The association between stress and illness is well recognized. One recently proposed pathway between these constructs is the Allostatic Load framework, which is a biological-process model in which chronic stress is linked to physiological dysregulation. The current study tested one part of the Allostatic Load process model by looking at a spectrum of chronic stressors experienced in everyday life by healthy, typically-developing children during middle childhood, to find out whether their exposures are associated with the development of Allostatic Load at age 15. This was done by utilizing the National Institute of Child Health and Development Study of Early Child Care and Youth Development (NICHD SECCYD) data and drawing on the Allostatic Load model. The level of chronic stress experienced during middle childhood is associated positively with Allostatic Load in adolescence. The Home/Family context is more predictive of Allostatic Load in adolescence than stress experienced in the Extrafamilial context. However, this relationship is moderated by the sex of the research participant: the relationship between Home/Family stress and Allostatic Load was only significant for males. In contrast, the relationship between Extrafamilial stress and Allostatic Load was only significant for females.
APPROVAL PAGE

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ACKNOWLEDGEMENTS

The author wishes to acknowledge her dissertation chair and advisor Dr. Marion O’Brien and committee members Dr. Linda Hestenes, Dr. Danielle Crosby, and Dr. Mark Fine for their investment, support, and guidance during my doctoral education. A special thanks to Dr. Marion O’Brien for her continued support and encouragement. I greatly appreciate your mentorship throughout this journey and could not have accomplished this goal without you. Also, thank you to Drs. Margaret A. Pericak-Vance and Marcy C. Speer, who helped me take my first steps in research. To my sons, Max and Sam, thank you for the unconditional love, support and encouragement throughout my education. Finally, to my friend Sharon, you have given me more strength than you will ever know.
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CHAPTER I
INTRODUCTION

The association between stress and illness was recognized centuries ago (Celsius, trans. 1815). However, even in modern times the psychological and biological pathways that connect them remain largely unknown. In 1993, McEwen and Stellar (McEwen & Stellar, 1993) proposed the Allostatic Load framework, which is a biological-process model in which chronic stress is linked to physiological dysregulation, which, in turn, is linked to illness. To date, research testing this mediation model has focused mainly on adult populations (Kubzansky, Kawachi, & Sparrow, 1999; Seeman, Merkin, Crimmins, Koretz, Charette, & Karlamanga, 2008; Seeman, Rowe, McEwen, & Singer, 2001). In children, both the relationships between stress and illness and, more specifically, between chronic stress and allostatic load (a quantitative measure of the biological effects of chronic stress), are nascent areas of research. Within these defined topics, there is a disproportionate amount of attention focused on early childhood development compared to the middle childhood period, particularly on the relationship between toxic stress and later outcomes (American Academy of Pediatrics (AAP), 2012; Centers for Disease Control (CDC), 2010; DeBellis, 2001). In addition to the lack of attention on middle childhood, little is known about the more specific relationship between chronic stress and allostatic load. Thus, the current study proposed to test one part of the allostatic load process model by looking at a spectrum of chronic stressors experienced in everyday life
by healthy, typically-developing children during middle childhood, to find out whether their exposures are associated with the development of allostatic load at age 15. This was done utilizing the National Institute of Child Health and Development Study of Early Child Care and Youth Development (NICHD SECCYD) data and drawing on the Allostatic Load model (McEwen & Stellar, 1993; McEwen, 1998).

**Construct Overview**

**Chronic Stress**

Given the lack of a consensus theoretical framework or standardized nomenclature in stress research, it is notable that there is general agreement among researchers about what constitutes chronic stress. Conditions and circumstances that rise to the level of chronic stress include persistent situations of personal adversity and/or environmental deprivation (Compas, 1987; Kliwer, 1997; Pearlin 1983; Rutter, 1981; Wheaton, 1996). Some examples of chronic stress include enduring economic hardship, peer victimization, minority status, living in an unsafe neighborhood, and/or a prolonged illness.

Studies of children who have experienced chronic stress focus almost exclusively on mental health outcomes such as adjustment problems (Grant et al., 2003). Few studies have looked at chronic stress and physiological response or physical health outcomes in typically-developing children during middle childhood (Evans, 2003). Even fewer studies have looked at chronic stress in such children as they age from middle childhood to adolescence (Evans, 2007). As a result, little is known about these relationships. Yet, understanding how chronic stress may be associated with the development of allostatic
load in children and adolescents is both relevant and significant in terms of contributing to an overall understanding of child development and child health (CDC, 1999; Kahn et al., 1999; Rat, Botterweck, Landgraf, Hoogeveen, & Essink-Bot, 2005; Wagstaff, Bustreo, Claeson, & World Health Organization (WHO), 2004).

**Allostatic Load**

The Allostatic Load model posits that chronic stress experienced over months or years requires unremitting physiological adaptation within different systems (e.g., cardiovascular, neuroendocrine) and results in their physiological dysregulation. The sum of these changes is conceptualized as *allostatic load* (McEwen & Stellar, 1993; McEwen, 1998).

Thus, using the framework of allostatic load, we would expect that individuals who experience high levels of chronic stress will, over time, exhibit signs of physiological dysregulation consistent with this framework. And, consistent with this framework, this should be true for all individuals, including children exposed to chronic stress. However, this prediction is based on the supposition that individuals react biologically to chronic stress in the same manner and at the same magnitude. This is unlikely, as individual biological variation occurs, which is not taken into account. This supposition does not take into account the individual biological variation that occurs. Moreover, children may experience different types and magnitudes of chronic stress than adults, and this will also influence whether they exhibit physiological dysregulation. There is limited research on the presence and degree of allostatic load in typically-developing children.
Ecological Framework

Children do not develop from middle childhood to adolescence in isolation; their development occurs within different contexts (Bronfenbrenner & Morris, 1998). For the purposes of this study, the primary contexts considered are categorized as Home/Family or Extrafamilial. Chronic stressors likely vary within and across these two domains; thus, elucidating such processes may allow us to understand how context-specific chronic stress affects or does not affect the presence or magnitude of allostatic load (Evans, 2003; Morales & Guerra, 2006). Maternal sensitivity is also considered as a protective factor (Ainsworth, Blehar, Waters, & Wall, 1978). This is because prior research suggests that maternal sensitivity may protect by processes that ultimately result in children being able to develop a regulated response to stress, including chronic stress (Blair, Granger, Willoughby, & Kivlighan, 2006; Crockenberg, Leerkes, & Lekka, 2007). Understanding these processes would be of value to developmentalists, other researchers, health care providers, and policy makers.

Home/Family Stress

Six chronic stressors were considered within the Home/Family context for this study: family income, level of maternal education, maternal partner status, child’s minority status, level of household chaos, and level of maternal depressive symptoms. These chronic stressors were chosen based on both theoretical deductions and empirical evidence.
**Extrafamilial Stress**

Six Extrafamilial chronic stressors are considered in this portion of the analyses: Maternal and child reports of neighborhood safety, child’s feelings towards school, school attachment, peer victimization, and loneliness. Theoretical and empirical evidence guided the choice of these chronic stressors.

**Maternal Sensitivity**

Various protective factors need to be examined empirically as they may moderate the relationship between chronic stress in either Home/Family and/or Extrafamilial contexts and allostatic load. For instance, Evans and colleagues (Evans, King, Ting, Tesher, & Shannis, 2007) showed that the relationship between chronic stress and allostatic load was moderated by maternal sensitivity in a sample of middle school children. These findings need to be replicated.

**Contributions of the Study**

The results from this study will likely inform a larger narrative. Allostatic load is a heuristic that informs a paradigm where at-risk and pre-disease states are conceptualized on a continuous spectrum between health and disease. The presence or magnitude of allostatic load may suggest a pre-disease or disease state (McEwen & Gianaros, 2010). Thus, interventions to eliminate or reduce allostatic load may ameliorate pre-disease or disease states. However, whether this biological process model has utility for considering child and adolescent health and disease remains to be determined. This study looks at one biological-process model that may have such utility for children and adolescents.
CHAPTER II
THEORETICAL FRAMEWORK

More than 2000 years ago, the relationship between stress and illness was recognized by the Roman medical writer Aulus Cornelius Celsus who said fear and anger, and any other state of the mind may often be apt to excite the pulse (Celsus, trans. 1815). Today, it is generally accepted that a relationship exists between stress and disease, yet processes linking stress to disease have not been clear. Critical to untangling the processes underlying this relationship is defining the concept of stress.

Concept of Stress

It has been said, stress is a universal human experience. There is some agreement about the descriptive terms associated with different types of stress (i.e., acute stress, chronic stress); yet, it has remained difficult for researchers to conceptualize and define exactly what constitutes stress (Compas, 1987; Compas, 1993; Mason, 1975; McEwen, 2006; Rutter, 1981). It has been noted by various researchers from different disciplines that the term stress is either not defined or definitions of stress will differ across studies even within the same discipline (Compas, 1987; Compas, 1993; Mason, 1975; McEwen, 2006). Furthermore, when a definition is provided, it usually does not adequately reflect the associated theoretical framework (Compas, 1987). Despite these ambiguities, stress
remains a phenomenon of strong research interest (Becker, 2013; Casey, Jones, Levita, Libby, Pattwell, Ruberry, et al., 2010; Grant, Compas, Stuhlmancher, Thurm, McMahon, & Halpert, 2003).

Given the lack of consensus about the concept and definition of stress, it is not surprising that multiple theoretical frameworks have been used to examine stress. The most relevant theoretical perspectives for the proposed study fall into three broadly-defined domains: 1) stress theories, 2) systems theories and 3) biological stress frameworks. These will be briefly reviewed and discussed in terms of informativeness for the proposed study.

**Stress Theories**

Prior social science research looking at the relationship between stress and health in children and adolescents has been informed by three types of stress theories: 1) social stress theory (Holmes & Rahe, 1967), 2) the transactional model of stress and coping (Lazarus, 1966; McGrath, 1970; Moos, 1984), and 3) traditional stress theories such as the double ABC model of adjustment and adaptation (Hamilton, McCubbin, & Patterson, 1983), the resiliency model of family stress, adjustment, and adaptation (McCubbin & McCubbin, 1989), and the family stress model (Boss, 2002). Given their primacy to stress research these three stress theories are acknowledged, but because they have not been applied to analyses of physiological responses to stress or physical health outcomes associated with stress they will not be used in the present study.
Systems Theories

A system is a group of interrelated components or parts that function as a whole; systems theorists posit that any single entity (e.g., component, organelle, or individual) is part of a larger dynamic system (White & Klein, 2002). Systems theories have been applied to living systems, social systems, and other types of dynamic systems, including family systems (Miller, 1989; Steinglass, 1987). In any type of system, there is a dynamic interplay between maintaining a balance between change and stability while dealing with pressures from within and outside of the system. One of the core concepts of systems theory is morphostasis, which is conceptualized as a dynamic process by which any system tries to maintain coherence, regularity, and a sense of equilibrium while being continually faced with external and internal stressors (Speer, 1970; White & Klein, 2002). The concept of morphostasis is similar to the concept of homeostasis; the distinction between these two concepts is subtle with morphostasis referring to the stability of a system in the faces of change while homeostasis is the disposition in a system towards a state of stability.

According to a systems theory framework, an individual’s physiological response to chronic stress is a product of the entire system. Stress is conceptualized as an outside force acting on a system, and an individual’s physiological response to stress can be viewed as morphostasis or allostasis. That is, in responding to stress, morphostasis involves the process of actively maintaining physiological equilibrium while preparing to take action (e.g., the classic fight or flight response) in response to the outside force, stress. When stress is chronic, the body may ultimately adapt physiologically to be in a
more prepared state without having to make constant adjustments (McEwen & Stellar, 1993; McEwen, 1998). From a systems perspective, a change has occurred in the system and with adaptation a new level of morphostasis is reached. Over time this repetitive adaptation carries with it some physiological costs, referred to as allostatic load.

While general systems theory provides a global perspective for thinking about stress and health in children and adolescents, it does not focus on the types of stress or the environments, social or contextual, from which those stressors arose. Bioecological and ecological systems theories, with their focus on environments, better informs this aspect of the proposed research.

**Bioecological Systems Theory**

An ecological perspective posits that behaviors cannot be understood apart from their context (Lewin, 1935). The ecosystem is composed of interconnected and interdependent parts. In human development, the term ecological refers to the interrelations between individuals and the environments, social and/or contextual, in which humans are embedded (Bronfenbrenner, 2005; Bronfenbrenner & Morris, 1998). Applying Bronfenbrenner’s bioecological systems theory to the study of stress suggests conceptualizing stress as experiences embedded within different contexts (Bronfenbrenner & Morris, 1998). Moreover, the total experience of stress for a child or adolescent involves the simultaneous exposure of stress within the environment, which is further conceptualized as being composed of interconnected, but distinct systems.
Specifically, in Bronfenbrenner’s bioecological theory (1998), human development occurs within four distinct interconnected systems: the microsystem, the mesosystem, the exosystem, and the macrosystem.

A microsystem is any setting in which a developing individual has face-to-face interactions with others; microsystems are also where proximal processes occur. Proximal processes are the reciprocal interactions that occur over time, between developing individuals and their environments (e.g., other people, objects, and symbols); these processes become progressively more complex. The mesosystem includes the connections between two or more microsystems. In practical terms, what happens in one microsystem influences other microsystems. For example, the interactions between the school environment microsystem and the home environment microsystem would form a mesosystem. The exosystem refers to the social structures and policies which may influence an individual even though that individual does not participate directly in the exosystem. The macrosystem involves the broader culture, political systems, global economy and subcultures in which a child or adolescent lives; this system influences a child or adolescent through societal norms and traditions.

Of most direct importance to the health of the developing child or adolescent is the microsystem, which includes the environmental factors that directly influence the individual. The proposed study focuses on two principal microsystems for children and adolescents broadly conceptualized as: 1) the Home/Family environment and 2) the Extrafamilial environment. The Home/Family environment includes factors such as family income, maternal education level, maternal partner status, and level of home
chaos. The Extrafamilial environment includes a child’s neighborhood, school, and peers. It is within Microsystems that proximal processes take place; proximal processes include anything that goes on regularly and consistently in the life of an individual. By examining these various Microsystems for both the presence and number of chronic stressors, we can determine if there is a longer-term influence of proximal processes through physiological regulation.

In the Home/Family microsystem the presence of a chronic stressor is likely to influence different proximal processes. For instance, mothers with depressive symptoms have been shown to be more withdrawn and not as involved with their children (Gartstein, Bridgett, Dishion, & Kaufman, 2009). In turn, this behavior may lessen the quantity and quality of maternal-child interactions. Some proximal processes that may be disturbed might include family dinner, checking homework, and/or the bedtime routine. In an Extrafamilial microsystem a chronic stressor such as living in an unsafe neighborhood might result in a proximal process of a child having to stay indoors after school. In turn, this might mean having fewer peer interactions and less physical activity. Experiencing peer victimization at school might be associated with less investment by a child in school and could be evidenced by poor attendance, which in turn is likely to be associated with poorer academic outcomes. These examples of chronic stressors occurring in two different Microsystems might be associated with repetitive or sustained physiologic response to stress and over time may be associated with physiological dysfunction.
The Home/Family environment operates concurrently with the Extrafamilial environment. If a chronic stressor is present in one of these microsystems it might influence the other microsystem. The connection or interactions between two microsystems is known as the mesosystem. Consider this example: A child from a home environment with a chronic stressor of low family income might not have socially acceptable clothes to wear to school, and, as a result, the child may experience peer victimization and loneliness. Over time chronic stress occurring in either of these microsystems has the potential to influence individuals’ physiology resulting in dysfunction, which theoretically could be measured in terms of allostatic load during adolescence.

