a community sample of adolescent girls, McLaughlin and colleagues found that heart rate variability moderated the association between exposure to childhood adversity and internalizing symptoms (Mclaughlin et al., 2014). Specifically, low resting heart rate variability and blunted heart rate variability reactivity to a psychosocial stressor exacerbated the association between childhood adversity and internalizing symptoms. There was a similar pattern of results in a study performed by El-Sheikh and colleagues (2006) using a sample of elementary-aged children. Exposure to family conflict was associated with greater internalizing symptoms, and this was exacerbated by blunted heart rate variability (i.e., lower vagal withdrawal during a stressor; El-Sheikh & Whitson, 2006). Thus, the absence of a direct link between heart rate variability reactivity and youth psychopathology in the present study may be attributed to the role of parasympathetic activity as a moderator, instead of a direct risk factor, in the etiology of youth internalizing and externalizing symptoms.

Sympathetic Nervous System

Although findings showed that child maltreatment was significantly associated with cortisol hyper-reactivity and blunted heart rate variability, there was no direct association between child maltreatment and sympathetic nervous system reactivity (PEP-R). Hypothetically, child maltreatment may have a greater impact on systems that play a regulatory role in the stress response such as the parasympathetic system and the HPA axis rather than the more reflexive fight-or-flight response of the sympathetic nervous system. There is some evidence that chronic stress has differential impacts on the sympathetic nervous system as compared to other stress-response systems. In one study,

researchers utilized a group of healthy adults to test stress reactivity of the HPA axis and sympathetic nervous system after a laboratory stressor was administered repeatedly for four weeks. They found that this repeated stress caused habituation (decreased response) of the HPA axis, but there was no impact of the repeated stress on the sympathetic nervous system (Schommer, Hellhammer & Kirschbaum, 2003). It is also possible that the laboratory stressor that was used in the present study did not sufficiently activate the SNS. Different stress tasks have been shown to produce differential responses in the sympathetic, parasympathetic, and HPA systems. Scholars such as Obradovic (2012) have suggested that characteristics of the stressor can impact stress reactivity. Mild stressors that are social in nature (e.g., mental arithmetic in front of peers) have been reliably shown to activate the acute stress response in the parasympathetic nervous system and the HPA axis. Alternatively, stressors related to the threat of punishment have been shown to consistently activate a response in the sympathetic nervous system (Obradović, 2012). Additionally, pre-ejection period reactivity (the marker of sympathetic reactivity utilized in the present study) has been implicated in reward sensitivity and approach behavior (Beauchaine, 2012). Thus, the characteristics of the laboratory stress task utilized for the present study may have influenced the parasympathetic nervous system and HPA axis more than it affected the sympathetic nervous system.

Maltreatment Type

Present findings indicated that emotional maltreatment may be particularly detrimental for youth in terms of their HPA stress response and internalizing symptoms. In a path analysis model, emotional maltreatment significantly predicted cortisol

reactivity and subsequent internalizing symptomology independent of the effects of physical abuse and neglect. Several other studies have highlighted the important role that emotional abuse plays in predicting a diverse array of adverse youth outcomes. In a recent study investigating the impact of child maltreatment on mental health symptoms in a large birth cohort, Mills et al. (2013) found evidence for the harmful role of emotional abuse on psychopathology during adolescence. Results showed that adolescents with a history of substantiated emotional abuse were 3.38 times more likely to exhibit internalizing behavior and 2.88 times more likely to exhibit externalizing behavior, as compared to youth with no maltreatment histories and after controlling for other maltreatment types (Mills et al., 2013). Additional evidence for the role of emotional abuse in predicting poor mental health outcomes comes from a study completed by Duprey and colleagues using a sample of low-SES young adults. In this study, experiences of childhood emotional abuse predicted lower self-esteem and subsequent suicidal ideation, beyond the influence of other maltreatment types (Duprey, Oshri & Liu, 2018).

The HPA axis is socially-regulated, and thus it may be more influenced by emotional abuse as compared to other abuse types. The parent-child relationship plays a crucial role in the development of the HPA system (Gunnar et al., 2015). Research from animal models and humans has converged to suggest that caregivers help to regulate cortisol levels for infants and young children (Hostinar & Gunnar, 2013). According to Tarullo and Gunnar (2006), child maltreatment in infancy and childhood may disrupt the HPA system and result in acute stress dysregulation. This may be particularly true for parent-child dyads in which emotional abuse is present. Emotional abuse is comprised of

a variety of parental behaviors towards the child and can be defined as "persistent, non-physical, harmful interactions with the child by the caregiver, which include both commission and omission" (Glaser, 2011). In parent-child dyads in which the parent is emotionally abusive, there are likely fewer opportunities for the child to learn to self-regulate their emotions via coregulation with a caregiver. However, more research is needed that directly investigates the role of emotional abuse in the development of stress reactivity in youth.

Implications

The findings of this dissertation have several implications for clinical and prevention science. Results suggested that child maltreatment can disrupt physiological stress-response systems during preadolescence, giving rise to a dysregulated response to acute psychosocial stressors. Thus, clinicians who work with youth who have experienced child maltreatment may work with their clients to develop more adaptive responses to acute stress. Adolescence is a transitional developmental period that is marked by an array of stressors in the family, school, and peer domains. Youth who have been maltreated and who do not respond adaptively to these stressors may be at risk for internalizing psychopathology, as indicated by the present study. Techniques such as mindfulness relaxation have been shown to produce changes in stress-response systems and therefore might be incorporated into therapeutic services for maltreated youth in order to facilitate more adaptive responses to stress (Garland, Gaylord, Boettiger & Matthew, 2010). Additionally, cognitive-behavioral therapeutic techniques that enhance emotion regulation and coping may be particularly beneficial for maltreated youth who are highly reactive to acute psychosocial stressors (Feather & Ronan, 2009). The findings

of the present study also emphasize the important role that family plays in the etiology of adolescent mental health symptomology. Specifically, findings showed that maltreatment in the family context increases the risk for internalizing and externalizing psychopathology for preadolescents. Therapeutic techniques that enhance parenting skills and repair parent-child relationships (e.g., attachment-based treatments) have the potential to prevent or alleviate preadolescent psychopathology symptomology (Liddle et al., 2000).

There are also several implications of the present study for prevention science. The findings are consistent with the literature suggesting that child maltreatment predicts youth psychopathology, including both internalizing and externalizing symptomology (Cicchetti & Banny, 2014). Primary prevention efforts to reduce child maltreatment are vital for preventing mental and behavioral disorders for youth. For example, the Nurse-Family Partnership (NFP) is a prevention program that aims to reduce the risk for child abuse and neglect by providing support to new mothers during pregnancy and their child's first two years (Olds, 2008). Primary prevention programs that target families when children are young may reduce the likelihood of child maltreatment and have positive cascading effects on youth mental health. The present findings also have relevance for selective prevention programs that aim to reduce psychopathology and increase overall wellness for youth who have been maltreated, including youth in foster care settings. These targeted prevention programs may benefit from the inclusion of activities that help youth adaptively cope with the acute stressors that they experience in daily life. One such activity might be mindfulness-based meditation, as there is some experimental evidence that mindfulness-based prevention programs result in more

adaptive physiological and psychological responses to acute stress (Garland et al., 2010; Hoge et al., 2013). However, it will be important for future translational research to clarify the etiological pathway between child maltreatment and youth psychopathology via stress reactivity in order to better inform prevention efforts.

Limitations

There are several limitations to the present study that should be considered. First, the present sample consisted of 101 youth and their caretakers, which limited the statistical analysis that could be performed due to power considerations. Additionally, the sample was recruited from a non-urban setting in Northeast Georgia and consisted of families who were at or below 200% of the poverty level. The majority of the sample (75%) identified as African-American. Due to these unique sample characteristics, the results from the present study may not generalize to the general population of youth in the United States nor to other specific youth demographics. Additionally, the families in the sample were not recruited on the basis of risk for either child maltreatment nor for mental health disorders despite being at or below 200% or below of the poverty line. The findings in the present study may differ in a more at-risk or a clinical sample of youth. Additionally, the maltreatment that youth in the present sample experienced, per the parent-reported measures used, was not particularly severe compared to other samples of maltreated or foster-care youth (for example, the sample of institutionalized children in the Bucharest Early Intervention Project). It is possible these youth simply were not exposed to enough chronic and toxic stress to exhibit the hypo-cortisolism that studies have often found among maltreated youth, or that the parent-reported measures did not adequately capture the severity of maltreatment in the sample. However, the sample

utilized for the present study is also a strength. There is limited research on low-SES youth from non-urban populations despite the heightened risk for adverse childhood experiences and psychopathology in this population (Fontanella et al., 2015; Sedlack et al., 2010).