**Cumulative Risk**

Cumulative risk models provide another way to apply a systems framework to the study of stress, enabling researchers to look at the collective impact of chronic stressors. Cumulative risk models posit that the combination of risk factors is a better predictor than individual factors alone (Dawber, 1980; Rutter, 1979; Sexton, 2011). This idea draws on two concepts from systems theory: emergent properties and equifinality (van Bertanlaffy, 1968). First, an emergent property is related to a characteristic of systems called wholeness, which means there are properties that result from the dynamism of a system and these properties are not present when components of a system are considered separately (Whitechurch & Constantine, 1993). Equifinality is the concept that different paths or processes can lead to the same or similar outcomes (van Bertanlaffy, 1968). When considering how cumulative stress may impact child or adolescent physiologic
regulation, the principle of equifinality suggests that exposure to stress will be associated with an increased risk for physiological dysregulation regardless of the type of stressor or the context. Thus, no matter the type of stressor or context, an individual’s physiological dysregulation would still be represented by the same findings such as increased blood pressure or increased cortisol levels.

Prior research has shown repeatedly that for children and adolescents, it is the number of simultaneous risks that is most predictive of negative child outcomes compared to type of risk (Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Morales & Guerra, 2006; Rutter, 1979; Sameroff, Gutman, & Peck, 2003; Sanson, Oberklaid, Pedlow, & Prior, 1991; Suglia, Duarte, Chambers, & Boynton-Jarrett, 2012; Trentacosta, Hyde, Shaw, Dishion, Gardner, & Wilson, 2008). Notably, Morales and Guerra (2006) went a step beyond these prior research studies and compared context-specific stress — stress experienced in school, within the family, and in the neighborhood — against cumulative stress to find out whether one model was a better predictor of negative outcomes than the other. They showed that although stress from all three contexts was related to negative outcomes, cumulative stress, or stress experienced across contexts, was most predictive.

Systems theories provide a useful way to look at physiological responses to chronic stress during middle childhood and adolescence. This is because each of the three theories reviewed provides a different, but testable perspective of the effect of chronic stress. General systems theory provides a view of the overall impact chronic stress may have on physiological regulation. An ecological systems perspective allows
for a focus on developmental effects of chronic stress. Finally, cumulative risk models enable researchers to consider and empirically test the cumulative impact of chronic stressors on physiological regulation.

**Biological Frameworks**

At the beginning of this chapter it was noted that the relationship between stress and physiological response was recognized at least 2000 years ago. Since that time, the relationship between stress and illness has been repeatedly noted. However, it was not until the mid-1800s that one of the foundational concepts linking stress to health was put forth formally, homeostasis. Since that time biological frameworks have centered on the idea of equilibrium being central to health. That is, too much stress resulted in illness. However, the intervening processes remained elusive until the heuristic concepts of allostasis and allostatic load were introduced.

**Homeostasis**

The first formal mention of the concept of homeostasis was set forth in 1856 by Claude Bernard when he described the importance of the *milieu interieur* (internal environment) surrounding the body's cells as essential for the life of the organism (Bernard, 1856). In 1929, W. B. Cannon extended this idea and codified it by discussing the stability of physiological systems as necessary for maintaining life (Cannon, 1929; Schulkin, 2003). Today, homeostasis is understood to pertain to an organism’s making changes or adjusting body systems to match environmental demands (Sterling & Eyer, 1988). Until recently, this concept was broadly accepted and used across disciplines.
In 1946, Hans Seyle presented a stimulus-reaction model that he believed explained how stress resulted in the body having to respond for a long time resulted in physical harm and, thereby, resulted in illness (Seyle, 1946). He called this nonspecific physiological activation General Adaptation Syndrome.

**Allostasis**

In 1988, Sterling and Eyer extended the classical theory of homeostasis in a fundamental way by describing how the body adapts to challenges in order to maintain homeostasis, a process Sterling and Eyer called *allostasis* (Bernard, 1856; Cannon, 1929; Sterling & Eyer, 1988). The term literally means maintaining stability. The body maintains stability in the face of stress through moment-to-moment changes in physiological systems. Examples of allostasis provided by Sterling and Eyer (1988) included blood pressure rising in the morning when we get out of bed thereby keeping us conscious.

During a typical day, one’s body is continually responding to environmental stressors by calibrating physiological systems to maintain homeostasis. These stressors range from barely-noticeable temperature changes and ambient noise to daily hassles and interpersonal conflicts to major stressors such as accidents or other situations which may be perceived as life-threatening. According to Sterling and Eyer (1988), stressors requiring a physiological response can range from low magnitude events that are outside one’s conscious awareness to high magnitude events necessitating one’s full attention and a full blown fight or flight response. In short, any type of stressor challenges homeostasis, and the process of allostasis restores and maintains physiological stability.
In 1993, McEwen and Steller suggested that the definition of allostasis be broadened to emphasize that maintaining homeostasis is an active process. In their view, allostasis is a dynamic process involving the active adaptation to stress of different physiological systems (i.e., regulation of pH level, body temperature, and oxygen tension) in order to maintain homeostasis. Thus, the definition of allostasis was expanded to refer to the *process* of active adaptation to acute stress. This process has been judged as being entirely biologically based; however, other stress researchers highlight the fact that perceptions of stress are intimately entwined with biological response to it (Cohen, Karmake, & Mermelstein, 1983; Dufton, Konik, Colletti, Stanger, Boyer, Morrow, & Compass, 2008; Whalen et al., 2004).

To underscore that the allostatic process is one of active adaptation, consider an example of a child experiencing an acute stressor significant enough to be perceived as a threat (e.g., a loud, angry argument between parents). The response to an acute stressor involves activation of two systems: the sympathetic-adrenomedullary (SAM) system and the hypothalamic-pituitary-adrenal (HPA) axis. This activation causes the release of hormones from the adrenal glands (i.e., epinephrine & cortisol) (Gunner & Quevedo, 2007; Lupien, McEwen, Gunner, & Heim, 2009). These hormones help us adapt to stress by causing physiological changes that make possible all the features associated with the classic fight or flight response (Cannon, 1929). For instance, the release of the hormone epinephrine acts on multiple systems throughout the body: 1) increasing heart rate, 2) causing vasodilatation of the blood vessels associated with the muscles, 3) causing vasoconstriction to the skin and the gut, and 4) increasing arousal, vigilance and
narrowing attention in the HPA system (Gunner & Quevedo, 2007). The hormone cortisol acts on the metabolic system to mobilize resources for energy, the immune system to fight inflammation, and – once the threat ceases – the HPA system to help stop the stress response (Gunner & Quevedo, 2007). These short-term physiological changes in response to acute stress are considered adaptive because they enable us to respond to threats (McEwen, 2006).

**Allostatic Load**

Although everyday adaptation is a normal process, high levels of stress result in long-term repeated activation of the multiple physiological systems. When physiological systems are continually bathed in stress hormones, they must react repeatedly (e.g., increases in cortisol to mobilize energy resources). Over time these physiological systems will experience “wear and tear” (McEwen, 1998, p. 171). This wear and tear on the physiological systems (e.g., cardiovascular, metabolic) is associated with dysregulation. For instance, when the cardiovascular system is continually exposed to epinephrine, this can result in changes in the vascular tissue, which in turn can be associated with increased or high blood pressure (Lucini, Norbiato, Clerici, & Pagani, 2002; Steptoe, & Marmot, 2005). In some individuals, prolonged dysregulation of a physiological system may lead to pre-disease and disease states (Danese & McEwen, 2012; McEwen, 1998; Seeman, Rowe, McEwen, & Singer, 2001). Thus, long-term physiological stress changes are considered maladaptive and are termed allostatic load (McEwen & Stellar, 1993).
Summary

Allostasis and allostatic load are important concepts because they provide a conceptual basis for quantifying the biological effects of chronic stressors in various populations and, thus, potentially identifying and teasing apart the processes linking stress to health. Studies in adult populations show that various types of chronic stress across different contexts are associated with allostatic load; this research is only beginning for the child and adolescent populations. Much more research needs to be done to confirm whether the relationship between chronic stress during middle childhood results in allostatic load in adolescence.
CHAPTER III
LITERATURE REVIEW

Until recently, there has been little research focus on childhood stress, and what does exist has concentrated primarily on both the early developmental period and upon those children who have been exposed to a non-normative stressor; examples of such non-normative stressors include maltreatment, abuse, adverse childhood experiences and/or extreme environmental contexts such as growing up in an orphanage or war zone (CDC, 2010; AAP, 2011). As a result, there are gaps in the literature for the developmental periods of middle childhood and, to a lesser extent, adolescence with regard to the effects of exposure to chronic stress. Furthermore, almost no research has examined the relation between chronic stress in middle childhood and physiological functioning in adolescence.

Chronic stress is differentiated from other types of stress by its persistent and open-ended nature (Kliewer, 1997, Pearlin, 1983; Wheaton, 1996). Chronic stress is typically defined in terms of environmental conditions of deprivation and disadvantage or a personal condition creating a hardship (Compas, 1987; Rutter, 1981). Some examples of chronic stress include enduring economic hardship and living in an unsafe neighborhood.

In this literature review, I initially provide a brief historical overview of stress research done with children and adolescents. This provides a background for
understanding the relevance of looking at chronic stress in middle childhood. Then, some known chronic stressors are considered within the context of home and family and extrafamilial domains. Next, allostatic load as a long-term adaptation to chronic stress is reviewed. Finally, a protective factor is considered – maternal sensitivity.

**Stress**

Researchers studying stress in children and adolescents have used a range of measurement approaches: objective questionnaires, subjective questionnaires, observation, and physiological measures. Each of these approaches is described briefly in the following section.

**Objective Questionnaires**

In 1967, Holmes and Rahe published the Social Readjustment Rating Scale (SRRS). This checklist of significant life events, positive and negative, was developed for use with adults. The underlying premise is that major positive or negative life events require readjustment, which in turn predicts psychological and physical health. Using the SRRS, major negative, but not positive events were found to be related to adult psychological impairment and physical health (Ross & Mirowsky, 1979; Vinokur & Selzer, 1975; Zautra & Reich, 1983). This finding has been replicated many times; thus, even today, the SRRS remains one of the most widely used research measures for assessing stress (Hock, 1995).

Shortly after the publication of the Holmes and Rahe (1967) study, research efforts to understand how children and adolescents experience stress were started. These initial efforts mirrored what had been done with adults. In 1972, Coddington worked with
a range of professionals who worked with children and adolescents (i.e., pediatricians, teachers, and social workers) to modify the SRRS so that it could be used with children and adolescents, replacing items such as *wife starting work outside home* (Homes & Rahe, 1967, p. 216) with *beginning nursery school* (Coddington, 1972a, p. 13). Coddington tested this modified SRRS in a large population of healthy children and adolescents to gauge how much social readjustment typical children and adolescents experienced. Parents completed the SRRS for children, and adolescents were asked to complete a self-report checklist. Using these data, Coddington created an age curve of average social readjustment scores noting that there are two periods when there is a jump in the amount of social readjustment: when children started school and at the onset of puberty.

By the end of the 1970s, there were at least six different life events measures for use with an adolescent population. In general, these measures were scored by creating a cumulative score or by using an assigned value if a specific life event had occurred by a specific age (Coddington, 1972a, Johnson and McCutcheon, 1980; Monaghan et al., 1980; Yeaworth et al., 1980).

By the early 1980s, use of these various life event checklists in different populations of adolescents yielded empirical evidence for a relationship between negative life events and psychological problems such as emotional and behavioral maladjustment (Gad & Johnson, 1980; Hotaling, Atwell, & Linsky, 1978; Johnson & McCutcheon, 1980). Positive life events were not consistently associated with any of the outcome variables. Furthermore, research with adults showed evidence that daily stressors were at
least as predictive as major stressful events, and possibly more so, in terms of psychological or physiological problems (DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1983; Kanner, Coyne, Schaefer, & Lazarus, 1981; Monroe, 1983).

During the early 1980s, data continued to accumulate supporting the association between negative life events and emotional and behavioral maladjustment and physical illness (Newcomb, Huba, & Bentler, 1981). Nonetheless, because the data were primarily cross-sectional, there was no information about whether there was a causal relationship between negative life events and health. Notably, life events checklists were still undeveloped, lacked psychometric rigor, with only one exception lacked input from children and adolescents (Swearingen & Cohen, 1985), and failed to capture the entirety of adolescent experiences (Compas, Davis, & Forsythe, 1985; Newcomb, Huba, & Bentler, 1981).

In an attempt to elucidate how children and adolescents did experience stress, researchers begin interviewing children and adolescents. Lewis and colleagues (1984) interviewed fifth and sixth graders to find out what made them feel bad, nervous, or worried. This information was used to design a checklist similar to the SRRS. After pilot testing, this checklist contained 20 items, the majority of which were equivalent to daily stressors on the adult version of the SRRS (Lewis, Siegel & Lewis, 1984).

This checklist was administered to a large group of fifth graders who reported that there main sources of distress were: 1) not spending enough time with parents, 2) having parents argue in front of me, 3) turning in homework late, 4) having nothing to do, and 5) not having enough money to spend (Lewis, Siegel & Lewis, 1984). These sources of
distress were consistent with persistent role problems (e.g., being left out of a group, being pressured to get good grades) or persistent ongoing stressful processes (e.g., parents arguing). Thus, similar to the research with adolescents, children reported that daily stressors and chronic stressors were the major sources of distress.

Similarly, Compas and colleagues (Compas, Davis, & Forsythe, 1985; Compas, Davis, Forsythe, & Wagner, 1987) began developing a measure of daily stressors and major events during adolescence by asking adolescents directly what they believed were significant events in their lives. Adolescents were asked about both positive and negative events. This work was based on a cognitive-transactional model of stress (Lazarus & Folkman, 1984; McGrath, 1970; Moos, 1984), so for each major event or daily hassle adolescents were asked to respond about the desirability of the event or hassle and the impact it had on their lives.

Prospective studies of adolescents showed that major life events were not directly related to psychological symptoms (Swearingen & Cohen, 1985; Wagner, Compas, & Howell, 1986). Wagner and colleagues (1986) found evidence that major life events did predict daily stressors and, in turn, daily stressors predicted psychological symptoms, but there was no direct relationship between major events and psychological symptoms (Wagner, Compas, & Howell, 1986). Thus, chronic stressors and daily stressors were indirectly related to psychological symptoms. However, there was a relationship between psychological symptoms and later or subsequent negative life events (Burt, Cohen, & Bjorck, 1986; Swearingen & Cohen, 1985; Wagner, Compas, & Howell, 1986).
The Perceived Stress Scale (PSS) (Cohen, Karmack, & Mermelstein, 1983) is a well-known measure of stress that has been used with adolescents. Rather than looking at a checklist of specific life events or daily stressors, children or adolescents are asked to what degree do they find their lives are “unpredictable, uncontrollable and overloading” (Cohen, Karmack, & Mermelstein, 1983, p. 387). Children and adolescents who report high levels of perceived stress are at increased risk for negative outcomes such as depression (Martin, Kazarian, & Breiter, 1995) or substance abuse (Galaid, Sussman, Chou, & Willis, 2003).

Since the participant’s cognitive appraisal is integral to measuring perceived stress, participants are asked about both the source of stress and asked to provide an appraisal. This might be as simple as an open-ended question as was done in a 1985 study when adolescents were asked directly about what they believed were significant events in their lives, and, equally important, they were asked to respond about the desirability of an event and the impact it had on their lives (Compas, Davis, & Forsythe, 1985; Compas, Davis, Forsythe, & Wagner, 1987). Previous research utilizing the transactional model of stress and coping is, in part, responsible for why self-reports of stressful life experiences are now used with adolescents, in both clinical and research settings (Martin et al., 1995; Schmeelk-Cone, & Zimmerman, 2003). Adolescents’ self-reports of stressful life experiences are predictive of their own emotional and behavioral problems, as well as overall health, and abdominal pain (Compas, Howell, Phares, Williams, & Giunta, 1989; Dufton, Konik, Colletti, Stanger, Boyer, Morrow, & Compas, 2008; Thomsen, Compas, Colletti, Stanger, Boyer, & Konik, 2002; Wu & Lam, 1993).
Observational Measures

In an elementary school setting, Lewis and colleagues (Lewis, Lewis, Lorimer, & Palmer, 1977) found that for children 6-12 years of age who could see a school nurse, without having to get permission from a teacher or other school professional, there was a subgroup of children who were observed to be high utilizers. These children did not have chronic illnesses. The nurses and teachers believed these self-initiated visits were for psychological distress. In response to this observation, the researchers introduced psychological services for the children, which resulted in a 60 percent reduction in visits made to the school nurse by the high utilizers. The researchers suggest that this was indirect evidence that children who frequently visited the school nurse were likely to be experiencing emotional distress. There are other observational measures of stress in children, but these are limited to early childhood or for instances of extreme stress such as child maltreatment (Fletcher, 2003; Mash, 2008).