Limitations also exist in regard to the measures that were utilized. First, there are limitations regarding the physiological stress response parameters that were used. Specifically, we used peak cortisol reactivity to assess the activity of the HPA axis in response to a psychosocial stressor. Peak cortisol reactivity assesses the individual's cortisol levels 20 minutes after a stressor has been administered, which is the average amount of time that cortisol levels take to reach their highest level during the stress response. Many experts have also recommended measuring the recovery of the HPA axis by obtaining a third cortisol measurement approximately 45-minutes or more after the stressor has been administered (Clements, 2013). However, the present study did not include this 40-minute measurement due to logistical constraints. Thus, it is unknown whether the recovery of the HPA axis was related to childhood maltreatment or psychopathology in the present sample. Additionally, as described prior in the discussion, the psychosocial stress task utilized in the present study may have influenced the psychophysiological stress parameters that were obtained. Specifically, the presence of a parent may have buffered youths' HPA reaction to stress, while the mental math task may not have elicited enough threat to produce a sympathetic nervous system stress response. Additionally, the data used for the present study did not include physiological stress reactivity parameters for Wave 2. This limits the ability to make causal inferences about the associations between physiological stress reactivity and internalizing symptomology.

Second, parent report on the Conflict Tactics Scale was used to assess childhood maltreatment. It is likely this measurement was biased due to participants' hesitation to report adverse parenting practices. Parents were informed by researchers during the informed consent that the research team would be mandated to report any suspected child abuse and neglect to authorities. Thus, it is likely that harsh parenting and neglect were underreported by parents. However, despite this likelihood of underreporting, the present study did reveal associations between child maltreatment and youth outcomes. Thus, these associations would likely be stronger if parents fully reported on physically abusive, emotionally abusive, and neglectful parenting practices.

Further, the measurement of child maltreatment did not include sexual abuse due to unavailability of these data. Sexual abuse is one of the primary types of child maltreatment and has been shown to have relations with youth physiological reactivity and psychopathology (Heim et al., 2000; Tremblay et al., 2008). The inclusion of sexual abuse in the analysis may have provided unique information about the associations between child maltreatment, physiological stress reactivity, and preadolescent psychopathology. Lastly, the Conflict Tactics Scale does not allow for the examination of how childhood maltreatment timing affects youth outcomes. It has been suggested in the literature that timing of maltreatment (e.g., the age of a child at which maltreatment occurs) can have a significant impact on developmental outcomes. For example, neglect occurring in early childhood may have more detrimental impacts on youths' internalizing symptomology and cognitive outcomes, as compared to neglect occurring later in childhood (Duprey, Oshri & Caughy, 2017; Sheridan et al., 2017).

Directions for Future Research

Due to the mixed findings in the literature, further research is required to elucidate the associations between child maltreatment, physiological stress reactivity, and adolescent psychopathology. It may be important for future research to consider developmental timing in the pathway from childhood maltreatment to stress reactivity and psychopathology. As noted previously, the age of a child at which maltreatment occurs and the chronicity of maltreatment can likely influence youth outcomes (Hostinar & Gunnar, 2013). For example, maltreatment that occurs during sensitive periods in development (i.e., windows in development when experience has a particularly large impact) may be more strongly linked with adverse outcomes (O'Connor, 2016). Much of the literature has identified early childhood as a sensitive period for youth's socioemotional development, as well as for the attunement of the HPA axis (Hostinar & Gunnar, 2013). For example, using data from the National Longitudinal Study of Adolescent Health, researchers found that physical and sexual abuse that occurred in early childhood (defined as between birth and age-5) was more strongly linked with adolescent depressive symptoms, compared to maltreatment occurring in other developmental periods (Dunn et al., 2013). Additionally, results from a longitudinal neuroimaging study conducted by Luby et al. (2016) showed that maternal support during the preschool years had a lasting impact on hippocampal volume during adolescence, a brain area salient to the stress response. Recent research has also identified adolescence as a sensitive period for the development of stress response systems and psychopathology due to the heightened brain plasticity during this time (Fuhrmann et al., 2015). Pertinent to the present study, several investigations show that adolescents may be particularly