Other Research Approaches to Studying Stress

Following this initial work in (from 1970 to the early 1990s), stress research with children and adolescents proceeded in several directions. First, using a transactional model of stress and coping, the constructs of stress and coping were yoked together (Compas, 1993; Lazarus & Folkman, 1984). Second, research started to focus on resilience research – specifically, the successful adaptation of children and adolescents developing in adverse circumstances (e.g., low income, parent with mental illness) (Masten, 2001). A third line of research built on the initial work of looking at life events and daily stressors, but started looking at types of stressors in exceptional populations
such as academically-gifted high school students (Suldo, Shaunessy, & Hardesty, 2008), minority populations such as individuals of Navajo descent (Wadsworth, Rieckmann, Benson, & Compas, 2004). Most recently, stress research has begun to focus on physiological responses. This line of work is reviewed in the next section.

**Biological Measures**

Research examining the relationship between stress and health needs to account for the all aspects of stress response. This means accounting for responses to stress that are involuntary or outside of an individual’s conscious awareness (Connor-Smith, Compas, Wadsworth, Thomsen, & Saltzman, 2000; Davis & Whalen, 2001; Whalen, Kagan, & Cook, 2004). For these reasons and others, biological measures of stress response are now routinely used in stress research (National Institutes of Health Biomarker Definitions Working Group, 2001; Xu & Zeger, 2001). Two commonly used physiological measures are cortisol and heart rate variability.

*Cortisol.* This hormone is one of the most frequently used biomarkers in developmental research and can be tested with saliva, blood, or urine samples (Hellhammer, Wist, & Kudielka, 2009; Nater et al., 2007; van Cauter, Leproult, & Kupfer, 1996). Cortisol is a steroid hormone secreted by the adrenal glands, and its release is regulated by the hypothalamic pituitary axis (HPA) (Dickerson & Kemeny, 2004). Cortisol is involved in normal physiological function and regulation of biological systems. Cortisol levels reflect HPA axis activity and functioning. Levels of cortisol in humans vary throughout a 24-hour period with the peak level normally occurring in the early morning, usually within the first 30 to 45 minutes after waking (Kirschbaum &
Hellhammer, 1989). This peak is followed by a gradual decrease throughout the day with the lowest level occurring around midnight, and then the 24-hour cycle repeats.

Over and above this regular physiological functioning, cortisol rises in response to both acute and chronic stressors (Bremmer & Vermetten, 2001; Sapolsky, Romero, & Munck, 2000). Cortisol response to stressors is overviewed later in this chapter in the allostatic load section. However, once an acute or chronic threat stops, cortisol levels, typically, will return to baseline levels. Given this response property, cortisol is used as an index of stress across many diverse populations. For instance, a rise in cortisol levels and other changes in the daily pattern of cortisol levels occur in individuals exposed to chronic stress. In adults, exposure to chronic stress is associated with changes in the daily pattern of cortisol activity (Heim, Ehlert, & Hellhammer, 2000; Millea & Holloway, 2006; Pruessner et al., 1999). Similarly, children and adolescents exposed to chronic stressors such as poverty or maternal depression are more likely than non-exposed children and adolescents to have changes in cortisol levels (Lupien, King, Meany, & McEwen, 2000; Mannie, Harmer, & Cowen, 2007).

Also, young children who receive child care outside the home show different cortisol patterns compared to children who received home-based child care (Dettling, Gunnar, & Donzella, 1999; Watamura, Donzella, Alwin, & Gunnar, 2003). Children in center-based care show rising levels of cortisol throughout the day. In contrast, children cared for at home show a decrease in cortisol over the day, which is considered normal. This difference in cortisol patterns between these two population types was present, although less pronounced; in children who received center-based care in very high quality
centers (Dettling, Parker, Lane, Sebanc, & Gunnar, 2000; Tout, de Haan, Campbell, & Gunnar, 1998). The effect of this early chronic stress is predicted by the Allostatic Load framework and was observed by Roisman and colleagues (Roisman et al., 2009). They found that children who experienced chronic stress in the first three years of life in terms of low levels of maternal sensitivity and longer times spent in center-based child care showed effects many years later as evidenced by lower awakening cortisol levels at age 15.

*Heart rate variability (HRV).* Heart rate variability is a measure which is being looked as possible marker for stress response. HRV refers to regularity or irregularity of intervals between consecutive heart beats, which is observed on an electrocardiogram (Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). In a population of healthy individuals, HRV changes to meet the demands of different situations, including stressful situations (Appelhans & Luecken, 2006; Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). Generally, high HRV, or the ability to change cardiac functioning quickly, reflects the elasticity or functioning of the autonomic nervous system in terms of balancing parasympathetic and sympathetic input (Porges, 2001; Thayer & Lane, 2000). High HRV is also positively associated with emotional regulation in children (Eisenberg et al., 1996; Fabes, Eisenberg, Karbon, Troyer, & Switzer, 1994). In contrast, low HRV is believed to index impaired autonomic nervous system response. Low HRV, which is a decreased ability to adapt quickly to changing demands, has been found in adults with anxiety or
depressive symptoms (Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). Moreover, low HRV is linked with an increased risk of all-cause mortality and has been proposed as a marker for pre-disease or disease states (Thayer & Lane, 2007; Thayer et al., 2010).

More recently, HRV has been studied as a promising measure to assess individual differences in response to stress. In a sample of young adult medical students, HRV was measured on a day when the students were taking an exam, which was the stressor in this study, and on a control day (Lucini, Di Fede, Parati, & Pagani, 2005). These young adults exhibited signs of dysregulation on exam day as evidenced by decreased HRV and increased arterial blood pressure; the opposite – increased HRV and decreased arterial blood pressure – was observed in these same individuals on a control day. Overall these findings suggest that the cardiovascular dysregulation observed on exam day was stress related.

In a two-wave study of adolescents and young adults, HRV was measured when the participants were at rest and again during an exposure to a stressful, laboratory-based situation (Li et al., 2009) and this assessment was completed again 18 months later. As predicted, adolescents demonstrated increases in HRV in response to the stressful situation. However, these responses varied by ethnicity and gender. African Americans had a higher resting HRV compared with European Americans, and females showed larger HRV decreases than males in response to the stressful situation. These results were stable over an 18 month period. This preliminary data indicates HRV may be a useful research measure for examining adolescent response to stress.
Since formal stress research with children and adolescent populations began more than forty years ago, empirical studies show that chronic stress is predictive of negative outcomes in adolescents (Gersten, Langner, Eisenberg, & Simcha-Fagan, 1977; Wagner, Compas, & Howell, 1986). Specifically, adolescents who experience chronic stress are at risk for adjustment problems, internalizing problems, and externalizing problems (Compas, Orosan, & Grant, 1993). Likewise, children exposed to chronic stress during early or middle childhood have been shown to be at increased risk for adjustment problems during adolescence (Appleyard, Egeland, van Dulmen, & Sroufe, 2005).

**Contexts of Stress and Relation to Physiology and Health**

The primary contexts in which children are likely to experience stress are the Home/Family environment and the Extrafamilial environment, including neighborhood, school, and peer relationships. In this section, I consider some of the known chronic stressors occurring in both these contexts and what is known about the direct relation between stress and physiological health outcomes.

**Home and Family Stress**

Family socioeconomic disadvantage is widely considered to be stressful for all family members (Barrett & Turner, 2005). Factors such as single parent families, low maternal education, low family income, high home chaos, and minority status are considered chronic stressors (Bechtold, Manson, & Shore, 1994; Compas, Orosan, & Grant, 1993; Duran & Duran, 1995; Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005; Frydenberg & Lewis, 1993; Goodman, McEwen, Huang, Dolan, & Alder, 2005;...
Rutter, 1981; Siddique and D’Arcy, 1984; Walters & Simoni, 2002). Each of these chronic stressors is associated with a range of negative outcomes. These chronic stressors and the related negative outcomes are discussed in this section.

**Single Parent Status**

Single parent families’ children have lower levels of well-being (McLanahan & Sandefur, 1994). National survey data shows that compared with children from two parent households, children from single parent households have higher frequencies of emotional and behavioral problems, developmental problems, learning disabilities, and asthma (Bramlett & Blumberg, 2007).

**Low Level Of Maternal Education**

Low levels of maternal education are associated with less cognitive stimulation during early childhood outcomes, less sophisticated language development and reduced vocabulary (Bornstein et al., 2010; Currie, 2003; McLoyd, 1990, 1998). In a population of adolescents, researchers found an inverse relationship between level of parental education and a panel of biomarkers used to diagnose metabolic syndrome with lower parental education associated with an adolescent’s being at higher risk for developing metabolic syndrome (Goodman, McEwen, Huang, Dolan, & Adler, 2005). Low levels of maternal education are associated with having low wage jobs, which, in turn, is related to low family income (Jackson, Brooks-Gunn, Huang, & Glassman; Moore & Driscoll, 1997). While this clustering of stressors has the potential to confound research results
(Babbie, 2004), it is clear that all these aspects of family disadvantage play a major role in influencing child and adolescent outcomes (Bradley & Corwyn, 2002; Brody & Flor, 1997).

**Poverty and Low Family Income**

Although all these stressors are associated with negative child outcomes, low income or poverty is an independent predictor of a wide array of negative outcomes in children and adolescents. Adolescents who are raised in poverty are at risk for a range of psychological problems such as externalizing and internalizing behaviors (McLoyd, Jayaratne, Ceballo, & Borquez, 1994; McLoyd et al., 1998). Family economic hardship has been shown to be related to aggression and anxiety/depression in adolescents (Wadsworth & Compas, 2002). In addition, the duration or intensity of poverty makes a difference to child outcomes. Parent reports of the cumulative amount of economic strain the family is experiencing are associated with symptoms of depression and anxiety and antisocial behavior in children and adolescents (Conger, Matthews, & Elder, 1999; Hackman & Farah, 2009; Shonkoff, Boyce, & McEwen, 2009).

There is a clear association between poverty or low income and low academic achievement (Montgomery et al., 1993; Ramey & Ramey, 1990). Using two different analytical models, a multiplicative and a cumulative risk model, Pungello and colleagues (1996) followed children from 2nd through 7th grade and showed that low family income was an important predictor of children’s academic achievement. Duration of poverty has been found to be associated with specific cognitive outcomes. A recent study showed duration of childhood poverty was associated with deficits in working memory among...
older adolescents (Evans & Schamberg, 2009). In addition, Farah and colleagues (Farah et al., 2006) found that when compared to children from high income households, children from low income households had lower performance on working memory, cognitive control and especially language and memory tasks.

Physiological dysregulation has been observed in children from low income families. Morning cortisol levels were higher in children from low income homes compared with children from higher income homes (Lupien, King, Meany, & McEwen, 2000). These differences in stress hormone levels (i.e., morning cortisol levels) have been found in children as young as 6 years of age. Evans and Kim (2007) showed that, for young adolescents, the greater the number of years in poverty, the more elevated was overnight cortisol and the more dysregulated cardiovascular response.

In terms of physical health, in Western countries health follows a gradient pattern, meaning that there is a pattern in society of individuals at lower socioeconomic levels being less healthy than individuals from the middle class, and, in turn, middle class individuals are less healthy compared with individuals in from the upper class (Adler, Boyce, Chesney, Cohen, Folkman, Kahn, & Syme, 1994; Angell, 1993; Shonkoff, Boyce, & McEwen, 2009). In US children, this gradient effect is present from the prenatal period to preadolescence, but does vary by age for some conditions such as childhood injuries and acute respiratory illnesses (Backlund, Sorlie, & Johnson, 1996; Chen, Martin, & Matthews, 2006; Ecob & Davey-Smith, 1999). For instance, a relationship between family income and blood pressure was present in childhood but not during adolescence (Chen, Matthews, & Boyce, 2002). Adolescents, but not children, from families with
lower incomes had higher rates of respiratory conditions and were also more likely to have morbidity associated with risk-taking behaviors (Chen, Matthews, & Boyce, 2002). Additionally, children and adolescents with lower income have been shown to have higher rates of physical health problems such as clinical frequency of infectious and chronic diseases compared with children from families with higher levels of income (Duncan, 1998; Durkin et al., 1994; US Dept. of Health & Human Services, 2000). So, it was generally accepted that the relation between income and health is mediated by resource constraints that are associated with higher stress; and, specifically, this higher stress was lower socioeconomic factors. It has been generally accepted that the relation between low income and poor health is mediated by resource constraints such as low income, education, and/or social status. However, the empirical evidence supporting such relationships are inconsistent (Braverman & Barclay, 2009; Dowd & Goldman, 2006).

**Home Chaos**

For children who live in a chaotic environment, defined as one characterized by the lack of regular predictable and controlled exchanges, it has been suggested that the full effect of beneficial proximal processes on development is unlikely to occur (Bronfenbrenner & Evans, 2000). For example, if a child lives in a home characterized by disorder and high noise the child may not learn how to organize his or her school papers or develop a consistent sleep schedule; this chaos might lead to physiological dysregulation in and of itself, but it might also be an opportunity cost resulting in the disruption or displacement of other positive proximal processes. Evans and colleagues
found that adolescents from low income families had higher levels of chaos in their home environments compared with adolescents from higher income families. Furthermore, level of home chaos was found to mediate the relationship between poverty and socioemotional adjustment.

**Minority Status**

In the United States, it is recognized that there is specific stress associated with being a member of an ethnic minority group (Bechtold, Manson, & Shore, 1994; Duran & Duran, 1995; Walters & Simoni, 2002). This is consistent with the view of stress researchers who conceptualize discrimination and racism as chronic stressors (Clark, 2000; Compas, Orosan, & Grant, 1993; Taylor & Repetti, 1997). Some of the most illustrative studies of the impact of minority status on development have been conducted with African American samples. African American adolescent males show greater peripheral vascular reactivity compared with females (Wilson, Kliwer, Teasley, Plybon, & Sica, 2002). Comparison studies looking at minority status stress and outcomes have been limited to African American and European American children. However, it is clear that when comparing children and adolescents with majority status to those with minority status, the latter have a wide range of negative outcomes. For instance, in terms of academic outcomes, African American children do less well than European American
children (Gutman, Sameroff, & Eccles, 2002; Montgomery et al., 1993). Remarkably, this effect is seen even after controlling for socioeconomic background (Braveman, 2011).

In terms of health, children and adolescents with minority status are more likely than their majority-status peers to have both physiological dysregulation and poor physical health compared to their counterparts with majority status (Braverman & Barclay, 2009; Geronimus, Hicken, Keene, & Bound, 2006; Larson, Russ, Crall, & Halfon, 2008; Spencer, Thanh, & Louise, 2012). For instance, when cortisol levels were examined in preadolescent children from immigrant and non-immigrant families, morning cortisol levels were significantly higher in the children of immigrant parents (Gustafsson, Gustafsson, & Nelson, 2006). It has been shown that significantly elevated morning cortisol levels are associated with both increased stress levels and chronic conditions in adults (Grossi, Perski, Lundberg, & Soares, 2002; Powell et al., 2002; Steptoe, Cropley, Griffith, & Kirschbaum, 2000); thus it may be that children in immigrant families experience more stress than other children. Studies of physical health outcomes show a higher clinical frequency of chronic conditions among minority children and adolescents. For example, there is a high prevalence of asthma among minority children in the US (CDC, 2011), and the clinical frequency of children who are overweight is greater among black youths (6 to 17 years) compared with either Mexican American or white youths (Freedman, Kettel, Khan, Serdula, Ogden, & Dietz, 2006).
These health disparities hold throughout the lifespan (Williams & Collins, 1995). In total, these correlational data are suggestive of a possible association between chronic stress and physical health outcomes.

**Extrafamilial Stress**

Children also experience stress in environments outside the home and family context, primarily in their neighborhoods and schools and with peer relationships.

**Neighborhood**

Neighborhoods in which children do not feel safe, which are characterized by high crime rates or exposure to violence, decrease children’s sense of well-being (Attar, Guerra, & Tolan, 1994; Homel & Burns, 1989; Osofsky, 1995). When children live in neighborhoods where violence is common, they are more likely to either witness or be a victim of neighborhood violence. Specifically, children or adolescents who have witnessed or been a victim of neighborhood violence are more likely to experience psychological distress (Hill, Levermore, Twaiite, & Jones, 1996; Jenkins & Bell, 1994), anxiety (Kuther & Fisher, 1998), or even symptoms of post-traumatic stress disorder (Berman, Kurtines, Silverman, & Serafini, 1996; Berton & Stabb, 1996). Living in an unsafe neighborhood is conceptualized as a chronic stressor (McLoyd & Wilson, 1991; Rutter, 1981). Subjective and objective indicators of neighborhood danger account for a small, but significant proportion of variance in measures of well-being and adjustment as well as physical health (Bowen & Chapman, 1996).

Children and adolescents who live in neighborhoods characterized by violence are at an increased risk for exposure to violence (ETV), which is typically defined as
witnessing or experiencing neighborhood or community violence (Kliewer, 2006; Suglia, Staudenmayer, Cohen & Wright, 2010; Wilson, Kliewer, Teasley, Plybon, & Sica, 2002). ETV is a considered by researchers to be a chronic stressor (Murali & Chen, 2005), and children with ETV have been shown to have physiological dysregulation (Suglia, Staudenmayer, Cohen & Wright, 2010). For instance, in a group of African American adolescents, ETV was positively associated with increases in mean blood pressure and, in particular, males showed a higher degree of peripheral vascular reactivity (the pressure flow ratio of arteries in one's arms and legs following the introduction of a stimulus (Corretti et al., 2002)), which is a risk factor for the development of high blood pressure (Wilson, Kliewer, Teasley, Plybon, & Sica, 2002). These males also showed a higher daytime urinary epinephrine and norepinephrine levels compared to the females in this study. Kliewer (2006) showed that African American adolescents who were exposed to violence exhibited altered cortisol levels. In contrast to all these previous studies, other research with adolescents who have been exposed to violence indicates that adolescents may habituate to repeated exposures to violence (Clark, Benkert, & Flack, 2006). In this study, ETV was inversely related to task-induced changes in systolic blood pressure.

This study underscores the fact that physiological responses to chronic stress are not well understood and appear to be more complex that we may be thinking.

Other aspects of neighborhoods have been shown to be associated with physiological dysregulation in children. In a population of preadolescent children, both noise and crowding increased blood pressure and raised cortisol levels (Evans, 2006).
Children living in neighborhoods with exposure to road traffic noise or near airports or flight paths are at increased risk for poor cognitive development (Stansfield, 2005).

**School**

Stress can arise when children have problems at school, either academically or getting along with their teacher (Bond, Butler, Thomas, Carlin, Glover, Bowes, & Patton, 2007; Leung, Yeung, & Wong, 2010; Murray-Harvey, 2010; Wu & Lam, 1993). Children who report school as a negative experience tend to have a range of behavioral and academic difficulties (Blum & Rinehart, 2001; Bonny, Britto, Klostermann, Hornung, & Slap, 2000; Shochet, Dadd, Ham, & Montague, 2006). Less research has focused on the psychological outcomes related to school stress (Anderman, 2002). However, recent research shows that adolescents who self-report high levels of academic stress have been found to also have high levels of anxiety (Leung, Yeung, & Wong, 2009). In a group of adolescents starting high school, level of academic stress was related inversely to adjustment to high school (Hussain, Kumar, & Husain, 2008). Furthermore, high school students who reported high levels of stress were also more likely to engage in risk behaviors such as physical fights, having sex without birth control, using tobacco, and not eating a healthy diet (Brook, Harris, Thrall, & Woods, 2002). Thus, high levels of school-specific stress are associated with negative mental health outcomes and risk behaviors.

In addition to academic stress, level of school connectedness has been found to be an important predictor of mental health symptoms. In a group of young adolescents, level of school connectedness was predictive of depressive symptoms one year later for boys
and girls, anxiety symptoms for girls, and general functioning for boys (Shochet, Dadd, Ham, & Montague, 2006). The reverse, however, was not true. Prior mental health symptoms did not predict school connectedness one year later.

Little research has been done looking at physiological regulation in children and adolescents in a school context; the majority of this research focuses on early childhood. In one of the few studies, researchers observed a group of second graders and then compared adrenocortical responses to classroom activities (Tennes, Kreye, Avitable, & Wells, 1986). They found that students’ frequency of social engagement with peers was associated positively with both epinephrine and cortisol levels and that social approach were associated positively with epinephrine levels. In another study, high school students wore blood pressure monitors continuously while in school for two consecutive days. Those adolescents who self-reported experiencing more chronic, negative stressors compared to their peers had higher levels of systolic blood pressure (Brady & Matthews, 2006).

**Peer Relationship**

Research clearly indicates that being accepted by one’s peers is fundamental to healthy psychological development (e.g., Parker, Rubin, Price, & DeRosier, 1995). Once children reach middle childhood, they spend more time in the company of peers (Larson, Richards, Moneta, Holmbeck, & Duckett, 1996). As time spent with peers increases so does the likelihood of experiencing negative peer interactions ranging from interpersonal stress to peer victimization (Rudolph, 2002). Relationships shape a child or adolescent’s school experience (Murray-Harvey, 2010). In a group of children in grades 5 thru 9,
stressful relationships with peers was linked with lower levels of social-emotional adjustment and indirectly linked with academic performance. The converse was also true. Having good relationships with parents, teachers and peers was linked with higher levels of social-emotional adjustment and indirectly linked with academic performance.

Physiological dysregulation has been reported to be associated with peer stress. In a study of adolescents, reports of negative peer stress perceptions were linked with involuntary physiological responses including increased heart rate, racing or uncontrollable thoughts, and cognitive interference (Sontag & Garber, 2010). Other studies have shown that children who experienced repeated peer victimization appear to have greater physiological arousal including increases in skin conductance levels (Bollmer, Harris, & Milich, 2005; Hubbard, Dodge, Cillessen, Coie, & Schwartz, 2001). Children who have experienced peer victimization and who also had high levels of cortisol went on to subsequently develop depressive symptoms (Rudolph, Troop-Gordon, & Granger, 2011). Finally, Kliewer (2006) showed that peer victimization in preadolescents was associated with lower levels of cortisol for both females and males. Furthermore, cortisol awakening response differed by level of peer victimization witnessed. Females with a typical cortisol awakening response had witnessed less peer victimization and girls who had witnessed more peer victimization showed atypical cortisol awakening response patterns.

There is little research about the relationship between stress from peer relationships and physical health findings. Notably, however, children and adolescents
with health problems such as diabetes, obesity, short stature, or other health problems are more likely to report having experienced peer victimization compared with child and adolescents without health problems (Storch & Ledley, 2005).

**Constellations of Stressors**

Chronic stressors do not typically occur in isolation from one another. This phenomenon has been noted by researchers across disciplines who have reported, that chronic stressors co-occur for some individuals and within some families (e.g., many minority families also experience low income) (Conger et al., 1990; Dawber, 1980; Mair, Cutchin, & Peek, 2011; Repetti, Taylor, & Seeman, 2002). And, it is the presence of multiple, simultaneous chronic stressors that has been shown to be associated with negative outcomes in children and adolescents (Rutter, 1979; Sameroff, Gutman, & Peck, 2003; Suglia, Duarte, Chambers, & Boynton-Jarrett, 2012; Trentacosta, Hyde, Shaw, Dishion, Gardner, & Wilson, 2008). Research using a cumulative risk model shows that the greater the number of chronic stressors present from early to middle childhood, the worse the outcome in terms of adolescent externalizing and internalizing behaviors (Appleyard, Egeland, van Dulmen, & Sroufe, 2005). Stressors rarely occur in isolation, but rather co-vary both within and across contexts (Sameroff, Seifer, & Bartko, 1997). For this reason, many investigators studying stress have developed indices of stress that are cumulative in terms of considering the overall number of stressors experienced by an individual rather than studying the effects of specific stressors (Morales & Guerra, 2006; Rutter, 1979; Suglia, Duarte, Chambers, & Boynton-Jarrett, 2012).
Stress and Allostatic Load

Allostatic Load, or multisystem physiological dysregulation, is posited to have several causes, one of which is exposure to chronic stress (McEwen, 1998; McEwen & Stellar, 1993; McEwen, & Tucker, 2011). Consider an example of a child experiencing a chronic stressor significant enough to be perceived as a threat (e.g., daily loud, angry arguments between parents). In this context, a child would have repeated exposure to stress hormones, and over time it is postulated that physiological dysregulation may result. For instance, continued exposure to cortisol is known to affect functioning of the HPA system (Lupien, McEwen, Gunnar, & Heim, 2009). In turn, long-term dysregulation of the HPA axis is associated with a wide range of health outcomes including depression, diabetes, obesity, high blood pressure, and memory loss and other cognitive problems (McEwen & Naylor, 2004). Notice that this is a two-step process: First, chronic stress results in the dysregulation of physiological systems; secondly, prolonged dysregulation can lead to health problems.

The exact mechanism involved and the physiological systems that play key roles in this process are not yet agreed upon by all investigators, and therefore issues of measurement continue to be discussed. In the next section I describe several approaches to the measurement of allostatic load.

Measuring Allostatic Load

Allostatic load is an imperfect measure of dysregulation across multiple physiological systems over time (McEwen & Stellar, 1993; Danese & McEwen, 2012). There is no one single set of measurements in terms of both physiological systems
assessed and biological markers that constitutes an index of allostatic load accepted by all researchers. One defining feature is that it includes a range of physiological systems whose dysregulation is known to be associated with disease risk (McEwen, 1998; Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). Different investigators therefore use different indices of allostatic load. For example, in one study only two systems (i.e., HPA and metabolic systems) were examined (Buss, Davis, & Kiel, 2011) while in another study five physiological systems were assessed (e.g., cardiovascular, HPA, immune, metabolic, and neuroendocrine systems (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997).

Researchers have used different physiological systems to construct their indices of Allostatic Load. In the first empirical test of test of the Allostatic Load framework, Seeman and colleagues (1997) assessed four physiological systems using 10 biological markers: 1) cardiovascular system (i.e., systolic and diastolic blood pressure) 2) HPA (i.e., cortisol and dehydroepiandrosterone sulfate (DHEA-S)); 3) metabolic system (i.e., waist/hip ratio, total cholesterol / HDL ratio, glycosylated hemoglobin), 4) neuroendocrine system (i.e., urinary norepinephrine and epinephrine). Other researchers have used as few as three overall biological markers (i.e., cortisol, sleep and birth weight) (Buss, Davis, & Kiel, 2011) and one study of children used six biological measures across four systems (i.e., systolic blood pressure, diastolic blood pressure, overnight urinary neuroendocrine measures of cortisol, epinephrine, norepinephrine, and body mass index (BMI) to create an index of allostatic load (Evans, 2003; Evans, Pilyoung, Ting, Tesher, & Shannis, 2007).
Analysis of the physiological systems is done through the use of multiple biological markers such as blood pressure to assess cardiovascular risk; cortisol to assess functioning of the HPA axis; and, glucose, insulin and lipid profiles to assess metabolic functioning (NIH Biomarker Definitions Working Group, 2001; Seeman, Gruenewald, Karlamangla, & Liu, 2010). The use of multiple biomarkers allows for assessment of dysregulation both within and across physiological systems.

Despite the differences in measurement, allostatic load is calculated as a cumulative score of physiological dysregulation (Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010; Seeman, Rowe, McEwen, & Singer, 1998). Specifically, each biological marker to be used in the index is evaluated for the presence or absence of dysregulation. Researchers use different analytical methods to determine whether a physiological systems is dysregulated (e.g., clinical cutoffs, highest quartile, one standard deviation above or below the mean) (Singer, Ryff, & Seeman, 2004). A score of 1 is assigned to each dysregulated marker and then the scores are summed resulting in a continuous score representing allostatic load (Seeman, Rowe, McEwen, & Singer, 1998). The allostatic load score is similar in concept to risk indices that cumulate across various categories of demographic or psychosocial risk (Babbie, 2004). The rationale for the use of risk indices is that the accumulated effect of multiple categories of risk, which often co-occur, is more important to children’s development than the individual risks themselves (Morales & Guerra, 2006). A similar rationale holds for allostatic load, in that the assumption of researchers is that stress is associated with dysregulation across multiple systems. This finding in itself has utility for researchers as
they can quantify the biological effects in the form of physiological dysregulation from various stressors and contexts (Wade, 2008). However, it is the accumulation of dysregulation in multiple systems which may ultimately lead to negative health outcomes (McEwen, 1998; McEwen & Stellar, 1993; Seeman, Rowe, McEwen, & Singer, 2001).

**Allostatic Load and Health Outcomes**

Levels of allostatic load have been shown to be associated with health outcomes in adult populations with high levels associated with poor outcomes and low levels associated with good outcomes. In a cross-sectional multiethnic national sample, African Americans were found to have a both a higher clinical frequency of peripheral vascular disease and a higher allostatic load compared with Mexican and White Americans; the researchers suggested that co-occurrence of the peripheral vascular disease and higher allostatic load may be connected (Nelson, Reiber, Kohler, & Boyko, 2007). In a separate cross-sectional study, increased levels of allostatic load and of many of the individual biomarkers were associated with higher probabilities of health outcomes such as ischemic heart (Sabbah, Watt, Sheiham, & Tsakos, 2008). Seeman and colleagues (1997) studied an elderly sample Seeman and colleagues (1997) looked at the relation between level of allostatic load and four health outcomes: 1) mortality from any cause, 2) cardiovascular disease, 3) cognitive performance and 4) physical performance. High levels of allostatic load were linked both concurrently (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997) and in a 7-year follow-up (Seeman, Rowe, McEwen, & Singer, 2001), to all 4 health outcomes.
**Stress and Allostatic Load in Adults**

Most of the research on allostatic load has been conducted on adult samples. This research is briefly reviewed in this section.

**Family Income**

Income, education, social status and other demographic factors are most powerful and consistent predictors of health outcomes. The higher one’s social status – whether measured by income, educational attainment, or occupational rank – the better one’s health (Adler, Boyce, Chesney, Folkman, & Syme, 1993; Braverman & Barclay, 2009). Braverman and Barclay (2009) hypothesize that the added stressors and the resource constraints associated with income, education or social status disadvantage contribute to poor health. This idea has been adopted and slightly re-conceptualized by those researchers examining allostatic load; individuals at socioeconomic disadvantage are considered to experience a faster accumulation of allostatic load (Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010).

Prior research in different populations of adults demonstrates that individuals with the lowest levels of education had significantly greater allostatic load scores compared with those with the highest level of education (Kubzansky & Sparrow, 1999; Hawkley, Lavelle, Bernton, & Cacioppo, 2011). Dowd and Goldman (2006) found a similar effect, but it was gender specific, with increased education associated with a lower overall index of allostatic load in women, but not for men.

Seeman and colleagues (Seeman, Merkin, Crimmins, Koretz, Charette, & Karlamangla, 2008) showed evidence for a main effects relationship between
socioeconomic status and allostatic load indexed by 9 biomarkers representing the cardiovascular, HPA, metabolic, and immune systems. Using both indices of education and income, consistent patterns of increased prevalence of allostatic load were found for each biomarker, for each of the physiological systems, and for overall composite score for allostatic load. This gradient pattern was seen in all ethnic groups. This study shows that individuals at lower levels of income, education, and social status show greater physiological dysregulation.

Over and above income and education, minority status is an independent predictor of health, with individuals of minority status being less healthy compared with individuals with non-minority status (Geronimus, Hicken, Keene, & Bound, 2006). This phenomenon appears to be particularly true as individuals’ age (Geronimus, Bound, Waidmann, Hillemeier, & Burns, 1996), which Geronimus (1992) explains with the “weathering” hypothesis. This hypothesis states that Blacks experience early health deterioration as a consequence of the cumulative impact of repeated experience with social or economic adversity and political marginalization. Geronimus used allostatic load to test this hypothesis and found that the mean allostatic load score was significantly higher for blacks compared to whites in all five age groups tested. Also, although there were small racial differences for individuals in the younger age groups (i.e., late teens and early 20’s), racial differences for allostatic loads widened quickly and were the greatest for individuals between the ages of 35 and 64. Black women were most likely to have the
highest allostatic load. Other researchers have found comparable evidence for increased allostatic load among individuals of minority status compared with those of majority status (Seeman, Merkin, Crimmins, Koretz, Charette, & Karlamangla, 2008).