sensitive to the effects of stress (Holder & Blaustein, 2014). Accordingly, the associations amongst childhood maltreatment, acute stress responsivity, and psychopathology may differ depending on the age in which these constructs are measured. Future studies should consider developmental timing and sensitive periods when examining the impact of maltreatment on adolescent stress reactivity and socioemotional outcomes.

Related to the issue of developmental timing and sensitive periods, it may be important to consider the role of puberty in future investigations of early life adversity, stress reactivity, and adolescent psychopathology. Some research indicates that the association between cortisol reactivity and youth psychopathology may differ depending on youths' pubertal stage. For instance, Hankin et al. (2010) investigated the role of cortisol reactivity in predicting depression among a group of children and adolescents. Their findings showed that cortisol hypo-reactivity in response to the Trier Social Stress Test was related to depressive symptomology among pre-pubescent youth, and cortisol hyper-reactivity was related to depressive symptomology among post-pubescent youth. Thus, puberty may act as a "switch-point" for youths' physiological response to stress. Indeed, the gonadal hormones that are the primary mediators of puberty (androgens and estrogens) impact brain development including areas in the brain that are associated with function of the HPA axis (Heim & Binder, 2012). In order to develop a more robust body of knowledge on the association between stress reactivity and psychopathology during adolescence, it is important for future studies to consider the role of puberty.

Lastly, more research is needed to determine how the interaction of the autonomic nervous system and HPA systems influence the etiology of youth psychopathology. Due

to limitations of sample size, the present study was unable to examine the interactive role of the sympathetic, parasympathetic, and HPA systems. These systems work together to produce an integrated physiological response to stress. Thus, the interactive effect of these systems may be a better predictor of psychopathology as compared to a unitary measure. Indeed, a study using a sample of youth who had recently experienced Hurricane Katrina showed that blunted cortisol was related to lower self-esteem, but only for youth with heightened sympathetic nervous system reactivity (Vigil, Geary, Granger & Flinn, 2010). Alternatively, researchers have also found that cortisol hyper-reactivity was related to internalizing and externalizing problems only for youth with reduced sympathetic reactivity (Allwood, Handwerger, Kivlighan, Granger & Stroud, 2011). Thus, future research is needed to determine how the autonomic nervous system and HPA axis interact to predict psychopathology, particularly during the adolescent developmental period.

Conclusion

The present study aimed to disentangle the association between childhood maltreatment, acute stress reactivity, and internalizing and externalizing symptomology in a sample of low-SES preadolescent youth. Youth raised by abusive or neglectful caregivers experience chronic stress, and this stress can get "beneath the skin" and change the way individuals react physiologically to acute stress. Stress dysregulation, in turn, has various negative impacts that include internalizing symptomology. The results of the present study provide further evidence that child maltreatment can negatively affect youth in terms of both physiological and socioemotional outcomes. Specifically, findings shed light on an etiological pathway between child maltreatment (and more

specifically, emotional abuse) and youth depressive symptoms via heightened cortisol responsivity to an acute psychosocial stressor. These findings enhance the present literature on the association between early life adversity, stress reactivity, and youth psychopathology, by examining three types of stress reactivity (e.g., heart rate variability reactivity, pre-ejection period reactivity, and cortisol reactivity), and by utilizing a sample of non-urban low-SES preadolescents. There are several practice and prevention implications for this work which I have delineated in this chapter. Ultimately, knowledge on the mechanisms that underlie the association between child maltreatment and adolescent psychopathology can inform clinical work and prevention programs to reduce the rates for psychopathology and increase mental wellness among at-risk adolescents.

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