**Environmental Stress**

Environmental characteristics may affect residents indirectly through chronic stress. Some collective neighborhood characteristics that have been studied in adult populations in relation to allostatic load include: amount of segregation, poor physical infrastructure, environmental hazards, resident overcrowding, and noise pollution.

Neighborhood segregation by race and ethnicity or by income is a known stressor. Bellatreche and colleagues (2011) demonstrated that over and above other individual characteristics, segregation was a significant predictor of allostatic load. Specifically, higher levels of segregation were associated with both total allostatic load and the subscale for inflammatory biomarkers (Bellatreche, Finch, Do, Bird, & Beck, 2011). In a separate study, neighborhood affluence was associated with allostatic load (Kim, Morenoff & House, 2011). Individuals living in more affluent neighborhoods had lower total allostatic loads. Other aspects of the environment, such as deteriorating built environment or living near a perceived environmental hazard, have been theorized to be associated with allostatic load (Simandan, 2010). In a cross-sectional study of adults, Mair and colleagues (Mair, Cutchin & Peek, 2011) used 16 biomarkers across 4 physiological systems to assess the relationship between allostatic load and chronic stress, including place-based stressors such as length of residency and proximity to environmental hazard (i.e., a large complex of petrochemical plants located near the city),
and whether this relationship was moderated by living in a place with significant environmental risks. Findings indicated that long-term residence in south western city was associated with a significantly higher cardiovascular allostatic subscale score, but only for females. These researchers raise the idea of “overlapping disadvantages” (Mair, Cutchin & Peek, 2011, p. 985) or “double jeopardy of chronic stressors and environmental hazard exposures” (Morello-Frosch & Shenassa, 2006, p. 1150). An example of this is an individual with minority status who has experienced prolonged exposure by living near an environmental hazard. Thus, it was not simply living near an environmental hazard that influenced allostatic load, but it was also other factors. These other factors act as overlapping disadvantages and also in ways we do not yet fully appreciate which bring about the circumstances making it more likely or result in an individual with minority status living in closer proximity to an environmental hazard.

**Allostatic Load in Children and Adolescents**

Developmental research looking at the relationship between stress and health in children and adolescents has progressed from using single biomarkers known to be associated with stress (e.g., cortisol, blood pressure) to a theoretically-derived profile of biomarkers. Only a few studies to date have examined allostatic load in children or adolescents (Evans, 2003; Evans, Pilyoung, Ting, Tesher, & Shannis, 2007; Johnston-Brooks, Lewis, Evans, & Whalen, 1998; Worthman & Panter-Brick, 2008). These are briefly reviewed in the following section.

One of the first studies looking at allostatic load in children examined the relationship between chronic stress and poor health in a population of school-aged boys
(Johnston-Brooks, Lewis, Evans, & Whalen, 1998). Specifically, the chronic stressor was household crowding, defined as the number of people per rooms in a household, the measure of allostatic load was cardiovascular reactivity (i.e., systolic blood pressure, diastolic blood pressure, and heart rate reactivity), and the health measure was school absences due to illness. Results suggested that cardiovascular reactivity mediated the relationship between chronic stress (i.e., household crowding) and to ill health (i.e., school absences). Essentially, this study provides some initial evidence supporting the hypothesis that chronic stress results in physiological arousal, which results in decreased health in school-aged boys.

In 2003, Evans showed that increased cumulative risk across three different domains 1) physical (i.e., crowding, noise, and substandard housing), 2) psychosocial (i.e., exposure to violence, family turmoil, and child-family separation), and 3) personal characteristics (i.e., poverty, single parenthood, and maternal high school dropout status) were positively associated with allostatic load in a sample of rural-dwelling European American young adolescents. In this study, allostatic load included systolic blood pressure, diastolic blood pressure, three neuroendocrine hormones, and body mass index. The cumulative risk score was positively associated with all of the biomarkers with the exception of diastolic blood pressure. Thus, there is a positive association between cumulative risk and allostatic load in adolescents. Additionally, cumulative risk was positively associated with each of the three allostatic subscales cardiovascular, neuroendocrine and, body mass index.
Evans and Kim (2007) conducted a two-wave study of rural-dwelling, European American adolescents, half of whom lived below the federal poverty level, and examined the relationship between duration of years living in poverty, cumulative risk exposure, and physiological stress. A cumulative risk index included the six factors, 1) physical (i.e., crowding, noise, and substandard housing) and 2) psychosocial (i.e., exposure to violence, family turmoil, and child-family separation). The number of years living in poverty (birth up to age 13) was positively associated with the cumulative risk index. And, duration of time living in poverty was positively related to both elevated levels of overnight cortisol and the more dysregulated (muted) the CVR, which are indicative of allostatic load.

Building on prior research (Evans, 2003; Evans & Kim, 2007) and using the same data set of rural-dwelling, European American adolescents (wave 1– age 9, wave 2 – age 13) (Evans, Kim, Ting, Tesher, & Shannis, 2007), looked at the relationships between cumulative chronic stressors and Allostatic Load and whether this relationship was moderate by maternal responsiveness. Cumulative chronic stressors included three different domains 1) physical (i.e., crowding, noise, and substandard housing), 2) psychosocial (i.e., exposure to violence, family turmoil, and child-family separation), and 3) personal characteristics (i.e., poverty, single parenthood, and maternal high school dropout status). Allostatic load was comprised of 6 biomarkers (i.e., overnight urinary cortisol, epinephrine, and norepinephrine; systolic blood pressure, diastolic blood pressure, and body mass index). There was not a main effect for cumulative chronic stress and allostatic load. However, there was an interaction effect for maternal
responsiveness and cumulative risk on allostatic load. Specifically, as cumulative risk increased so did allostatic load, but only for those children whose mothers exhibited low levels of maternal sensitivity.

Researchers are also looking at the relationship between chronic stress and an adapted version of allostatic load that includes markers associated with the risk for metabolic syndrome and cardiovascular disease. For instance, in a population of African American and European American adolescents, researchers found an inverse relationship between level of parental education and a panel of biomarkers used to diagnose metabolic syndrome (e.g., i.e., cortisol, plasma insulin, glucose, insulin resistance, glycosylated hemoglobin, fibrinogen, lipids, waist circumference, and body mass index). Lower parental education was associated with an adolescent being at higher risk for developing metabolic syndrome (Goodman, McEwen, Huang, Dolan, & Alder, 2005). More recently, a group of healthy adolescent females were followed for 2 years to examine the relation between their social interactions and biomarkers used to assess risk for metabolic disorder (i.e., systolic blood pressure, diastolic blood pressure, high density lipid protein, triglycerides, fasting blood glucose, and waist circumference) (Ross, Martin, Chen, & Miller, 2011). They found that both the number and intensity of negative social interactions were associated with a risk trajectory for the development of metabolic syndrome. Females with fewer or less intense negative encounters exhibited a decline in their composite metabolic syndrome risk score. Notably, positive encounters did not influence or moderate the metabolic syndrome risk trajectories.
Finally, in a sample of non-western children, Worthman and Panter-Brick (2008) compared Allostatic Load in Nepali boys between the ages of 10 and 14 years from four different social groups: 1) homeless street children in an urban area, 2) urban squatters, 3) urban middle class, and 4) villagers. Levels of allostatic load differed significantly across all four groups, which the researchers believed reflected the different ecological stressors faced by each group. Urban boys showed the lowest total allostatic load presumably meaning they were the least stressed. However, the urban boys did show elevated HPA axis activity, which was equivalent to that of the homeless boys; the authors speculated that this was likely due to the stress of schoolwork (Panter-Brick & Pollard, 1999). Squatter boys showed the next lowest total allostatic load, followed by the homeless boys and the village boys, respectively. This study showed that different contexts – even so-called good contexts – can be associated with chronic stress and subsequent physiologic dysregulation as indexed by allostatic load.

**Maternal Sensitivity as a Protective Factor**

Maternal sensitivity includes a collection of behaviors ranging from recognizing their child’s emotional state to responding in a timely and developmentally-appropriate manner that gives primacy to a child’s needs over one’s own (Ainsworth, Blehar, Waters, & Wall, 1978). Mothers displaying high levels of maternal sensitivity respond to their child with warm and affectionate behaviors, helping a child modulate both positive and negative states of emotional arousal (Davidov & Grusec, 2006; Seifer & Schiller, 1995). When an infant displays positive emotions, sensitive mothers will respond by matching that emotional state and responding in kind (Lohous, Keller, Ball, Voeker, & Elben,
Likewise, if an infant is frustrated or upset, sensitive mothers will respond by soothing and reassuring the infant while relieving the source of distress. High levels of maternal sensitivity to child needs is associated with a range of positive infant and toddler child outcomes, including emotion and behavior regulation, achievement of language milestones, and cognitive development (Bornstein & Baumwell, 2001; Crockenberg & Leerkes, 2004; Farah et al., 2008; Seifer & Schiller, 1995; Tamis- LeMonda, Bornstein, & Baumwell, 2001).

Maternal sensitivity is viewed as critical for infant and toddler development, and for this reason most of the research has focused on these age groups. Throughout childhood, however, maternal sensitivity is associated with positive outcomes. For example, young children of sensitive mothers show high levels of cooperation (Denham, Workman, Cole, Weissbord, Kendziora, Zahn-Waxler, 2000). In a population of preadolescents, high levels of maternal sensitivity were found to be associated with lower levels of anxious behaviors (McCabe & Clark, 1999). Furthermore, maternal sensitivity in childhood and adolescence has been shown to partially buffer the negative effects of difficult child or adolescent temperament on social development in adolescence (Jaffari-Bimmel, Juffer, van Jzendoorn, Bakhmers- Kranenburg, & Mooijaart, 2006). Finally, in terms of physical health in a population of first graders, high levels of maternal sensitivity were associated with a lower risk of being overweight (Rhee, Lumeng, Appugliese, Kaciroti, & Bradley, 2006).

Maternal sensitivity appears to provide broad protective effects on a variety of outcomes including social outcomes, as well as specific effects on different systems such
as executive functioning (Bernier et al., 2010; Hane, Cheah, Rubin, & Fox, 2008).

Maternal sensitivity appears to be protective in contexts or environments associated with chronic stress (Blair et al., 2008). For instance, Evans and colleagues (2007) found that the relationship between cumulative risk (e.g., low family income or poverty, overcrowding and noise, negative life events) and allostatic load was moderated by the level of mothers’ responsiveness. That is, allostatic load increased in relation to cumulative risk in a positive linear manner, yet only for those children whose mothers displayed low levels of maternal sensitivity.

Maternal sensitivity is believed to be protective, in part, because it ultimately results in children being able to develop a regulated response to stress (Blair, Granger, Willoughby, & Kivlighan, 2006; Crockenberg & Leerkes, 2004; Francis & Meaney, 1999). First, maternal sensitivity appears to be associated with helping a child develop emotional self-regulation abilities (Crockenberg, Leerkes, & Lekka, 2007; Eisenberg & Norris, 2002; Fist et al., 2004; Goldsmith & Davidson, 2004; Gunnar & Quevedo, 2007). Emotional self-regulation reflects an overall healthy functioning of the HPA axis (Coe & Lubach, 2003; Gunnar & Quevedo, 2007). In turn, this allows for a more regulated stress response. A regulated stress response would be characterized by low baseline cortisol level, a high surge in response to a moderate acute stressor, and, when the stress is over, the response is turned off (Kirschbaum & Hellhammer, 1989; McEwen, 2006); this is comparable to a regulated stress response observed in animal models. Regulated stress
responses in children have been shown to be associated with higher levels of both cognitive and social competence (Blair, Granger, & Razza, 2005; Davis, Bruce, & Gunnar, 2002).

Animal models provide insight into how maternal sensitivity might moderate the stress response. In rodents, the offspring of high-grooming mothers have a greater amount of glucocorticoid receptors in the brain regions (e.g., hippocampus) associated with the negative feedback system through which the HPA axis is regulated (Meaney & Szyf, 2005). These same offspring show a better response to stress as evidenced by a lower elevation in cortisol. Together, these two findings suggest that early maternal behavior was likely associated with the development of a more regulated stress response.

**Summary**

To date, the literature examining stress in childhood and adolescence has focused on mental health and behavioral outcomes. Current research with adults suggests an important outcome of stress is physiological dysregulation, characterized as allostatic load. In turn, allostatic load appears to be linked with physical health. Although research in this area with children is just beginning, there are indications that the process of dysregulation in response to stress may begin early in life. There is also some evidence that maternal sensitivity may be a protective factor against the physiological response to stress, in that children who receive sensitive maternal care early in life are better able to regulate their physiological response to negative emotions. This study will add
to the literature on stress in childhood by examining the relation between stress in
Home/Family and Extrafamilial contexts during middle childhood and Allostatic Load in
adolescence.

**Research Questions and Hypotheses**

The study had three primary aims: 1) Investigate the relationship between chronic
stress during middle childhood and allostatic load in adolescence; 2) Investigate whether
the context in which chronic stress is experienced makes a difference in adolescent
allostatic load; and 3) Determine whether maternal sensitivity moderates the relation
between stress in middle childhood and adolescent allostatic load. The specific research
questions and hypotheses to be tested are detailed below.

1. What is the relation between chronic stress during middle childhood and allostatic
load in adolescence?

   H.1. The level of chronic stress experienced during middle childhood is associated
   positively with allostatic load in adolescence.

2. Is stress experienced within the home and family context more predictive of allostatic
load in adolescence than stress experienced in the extrafamilial context?

   H.2.1 Chronic stress in the home and family context is more highly related to
   allostatic load than chronic stress in other contexts.

3. Is the relationship between chronic stress during middle childhood and adolescent
allostatic load moderated by levels of maternal sensitivity? If so, does this relationship
hold across the two contexts, Home/Family and Extrafamilial?
H.3.1 When the level of maternal sensitivity is low the relationship between chronic stress in middle childhood and adolescent allostatic load will be positive. When the level of maternal sensitivity is high we would expect the same effect, but the relationship between chronic stress in middle childhood and adolescent allostatic load will be weakened.

H.3.2. In general, when maternal sensitivity is low the relationship between chronic stress during middle childhood and adolescent allostatic load will be positive. It is predicted that this relationship would hold across the two different contexts (i.e., Home/Family and Extr familial).
CHAPTER IV

METHOD

Participants

This secondary data analysis is based on archival data from a major study cohort of healthy children followed from birth through age 15 years, which is the National Institute of Child Health and Human Development (NICHD) Study of Early Childcare and Youth Development (SECCYD). The SECCYD is one the most comprehensive study of children and the many environments in which they develop and provided longitudinal data about children’s health, development and behavior. More than 1,300 children within their families were followed from birth through age 15 years. Data were collected in four phases of development: infancy, early childhood, middle childhood and middle adolescence.

Participants were recruited throughout 1991 from hospitals in 10 locations across the United States (Little Rock, AK; Irvine, CA; Lawrence, KS; Boston, MA; Philadelphia, PA; Pittsburgh, PA; Charlottesville, VA; Morganton, NC; Seattle WA; and Madison, WI). Women giving birth in selected hospitals were screened during 24-hour recruitment windows, and were excluded from the sample if (a) the mother was under 18, (b) the mother was unable to speak English, (c) the family planned to move, (d) the mother delivered multiple births, (e) the child was hospitalized for more than 7 days following birth or had obvious disabilities, (f) the family lived too far away or in an
unsafe neighborhood, or (g) the mother had a known or acknowledged substance-abuse problem. Of the 8,986 mothers visited during the initial sampling periods, 5,265 met the eligibility requirements and agreed to be contacted upon return home from the hospital. From the eligible sample, 3,015 mothers were selected and contacted via phone using a random-sampling technique that was employed in order to identify participants that represented the economic, educational, and ethnic diversity of the specific collection site. A total of 1,364 mothers were enrolled in the study at a home visit when infants were 1 month old. Detailed information about this original cohort is presented elsewhere (National Institute of Child Health and Development (NICHD), 2012, Retrieved October 3, 2013). However, this secondary data analysis was restricted to those participants of the original SECCYD data set for whom full biological data at age 15 were available (N = 699).

This secondary data analysis was restricted to only data from Phases III and IV. Specifically, all the predictor data was from Phase III and all the outcome data was from Phase IV. Additionally, the outcome data for this study are from Phase IV and this secondary data analysis was restricted to those research participations of the original SECCYD data set for whom full biological data at age 15 years was available (n=699). Thus, for attrition analysis this subsample of 699 research participants was compared with research participants from the phase IV dataset who did not have complete biomarker data (n = 665). A Chi-square test for independence indicated a significant association between sex of the research participants and participation in this study, $X^2 (1, n = 1364) = 7.71, p = .006, \phi = .09$. The groups did not differ in terms of their minority
status (24.8% vs. 21.9% minority status), $X^2 (1, n = 1364) = .88, p = .35, \phi = -.03$.

Families of participants in this study had significantly lower mean income-to-needs ratio compared to families who did not participate in this study, $M = .23, SD = .42$ vs $M = .26, SD = .44; t (931) = 3.67, p = .00$, two-tailed. The participants in this analysis sample, compared with those who were not, also did not differ significantly on the following variables: 1) maternal depressive symptoms $M = 9.04, SD = 7.70$ vs $M = 8.82, SD = 7.63; t (931) = -.38, p = .70$, two-tailed; 2) mean maternal sensitivity $M = 16.45, SD = 2.27$ vs $M = 16.31, SD = 2.27; t (931) = -.48, p = .43$, two-tailed; and 3) maternal education $M = 14.41, SD = 2.40$ vs $M = 14.59, SD = 2.58; t (931) = .95, p = .12$, two-tailed.

**Procedure**

For this study data were collected during home and laboratory visits conducted at the age of one month, in third, fifth, and sixth grades, and at age 15. Child gender was reported at the 1 month home visit with the mother. At this and other subsequent data collection points, Home/Family data obtained included demographic information, including income, partner and employment status, as well as maternal report of depressive symptoms. For the Extrafamilial data, children reported or were interviewed about their perceptions of relevant issues such as neighborhood safety; feelings about school and school attachment; level of loneliness; and perceived peer victimization.

Maternal sensitivity was assessed during videotaped mother-child interaction during third and fifth grades. Biomarker data used for creating the Allostatic Load index was collected at the last data collection point in this study, which was at age 15. Specifically, blood pressure, waist-to-hip ratio, skinfold measurements, and body mass index (BMI)
were collected at the last laboratory visit. Salivary cortisol was collected by the adolescent and parent(s) at their home for three consecutive days during the last year of the study.

**Measures**

**Allostatic Load**

A single index for allostatic load was created by combining six biomarkers collected at age 15. Specifically, individual scores for allostatic load were calculated by summing the number of indicators (on which criterion for an *a priori* risk is met). In this study, these measures included 1) systolic blood pressure, 2) diastolic blood pressure, 3) waist-hip ratio, 4) skinfold measurement, 5) BMI, and 6) awakening salivary cortisol.

For each of these 6 biomarkers research participants were classified into quartiles based on the distribution of baseline scores (Seeman et al., 1997). An allostatic load score was created by summing the number of parameters for which the participants fell into the highest risk quartile. Given that there were 6 biomarkers, an individual’s allostatic load score could range from 0 to 6. This approach was based on the idea that adolescents in the top quartile were relatively more dysregulated than others in the sample (Seeman, McEwen, Rowe, & Singer, 2001).

**Blood Pressure**

Research participants’ blood pressure readings, including systolic and diastolic, were taken at age 15 years (JNC7, 2004). These blood pressure measurements were obtained in a standardized fashion by trained and certified site personnel. Prior to any measurements, each adolescent had a 2-minute rest period in the examining chair to allow
his/her blood pressure to get closer to a resting value. Five blood pressure and pulse rate readings were then automatically taken at 1 minute intervals. Readings were taken from the right arm with the adolescent seated using a DINAMAP Pro 100 model from GE Healthcare. The last three readings were averaged.

**Waist-Hip-Ratio**

Waist-hip-ratio is considered to be an index of adipose tissue deposition thought to be influenced by increased glucocorticoid activity. Waist and hip measurements were taken with the research participant standing straight with feet together. Using a 1.5 meter flexible plastic anthropometric tape applied without pressure, 2-measurements of the waist and hip were taken. As long as both waist measurements were within 1.5 cm, both measurements were acceptable and used to calculate an average waist circumference. If the two waist measurements differed by greater than 1.5 cm a third measurement was taken. Then, if the last two measurements were within 1.5 cm those last two values were averaged. If the last two measurements were not within 1.5 cm of each other than the average of the two closest values was taken. This same procedure was used for hip measurements. The waist-hip-ratio was calculated by dividing the waist measurement by the hip measurement.

**Skinfold Measurements**

Both triceps and subscapular skinfold measurements are known indicators of general adiposity, which is a known risk factor for both diabetes and cardiovascular disease. Whenever possible skinfold measurements were taken on the right side of the body. Lange Skinfold Calipers manufactured by Cambridge (Maryland) Scientific
Industries, Inc., were used for all skinfold measurements. Three measures each were obtained from each of the two sites. Of the three measurement values, if the first two values were identical, that value was used. If the last two measurements were identical, then that value was used. Otherwise the closest values were averaged. Total Skinfold Measurement was computed as the sum of both skinfold measurements.

**Body Mass Index (BMI)**

BMI is an indirect measure of an individual’s body fat; that has been shown to be a reliable tool to screen for underweight, healthy weight, overweight, and obesity (CDC, 2009; Jackson et al., 2002; Kuczmarski et al., 2002; WHO, 2010). At age 15, height and weight measurements were made at the lab visit. Height was measured using a standardized procedure with children standing with shoes off, feet together and their backs to a calibrated 7-foot measuring stick fastened to a wall. Weight was measured following a standardized procedure using a physician’s 2-beam scale (CDC, 2009). A ratio was calculated using a standard formula: an individual’s weight in kilograms (kg) is divided by their height in meters (m) squared, BMI = kg/m² (CDC, 2009).

**Cortisol**

Salivary cortisol is considered to be a measure of HPA axis function. In the present study, cortisol was collected upon morning awakening for three consecutive days and sent for analysis (i.e., cortisol assay). A salivette (Sarstedt, Numbrecht, Germany) was used; adolescents and their parents were trained to carry out this procedure and instructed about the importance of carefully following the instructions. Salivary samples were shipped on dry ice to *Salimetrics* lab (State College, PA, USA) for the cortisol
assay. All samples were tested using a highly sensitive enzyme immune assay specifically designed for use with saliva (Roisman et al., 2009; Salimetrics, 2012). Cortisol values were expressed in terms of μg/dl. An average cortisol μg/dl value was calculated from the 3 consecutive, morning awakening samples which were obtained from the data collection.

**Stress**

Dichotomous variables were created for each chronic stressor with a 1 indicating that the stressor was present and 0 indicating that the stressor was not present in a child’s life. Chronic stressor data included both categorical data (e.g., single parent status) and continuous data (e.g., neighborhood safety, peer victimization). The categorical data were dichotomized as described below. For continuous data, stressor dichotomization was done by 1) creating a distribution of the summary scores for each measure and 2) assigning a score of 1 for each child whose individual score equaled or fell within the top or bottom twenty-fifth percentile of the distribution, depending upon the measure. The analysis sample in the present report comprises 699 of the 1,364 participants, due to missing data or attrition. Of these 699 individuals, 17 were missing Home Chaos evaluations, six were missing mean income-to-needs ratio data, one was missing partner status data, and one was missing maternal depressive data.

A total stress score was created within each context by summing the number of chronic stressors experienced within that context; scores could range from 0 to 6. Each of these scores represented a context-specific score for chronic stress.
Home and Family Stress

Six sources of stress within the home and family context were examined.

Family Income

At each major data collection time point, mothers were asked about family income (for this study, the two major data collection points for stress included third and fifth grades). In order to capture income relative to family size, an income-to-needs ratio was used. This ratio was computed by dividing total family income by the poverty threshold for that household size (U.S. Department of Labor, 1994). Higher scores indicate greater financial resources in the household. For purposes of these analyses, the average income-to-needs ratio across third and fifth grades was calculated, and children living in families with an income-to-needs ratio higher than 2.0 (i.e., middle/upper income) were coded as 0 or not at risk; those with an income-to-needs of 2.0 or less (i.e., low income) were coded as 1 (chronic stressor).

Maternal Educational Status

Mothers reported on their number of years of education during the 1-month visit. This variable was dichotomized as 1 (chronic stressor) if the mother has high school education or less. Mothers with more than a high school education were coded as 0.

Single Parent Status

Single parent status was assessed by determining whether the youth participant resided in a household with a single/never married, separated, or divorced parent. If a child resided in a single parent household at either the third or fifth grade time point a value of 1 was assigned.
Minority Status

This was reported at the 1 month home visit by the mother. The SECCYD study sample included 24% of children with ethnic minority status. Within this overall minority status, 13% were African American, 6% Hispanic, 2% were Native American or Asian, and 3% self-reported as Other. Being a minority in current American culture is a known stressor and, therefore, all minority participants were assigned to the risk group and coded as 1. European Americans assigned to the (0) non-risk group.

Home Chaos

Perceptions of environmental confusion (e.g., noise, commotion, and crowding) in the child’s home were assessed by maternal self-report using a questionnaire entitled Confusion, Hubbub, and Order Scale (CHAOS) (Matheny, Wachs, Ludwig, & Philips, 1995). This measure includes 15 items associated with the characteristics of environmental confusion such as “There is very little commotion in our home,” “You can’t hear yourself think in our home,” or “We can usually find things when we need them” The mothers were asked to endorse each item as true or false. Eight of the 15 items are reflected. Possible scores range from 15 to 30. Higher scores represent more home chaos. Cronbach’s alpha was .81. This measure was collected at only one time point, when the child was in third grade. Individuals with a score in the top twenty-fifth percentile were considered to have been residing in a chaotic home environment during third grade and were assigned a score of 1 (chronic stressor); the remaining participants were assigned a 0.
Maternal Depressive Symptoms

Maternal depressive symptoms were measured at all major time points throughout the study including the two time points during middle childhood that are the focus of this study, third and fifth grades. Mothers completed the Center for Epidemiological Studies Depression Scale (CES–D; Radloff, 1977). The CES–D was designed to measure symptoms of depression in nonclinical populations. Mothers rated the frequency of 20 symptoms during the past week on a scale from 0 (rarely or none of the time) to 3 (most or all of the time). CES-D scores from the two time points were averaged and mothers who were in the top twenty-fifth percentile for depressive symptoms were coded as 1 (chronic stressor). Cronbach’s alpha was .91.

Extrafamilial Stress

Six indices of Extrafamilial stress were examined. In each case, stress was defined as falling within the top or bottom twenty-fifth percentile of the distribution.

Neighborhood Safety Parent Report

When the research participants were in third and fifth grades, mothers completed the Neighborhood Questionnaire (Greenberg et al., 1999), which is a 16-item measure used to assess neighborhood social involvement and safety. Likert-scale responses were offered for each item. The 5 items about neighborhood safety included questions such as 1) “How satisfied are you with police protection around here?” “0 = very dissatisfied, 1 = somewhat dissatisfied, 2 = somewhat satisfied, or 3=very satisfied; 2) How often are there problems with muggings, burglaries, assaults, or anything else like that around here?” The items of the neighborhood safety subscale are summed with summary scores
ranging from 0 to 16; higher scores reflected greater perceived neighborhood safety. Cronbach’s alpha was .77.

**Neighborhood Safety Child Report**

In third grade, the children were asked to complete the Self-Care Checklist (SCC)-Child (Shumow, Vandell, & Posner, 1998; Vandell & Pierce, 2000), which is a questionnaire used to assess a child’s perception of neighborhood safety and emotional readiness for self-care. The items related to neighborhood safety were a series of declarative statements. A child was asked how much he or she agreed with statements such as: “It is safe to walk around my neighborhood,” “I feel safe playing outside my house,” and “There are people with guns and knives in my neighborhood.” Responses to items were rated on a 5-point scale (i.e., 1 = not at all true, 2 = a little true, 3 = somewhat true, 4 = mostly true, and 5 = really true). Scores for the neighborhood safety subscale range from 8 to 40 with higher scores indexing the perception of greater neighborhood safety. Cronbach’s alpha was .82.

**Feelings about School**

In fifth grade children were asked to complete a 20-item questionnaire to assess their feelings about school, school-related perceived competence, school motivation, and school social aspects. The items related to feelings about school included declarative statements. Examples of these statements included the following: “In general, I like school a lot.” “School bores me.” “I don’t feel like I really belong at school.”
Responses to items were rated on a 4-point scale (i.e., 1 = not at all true, 2 = a little true, 3 = sort of true, and 4 = very true). A total mean score is calculated, which can range from 1 – 4, with higher scores indicating more positive feelings about school. Cronbach’s alpha = .85.

**School Attachment**

In sixth grade, the research participants completed a questionnaire about their perceptions of school climate (Johnson, Crosnoe, & Elder, 2001). The school attachment subscale of this measure includes 5 items: 1) I am happy to be at my school, 2) Teachers at school treat students fairly, 3) I feel close to others at school, 4) I feel safe at my school, and 5) I feel like I am a part of my school. These items are scored on a 5-point scale (i.e., 1 = not at all true, 2 = a little true, 3 = somewhat true, 4 = mostly true, and 5 = really true). Scores are computed as the mean of the responses with a possible range of 1 to 5. Higher mean scores indicate higher school attachment. Cronbach’s alpha was .74.

**Peer Victimization**

Perceived peer victimization was assessed in both third and fifth grades using the Perceptions of Peer Support Scale (PPSS), which is an 18-item, interviewer-guided, self-report measure used to assess peer victimization, peer support, and participation in bullying behaviors (Kochenderfer & Ladd, 1996). Interviewers asked each child the four questions that were used to assess peer victimization: “Does anyone in your class ever: 1) Pick on you at school? 2) Hit you at school? 3) Say mean things to you at school? 4) Say bad things about you to other kids at school?” Children were asked to provide one of five responses: 1 = never, 2 = hardly ever, 3 = sometimes, 4 = most of the time, 5 = always.
The mean score for the responses to these four items is the perceived victimization score with higher scores indicative of a higher frequency of victimization behaviors by child’s classmates. Internal consistency for this peer victimization subscale was 0.81.

**Loneliness**

In both third and fifth grades child distress was measured using a child report questionnaire entitled Loneliness and Social Dissatisfaction (Asher, Hymel, & Renshaw, 1984). Items on this questionnaire include, “It is easy for me to make new friends at school,” “I have lots of friends,” “I am lonely,” and “I feel left out of things.” Child participants were asked to provide 1 of 5 responses to each of these questions: 1 = not true at all, 2 = hardly ever true, 3 = sometimes true, 4 = true most of the time, 5 = always true. The score for child loneliness is computed from summing the scores which have a possible range of values of 16 to 80. Higher scores are indicative of more loneliness. For third grade, Cronbach’s alpha was .87 and for fifth grade Cronbach’s alpha was .91.

**Maternal Sensitivity**

During third and fifth grades, home observations between mother and child were conducted; these were done by way of semi-structured, 15-minute observations. For the third grade home visit observation the mother and child were given a shared, problem-solving task. This task was to plan errands around a make-believe town. For the fifth grade home visit observation, mother and child were asked to do two activities together. The first activity was to have a discussion about three topics of disagreement; these topics were selected by the mother and child together from a list provided, which contained 20 discussion topics such as personal appearance or swearing. The second mother-child
only observational activity was a game called Tower of Toothpicks. Child and parent had to build a tower from toothpicks and other materials. Both of these activities were videotaped.

The video-taped observations were coded by blind reviewers using a 7-point rating scale. Composite scores of maternal sensitivity were created using the sum of ratings for the following (age-modified) scales at each age: supportive presence, respect for autonomy, and hostility. Cronbach’s α for maternal sensitivity composites ranged from .71 to .87 across all the mother-child dyads and ages of measurement. Inter-observer reliability across independent raters ranged from .81 to .91. Higher scores are associated with higher levels of maternal sensitivity.

Analysis Plan

Preliminary Analysis

Analyses were performed using SPSS® 21.0 for Windows (IBM, Inc., USA) and JMP® 10.0 (SAS Institute, Inc., Cary, NC). Data analysis proceeded in the following manner. First, descriptive statistics were calculated for the demographic characteristics of the sample. Next, descriptive statistics were calculated for the allostatic load variables; cutoff for risk were those values that were equal to or greater than the 75th percentile. Next, zero-order correlations among the continuous allostatic load variables were calculated. Descriptive statistics were calculated for the Home/Family stressors. Cutoff for assigning risk scores were determined variable by variable. For instance, if a mother had 12 or less years of education a risk score of 1 was assigned; those mothers with greater than 12 years of education were not assigned a risk score. Zero-order
correlations were then run among the Home/Family continuous variables. For Extrafamilial stressors descriptive statistics were run. Next, cutoff values were calculated stressors using the highest or lowest quartile, depending on the how each measure was scored. Zero-order correlations were calculated among the Extrafamilial stressors. Lastly, zero-order correlations were run between the continuous allostatic load variables and the continuous Home/Family risk variables. This process was repeated between the allostatic load variables and the Extrafamilial risk variables.

Hierarchical regression analysis was employed to examine all four hypotheses. In all these analyses, sex of the child was entered as a control variable. Next, in block 2, the context-specific stress indices were entered, Home/Family stress and Extrafamilial stress. Centered terms were created for Home/Family stress and Extrafamilial stress (Aiken & West, 1991). Next, two-way interaction terms were computed for both centered-Home/Family stress x sex of research participant and centered-Extrafamilial stress x sex of the research participant.

This process was repeated in a second set of analyses involving maternal sensitivity. In block 1 sex of the research participant was entered followed by maternal sensitivity. In block 2, the Home/Family stress index was entered followed by Extrafamilial stress index. A centered term was created for maternal sensitivity (Aiken & West, 1991). Next, two interaction terms were created between Home/Family stress x maternal sensitivity and between Extrafamilial stress x maternal sensitivity. These two-
way interactions terms were entered in block 3. Finally, interaction terms were created for Home/Family x maternal sensitivity x sex and for Extrafamilial x maternal sensitivity x sex of the research participant. These three-way interaction terms were entered into Block 4.
CHAPTER V
RESULTS

Preliminary Analyses

Descriptive statistics for the six components of allostatic load were calculated and are shown in Table 2. Figure 1 shows the distribution of allostatic load scores. A quarter of the study sample had no elevated biomarkers, one third of the study sample had one elevated biomarker, less than 20 percent had two elevated biomarkers, 11 percent had three elevated components of allostatic load, 7% had four elevated components of allostatic load, 4% had five elevated components and 1% had elevation in all six components of allostatic load. Given the low number of individuals with 6 components of allostatic load, for analysis these individuals were grouped with those individuals with 5 components of allostatic load. Thus, for the primary analysis the Allostatic Load index ranged from 0-5, rather than 0-6.

Zero-order correlations were calculated between allostatic load components and are presented in Table 3. The three components associated with metabolic activity (i.e., BMI, waist- to-hip ratio, and total skin fold) were significantly associated with each other when compared with the non-metabolic allostatic load components (i.e., systolic blood pressure, diastolic blood pressure, and cortisol). Additionally, both systolic and diastolic
blood pressure values were significantly positively associated with each other. Cortisol
was significantly negatively associated with BMI, but was not significantly associated
with any other allostatic load component.

The descriptive data for the Home/Family stressors are shown in Table 4 and
Figure 2. As previously mentioned, the analysis sample in the present report is restricted
to a subsample of 699 of the original 1,364 participants. In terms of Home/Family
stressors, of these 699 individuals, 17 were missing Home Chaos evaluations, six were
missing mean income-to-needs ratio data, one was missing partner status data, and one
was missing maternal depressive data. Thirty-one percent of the research participants
had no significant Home/Family stressors during middle childhood while thirty percent
were found to have one substantial Home/Family stressor, which was risk from low
maternal education. Only 11 percent of this sample had three Home/Family stressors,
while 9 percent had four, 2 percent had five Home/Family stressors and only 1 percent
had all 6 Home/Family stressors. Since this was a similar frequency pattern as observed
with the allostatic load values, categories 5 and 6 were combined. Hence, for the primary
analysis the Home/Family stress index values ranged from 0-5.

Zero-order correlations were calculated between the Home/Family stressors and
are shown in Table 5. There were significant positive associations between all of the
Home/Family stressors with the exception of two non-significant associations involving
home chaos.
The descriptive characteristics for Extrafamilial stressors are shown in Table 6 and Figure 3. Of the 699 participants, twenty one participants had missing data for child report of neighborhood safety, twenty one participants also had missing data for school attachment, eighteen participants had missing data for child’s feelings toward school, six participants had missing data for peer victimization, one participant was missing data for maternal report of neighborhood safety, and one participants was missing data for loneliness. Twenty-seven percent, or slightly less than a third of the research participants reported no substantial

**Extrafamilial Stressors**

An additional 27% of the research participants reported one Extrafamilial stressor. Nineteen percent of research participants had two significant Extrafamilial stressors, while 15% reported three significant stressors, 7% reported four significant stressors, and 4% reported five significant stressors, finally, only, 1% reported six significant Extrafamilial stressors. The categories for 5 and 6 Extrafamilial stressors were collapsed into one category for analysis.

Zero-order correlations were calculated among the Extrafamilial stressors and are shown in Table 7. All of the correlations among the continuous Extrafamilial stressors are significant. Specifically, there are significant positive associations between maternal and child reports of neighborhood safety, the child’s report of feelings toward school and school attachment. Both mean peer victimization and mean loneliness were significantly and negatively associated with both maternal and child report of neighborhood safety as well as with a child’s report of feelings toward school and school attachment.
Next, zero-order correlations were calculated between the variables comprising the indices for Home/Family stress, as well as the overall Home/Family Stress Index and the variables making up allostatic load, including the allostatic load score. Results are shown in Table 8. There was a significant correlation between the Home/Family Stress Index and the allostatic load score. Additionally, four of the six Home/Family stressors were correlated with the Allostatic Load index.

Zero-order correlations were also calculated for the Extralimital stress predictor variables, the Extralimital stress index, and allostatic load variables as well as the allostatic load score (Table 9). Overall, the Extralimital stress index was not correlated with the Allostatic Load index. There was a significant correlation between BMI and the Extralimital stress index. There were two significant positive correlations between the individual Extralimital stressors and allostatic load; these were for mother report of neighborhood safety and child’s feelings about school.

To determine whether child sex needed to be used as a covariate, an independent-samples t-test was conducted to compare the allostatic load scores for females (n = 368) and males (n = 331). There was a significant difference in the scores for females (M = 1.37, SD = 1.31) and males [M = 1.75, SD = 1.50; t(659) = 3.53, p = .000]. Therefore, child sex was entered as a covariate in all analyses.
Primary Analyses

What is the relation between chronic stress during middle childhood and Allostatic Load in adolescence? And, is chronic stress experienced in a Home/Family context more predictive of adolescent Allostatic Load than chronic stress experienced in an Extrafamilial context?

To assess whether the level of chronic stress experienced during middle childhood is related positively to allostatic load during adolescence, a hierarchical regression analysis was performed. Sex of the research participant was dummy-coded with females as the reference group and entered as a control in block 1, and both Home/Family stress and Extrafamilial stress were entered in block 2. Although sex of the research participant was entered as a control variable, in order to find out if the results for the relationship between chronic stress and allostatic load were the same for males and females, two-way interactions between child sex and stress were considered in Block 3. Continuous variables were centered before creating the interaction terms.

Table 10 presents the results from the first set of hierarchical regression analyses. The overall model was significant: the total variance explained by the model as a whole was 4%, $F(5, 693) = 6.01, p < .001$. The covariate, sex of the research participant, explained 2%, of the unique variance in adolescent allostatic load, $R^2 = .02$, $F$ change (1, 697) = 12.63, $p < .001$. Specifically, as noted previously, males had higher allostatic load scores than females. The results for Block 2, in which the stress variables were entered, showed that stress accounted for an additional 1% of the variance, $\Delta R^2 = .03$, $F$ change (2, 695) = 5.66, $p < .004$. Within this block, only Home/Family stress was a significant
predictor of allostatic load. The results of Block 3 which included two-way interactions
between sex of research participant and type of chronic stress were marginally
significant, $\Delta R^2 = .01, F$ change $(2, 693) = 2.91, p \geq .055$, and each of the interactions
was significant. Follow-up simple slopes analyses, shown in Figures 4 and 5, indicated
that the relation between Home/Family stress and allostatic load was significant only for
males and the relation between Extrafamilial stress and allostatic load was significant
only for females.

These data provide evidence supporting the first hypothesis that chronic stress
experienced during middle childhood is related to adolescent allostatic load.
Additionally, these data indicate that it is primarily Home/Family stress that is associated
with allostatic load, but not Extrafamilial stress. Thus, chronic stress experienced in a
Home/Family context is predictive of adolescent allostatic load, but Extrafamilial stress
in this data set is only marginally significant. Finally, although not hypothesized, the
relation between contexts of stress and allostatic load differs for males and females.

*Is the Relationship Between Chronic Stress During Middle Childhood and Adolescent
Allostatic Load Moderated by Maternal Sensitivity?*

A second hierarchical regression analysis was conducted to address the third
research question. Continuous variables were centered prior to calculating the interaction
terms. Additionally, because sex of the research participant was significant in the
previous analysis, it was included in this model and three-way interaction terms -- type of
chronic stress x maternal sensitivity x sex -- were entered in Block 4. Table 11 shows the
results of this analysis. Thus, Block 1 included the two potential moderators, child sex
and maternal sensitivity; Block 2 the stress variables, Block 3 the interactions with maternal sensitivity, and Block 4 the 3-way interactions.

The overall model was significant. The total variance explained by the model as a whole was 2%, $F(8, 533) = 2.61, p < .008$. The first block explained 2% of the unique variance in adolescent allostatic load, $R^2 = .02, F(1, 697) = 5.28, p < .01$. Only child sex was significantly related to allostatic load. The results for Block 2 showed the same pattern as the previous analysis, in that the overall block was significant, $\Delta R^2 = .03, F(2, 695) = 4.10, p \leq .01$, but only Home/Family stress was related to allostatic load. The addition of Block 3 which included two-way interactions between maternal sensitivity and type of chronic stress did not add significantly to the prediction of Allostatic Load $\Delta R^2 = .02, F(4, 693) = .02, p = .98$. Block 4, which included three-way interactions, also did not reveal significant results, $\Delta R^2 = .02, F(6, 691) = 1.08, p = .36$. In total, these analyses do not provide support for the hypothesis that the relationship between chronic stress in either a Home/Family or an Extrafamilial context and adolescent allostatic load is moderated by level of maternal sensitivity.

**Additional Analyses**

Given that the allostatic load components associated with metabolic activity (i.e., BMI, waist-to-hip ratio, and total skin fold) were significantly associated with each other, compared with the non-metabolic components and the significant research interest in obesity and metabolic issues, a follow-up analysis was done examining just these variables. An Obesity Index was calculated from the three metabolic activity indicators, with a score ranging from 0 – 3. Using the Obesity Index value as the dependent variable
hierarchical regression analyses were done in the same manner as described above. Sex of the research participant was dummy-coded with females as the reference group and entered as a control in block 1 and both Home/Family stress and Extrafamilial stress were entered in block 2. Although sex of the research participant was entered as a control variable, in order to find out if the results for the relationship between chronic stress and allostatic load were the same for males and females, two-way interactions were considered in Block 3. Continuous variables were centered before creating the interaction terms. The goal was to assess whether the level of chronic stress experienced during middle childhood is related positively to an adolescent Obesity Index.

Table 12 shows the results from the hierarchical regression analyses. The overall model explained 5% of the unique variance in the adolescent Obesity Index, $F(5, 693) = 7.69, p < .000$. In Block 1, sex of the research participant explained 1% of the unique variance in the adolescent Obesity Index, $R^2 = .01, F(1, 697) = 9.59, p < .01$. The results for Block 2 showed revealed an adjusted $R^2 = .04, F(2, 695) = 12.73, p < .001$. An additional 3% of the unique variance in the adolescent Obesity Index was explained by stress, with only Home/Family stress showing a significant relation to the Obesity Index. The addition of Block 3, which included two-way interactions between sex of research participant and type of chronic stress, were significant, $\Delta R^2 = .05, F(4, 695) = 2.40, p < .05$. Only the interaction between Home/Family stress and child sex was significant.

A simple slopes analysis, shown in Figure 6, indicated that the relation between Home/Family stress and the Obesity Index is moderated by sex of the research participant, such that the relation between Home/Family stress and the Obesity Index is
only significant for males. In total, these data provide evidence that chronic stress experienced in the Home/Family context is more highly related to metabolic dysregulation, as measured by the Obesity Index, compared with chronic stress experienced in the Extrafamilial context, and this relationship is significant only for males.

Analyses were also carried out to examine whether maternal sensitivity moderated the relation between stress and the Obesity Index. As in the primary analyses, no significant results were found for maternal sensitivity.

Overall, these data provide evidence support the first hypothesis that chronic stress experienced during middle childhood is related to adolescent allostatic load. Additionally, these data support the second hypothesis that primarily Home/Family stress is associated with adolescent allostatic load; however, this relationship was only significant for males.

Furthermore, the marginal significance of the interaction between Extrafamilial stress and sex of the research participant suggests a relationship that is only significant for females. Further investigation is needed of this and why different contexts of chronic stress appear to affect males and females differently. Finally, none of the analyses indicated that the relationship between chronic stress during middle childhood and adolescent allostatic load were moderated by levels of maternal sensitivity.
CHAPTER VI
DISCUSSION

Biological frameworks used to study stress have centered on the idea of
equilibrium being central to health: too much chronic stress can disrupt physiological
equilibrium, which may lead to pre-disease or disease states. The intervening processes
between stress and health have remained largely elusive until the introduction of the
heuristic concepts of allostasis and allostatic load (McEwan & Stellar, 1993; Sterling &
Eyer, 1988). Allostasis is the body’s dynamic physiological adaptation to stressors in the
environment. Allostatic load is the cumulative burden from wear and tear on the
different physiological systems. In adults, the presence of allostatic load can be
associated with pre-disease states (McEwen, 1998; Seeman et al., 2008) while higher
levels of allostatic load are positively related to poor health outcomes (Nelson, Reiber,
Kohler, & Boyko, 2007; Seeman, McEwen, Rowe, & Singer, 2001; Seeman, Singer,

Prior Research Involving Children and Allostatic Load

To date, only a few studies have examined allostatic load in children or
adolescents. Johnston-Brooks and colleagues (Johnston-Brooks, Lewis, Evens, &
Whalen, 1998) examined the effects of household density on cardiovascular reactivity,
defined as changes in cardiovascular activity such as health rate and blood pressure in
response to stress, in a sample of 81 fifth and sixth grade boys. Using structural equation
modeling, they found that a mediating model accounted for 17% of the unique variance for number of days ill; their findings support the role of allostasis as an explanatory variable between chronic stress and illness.

When four very different groups (i.e., homeless street children, urban squatters, urban middle class, and village residents) of Nepali boys were compared in terms of allostatic load, results showed that the different contexts are associated with differing levels of allostatic load (Worthman & Panter-Brick, 2008). While it had been expected that those boys with the worse circumstances would have the higher allostatic load (e.g., boys who were urban squatters and homeless boys), it turned out that homeless and village boys fared the worst in terms of allostatic load. This study underscores the fact the children’s chronic stress varies by context, but comparative studies are needed to challenge our assumptions about some contexts and in order to accurately assess the context and extent of associated developmental risk.

The current study tested an allostatic load model by looking at chronic stress experienced during middle childhood in a population of healthy, typically-developing children. The study examined children in two different contexts, Home/Family and Extrafamilial, to find out whether such exposure was associated with the development of adolescent allostatic load.

**The Relation of Chronic Stress to Allostatic Load**

Overall, the findings were consistent with the first hypothesis, that the level of chronic stress experienced during middle childhood is associated positively with allostatic load in adolescence. In this study, it was also found that the context in which
children experience stress matters, as does the sex of the research participant. These findings were partially consistent with the second hypothesis, that stress experienced in the Home/Family context is more predictive of allostatic load in adolescence than stress experienced in the Extrafamilial context. However, while stress experienced in the Home/Family context is more predictive of allostatic load in adolescence, this relationship was moderated by the sex of the research participant: the relationship between Home/Family stress and allostatic load was only significant for males. In contrast, the relationship between Extrafamilial stress and allostatic load was only significant for females. These findings about sex of the research participant moderating the relationship between chronic stress and allostatic load were not anticipated and, therefore, no hypotheses were made. Understanding why these relationships differ for males and females calls for additional research to identify how chronic stress in varying contexts may be acting differently for boys and girls.

**Maternal Sensitivity as a Moderator of the Relation between Chronic Stress and Allostatic Load**

In the third hypothesis it was predicted that maternal sensitivity would moderate the relationship between chronic stress and allostatic load. This prediction was predicated on research suggesting that maternal sensitivity is associated with helping children regulate their response to stress, which in turn contributes to the overall healthy functioning of the HPA axis. In this sample, maternal sensitivity was not a significant moderator.
Comparison of Results with Earlier Literature

In this study, an effort was made to both replicate and extend prior research about maternal sensitivity as a moderator of the relationship between chronic stress and allostatic load. Evans and colleagues (Evans, Kim, Ting, Tesher, & Shannis, 2007) demonstrated that as cumulative stress increased so did allostatic load, but only for children whose mothers exhibited low levels of maternal responsiveness. In their sample, high levels of maternal responsiveness appeared to have a protective effect for the relation between cumulative stress and allostatic load. This previous study drew participants from a longitudinal study of rural poverty, so the data set was composed of rural-dwelling, European-American adolescents. Cumulative stress, or risk, was categorized into three different domains: 1) physical (i.e., crowding in housing, noise in housing, and substandard housing), 2) psychosocial (i.e., exposure to violence, family turmoil, and child-family separation), and 3) personal characteristics (i.e., poverty, single parenthood, and maternal high school dropout status). Allostatic load was comprised of 6 biomarkers (i.e., overnight urinary cortisol, epinephrine, norepinephrine, systolic blood pressure, diastolic blood pressure, and BMI).

The current study differed from the study by Evans and colleagues (Evans, Kim, Ting, Tesher, & Shannis, 2007) in terms of the sample size and composition, stressor data collected, and biomarkers used for allostatic load. First, Evans and colleagues (2007) drew on a sample of rural-dwelling, European-American adolescents (n = 207). In contrast, this current study has a larger sample size (N = 699), more diversity with 22% of the current sample having minority status, and participants drawn from families living...
throughout the continental United States with more income diversity (77% of participants came from families with an income-to-needs ratio of 2.0 or greater). Secondly, each of the studies defined chronic stress in different ways; Evans and colleagues (2007) focused on physical, psychosocial, and personal characteristics, while this study focused on Home/Family and Extrafamilial variables. The only overlaps between the two studies were poverty, single parenthood, and maternal high school dropout status. The current study conceptualized these characteristics as Home/Family variables, along with minority status, home chaos, and maternal depressive symptoms. The current study also differentiated itself with the Extrafamilial factors, which included data about perceived neighborhood safety, school attachment and feelings about school, and child reports of peer victimization and loneliness. Finally, the biomarkers used in each study differed. Both studies used systolic blood pressure, diastolic blood pressure and BMI. However, Evans and colleagues (2007) also used overnight urinary cortisol, epinephrine, and norepinephrine; these are all markers of HPA axis functioning. In contrast, the current study used skinfold measurements, waist-to-hip ratio, and morning salivary cortisol.

Although Evans and colleagues (Evans, Kim, Ting, Tesher, & Shannis, 2007) showed that the relationship between cumulative stress and allostatic load was moderated by maternal responsiveness, the relationship only held for children whose mothers exhibited low levels of maternal responsiveness. A simple slopes analysis revealed that as chronic stress increased so did allostatic load, but only for those individuals whose mother showed low responsiveness. This finding makes theoretical sense and is consistent with current research, as previously mentioned, which suggests that maternal
sensitivity is associated with helping children regulate their response to stress. In contrast, the present study found no evidence that maternal sensitivity moderated the relationship between chronic stress and allostatic load. This lack of moderation held across both contexts tested, Home/Family and Extafamial. Nor was there any evidence for a main effect.

The differences in the findings between these two studies are likely to be due to specific differences in the methodology of the studies. Several of these differences have been mentioned including sample size and composition, the variation in the stress variables, and the differing biomarkers used to measure allostatic load. However, another fundamental methodological difference is how maternal responsiveness was measured. Evans and colleagues (Evans, Kim, Ting, Tesher, & Shannis, 2007) measured maternal responsiveness one time, using a combination of each youth’s perception of maternal responsiveness and observation rating of mother-child interaction during a cooperative game. In particular, youth’s perception of maternal responsiveness was measured using an 11-item constructed scale which included items related to both instrumental (e.g., help with homework) and emotional (e.g., willingness to talk) responsiveness. In comparison, this current study used home observations between mother and child, during third and fifth grades. These semi-structured, 15-minute observations involved age-appropriate activities for the child. The difference in the measurement of maternal responsiveness in both studies likely contributed to the dissimilar findings.
The findings from this study show a small, yet significant positive relationship between chronic stress experienced during middle childhood and the presence of allostatic load during adolescence. These data support the first part of the biological-process model that chronic stress is linked with allostatic load. (The second part of the model that allostatic load is linked to disease states was not investigated in this study.) This finding contributes to the research literature in this area because to date most of the limited research studies about this topic involving children and allostatic load has focused on adverse childhood experiences or toxic stress as a proximal cause (AAP, 2012; CDC, 2010; DeBellis, 2001; Worthman & Panter-Brick, 2008). However, the focus of this study was on whether chronic stress experienced by healthy, typically-developing children is associated with allostatic load. This specific research is important because it provides information about whether the Allostatic Load framework might have utility for all children – even those not exposed to toxic stress. There is very limited research in this area with populations of children and adolescents (MacArthur SES & Health Network, 2013). The few studies to date show that specific types of chronic stress (i.e., household density) are associated with allostatic load and illness (Johnston-Brooks, Lewis, Evans, & Whalen, 1998) and that children in different contexts display different profiles and different levels of allostatic load (Worthman & Panter-Brick, 2008). Most of the research on allostatic load has been done with populations of adults (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997; Seeman, Rowe, McEwen, & Singer, 2001). Prior research in several different adult populations indicates that individuals with the lowest levels of education have significantly higher
allostatic load scores compared with those with the highest levels of education (Hawkley, Lavelle, Bernton, & Cacioppo, 2011; Kubzansky & Sparrow, 1999; Seeman et al., 2008). Other studies show that Blacks and other individuals with minority status have increased allostatic load compared to those with majority status (Geronimus, Hicken, Keene, & Bound, 2006; Seeman et al., 2008). In addition to looking at many different chronic stressors, research studies with adults have also focused on long-term health outcomes Nelson, Reiber, Kohler, & Boyko, 2007; Seeman, McEwen, Rowe, & Singer, 2001; Seeman, Singer, Ryff, Love, & Levy-Storms, 2002; Sabbah, Watt, Sheiham, & Tsakos, 2008). This is something that has not really been doing in populations of children and adolescents.

**Measurement of Stress**

In the present study, the focus has been on chronic stress. Chronic stress was chosen for several reasons. First, this is because the earliest theoretical conceptualizations about allostatic load indicated that it was the continual or repetitive arousal from chronic stress that would likely result in the wear and tear on the physiological systems (McEwen, 1998; McEwen & Stellar, 1993). That is not to imply that acute, toxic or other forms of stress do not result in physiological dysregulation, but rather chronic stress seem likely – theoretically speaking – to do the most harm. Second, the stress research literature indicates that daily stressors rather than major life events were related to psychological symptoms (Lewis, Siegel & Lewis, 1984; Wagner, Compas, & Howell, 1986).
In additional to focusing on chronic stress, efforts were made to use child report data for many of the chronic stressors included in this study (i.e., child report of neighborhood safety, school attachment, feelings about school, peer victimization, and loneliness). This heavy reliance on child reports of perceived stress is intentional as perceived stress has been shown to be a good indicator and, sometimes, a better indicator, of how an individual child experiences it physiologically (Compas, Howell, Phares, Williams, & Giunta, 1989; Dufton et al., 2008; Thomsen et al., 2002; Wu & Lam, 1993).

Finally, by using a bioecological framework with a developmental perspective (Bronfenbrenner & Morris, 1998) it was possible to target the primary contexts in which children are likely to experience stress, Home/Family and Extrafamilial environments. Furthermore, this framing allowed for the modeling of the interaction between individuals and context over time revealing that both sex of the research participant and context of chronic stress are important variables. Both variables need to be focused on in greater detail so that a more complete picture can be developed of how chronic stress impacts children as they develop from middle childhood into adolescence.

Other studies of children do not typically utilized a bioecological (developmental) framework, but instead focus on the total number of stressors present in a child’s life. This is because a cumulative risk model was first used in developmental psychology by Sir Michael Rutter with his Isle of Wight study (Rutter, 1979). Cumulative risk, or simply the sum of risk (stressors) experienced by a child, was used as a predictor of child outcomes. This classic research study showed an association between the number of cumulative risks and the present of externalizing and internalizing behaviors in children
with children with two of more stressors found to have an increase in negative adjustment. Multiple studies in various different child populations have been conducted and replicated this association (Appleyard, Egeland, van Dulmen, & Sroufe, 2005; Sanson, Oberklaid, Pedlow, & Prior, 1991; Trentacosta et al., 2008).

Studies about the association between stress and allostatic load in adults often conceptualize stress slightly differently focusing on a variety of issues ranging from socioeconomic disadvantage (Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010), to neighborhood segregation (Bellatore, Finch, Do, Bird, & Beck, 2011), or to length of residency and proximity to environmental hazards (Mair, Cutchin, & Peek, 2011). These approaches provide novel and valuable data, but also make comparisons between studies difficult.

One finding common to most studies looking at stress and allostatic load is the equal weighting of each stress variable. One reason for this occurrence is the custom for how cumulative risk studies are conducted in psychology and related research areas (Rutter, 1979). There are some limitations with this traditional approach of a stress index. First, linearizing all the stress variables and treating them in an equal manner likely result in the loss of statistical power. Furthermore, some stressors such as maternal depressive symptoms can be so pervasive that treating them in an equal manner may result in an underestimation of the true effect of such a particular stressor (Richters, 1992). There are several thoughts about handle predictors of equal weight. Wainer (1976) in is classic paper argued that using equal weights for predictors was likely to have a small effect on the accuracy of the outcome variable. Another methodological
approach is to test and compare a series of models ranging from a less restricted model with unequal weighting of all predictors to using a restricted model by weighting all predictors equally (Appelbaum & Cramer, 1974; Faldowski, 2012). Theory and research literature should guide which methodology to employ. For this study the traditional approach of equal weighting of all the predictors used by both developmental psychologists and allostatic researchers was chosen (Rutter, 1979; Seeman, Rowe, McEwen, & Singer, 2001).

**Studying Allostatic Load**

Allostatic load is important concept because it provides a conceptual basis for quantifying the biological effects of chronic stressors in many types of populations and, thus, is useful for identifying and teasing apart the processes linking stress to health, physiological dysregulation, pre-disease states, and disease. The findings in this study provide evidence that support further investigation of the possible use of this framework in populations of children during middle childhood and adolescence. And, although only 4% of the total variance was explained in the models tested in this study these data show that allostatic load does occur in a population of healthy, typically-developing children. It is likely that a higher percent of total variance might have been explained if different biomarkers had been available for the construction of the Allostatic Load index for this present study. And, this is one of the greatest methodological challenges facing this specific research -- the lack of a uniform designation of the biomarkers that should comprise allostatic load. Theoretically, the biomarkers used should be related to the body’s chronic stress response. However, in practice, the availability of biomarkers has
dictated which ones are used to create the Allostatic Load index. This is true for studies involving children or adults. This methodological problem has been noted by researchers who now endorse a fine-tuning of the definition of allostatic load to include primary chemical mediators involved in the stress response (i.e., cortisol, norepinephrine, epinephrine, and DHEA) (MacArthur SES & Health Network, 2013). Additionally, researchers suggest that the inclusion of other biomarkers be those that have direct relevance to the outcome being studied be that allostatic load, pre-disease, and/or disease states.

Despite the limited availability of biomarkers for the construction of the Allostatic Load index in this study, by age 15 years, two-thirds of the individuals in this data set showed evidence of allostatic load. A lesser percentage of this population would likely meet diagnostic criteria for metabolic syndrome; however, diagnosis of a pre-disease or disease state was beyond the scope of the present study. What is clear is that using multiple biomarkers, most of which are routinely done in clinical practice, with an allostatic load framework guiding the selection and interpretation of the biomarkers appears to merit further research for use in middle child and adolescent health settings. Identifying individuals in an adolescent population with allostatic load and, perhaps, even providing more extensive monitoring or intervention for those adolescents with high levels of allostatic load may ameliorate pre-disease states and, possibly, prevent or delay the onset of disease. However, much more research is needed before this framework can be considered for use in a clinical setting.
Strengths and Contributions of Current Study

One portion of the life course that has not been well studied is middle childhood; this research adds to the very limited research on chronic stress during that time period.

A second strength is the use of a bioecological framework with a developmental perspective (Bronfenbrenner & Morris, 1998). This framing allowed for the modeling of the interaction between individuals and context over time revealing that both sex of the research participant and context of chronic stress are essential variables needed to begin to appreciate a more complete picture of how chronic stress impacts children, especially as they develop from middle childhood into adolescence. The findings elucidated by this theoretical framing indicate that future research in this area would benefit from considering the context of chronic stress and the sex of the research participant. Finally, an additional strength of this study is that it relied heavily on child reports of perceived stress. This is significant because perceived stress has been shown to be a good indicator and, sometimes, a better indicator than other self-report stress measures, of how an individual child experiences it physiologically.

Limitations of This Study

This study was limited by at least three significant issues. First, the overall analyses were restricted to those research participants for whom full biomarker data were available, bringing the initial goal of more than 1200 participants down to 699. Second, morning cortisol was not added to the original study as a biomarker until the last data collection wave; therefore it was only possible to assess allostatic load at a single time point. Both of these factors made it impossible to assess allostatic load in prospective
longitudinal manner, over the course of childhood. A prospective longitudinal design should be considered for future studies, as this will allow us to begin to understand how allostatic load varies over the course of development in children and adolescents.

**Future Research**

As shown in this study, when the Allostatic Load framework when utilized to study a population of healthy, typically-developing children identified individuals with chronic stress and this was related in a positive linear manner to allostatic load. Thus, in general, we might expect that all individuals, including children, who experience chronic stress to eventually exhibit signs of allostatic load. This is unlikely to be true as acknowledged in the introduction. Thus, we continue to caution that this prediction was predicated on the supposition that individuals biologically react to chronic stress in the same manner and at the same magnitude. That is, understanding when and for whom the Allostatic Load framework and the associated intervening processes need further elucidation, and, as this study suggests appear to be more complex than has been previously thought.

Additional future research in this area should include prospective longitudinal studies of individuals over the life course in order to find out normal values and variations throughout the course of normal development. This greater methodological rigor will likely inform us of the extent to which the allostatic load framework has research and/or clinical utility for children, adolescents and onward over the life course.

Moreover, hormones and other biomarkers associated with HPA axis and autonomic nervous systems functioning likely also have effects on the cognition and
emotions involved in individuals’ stress responses (Peters & McEwen, 2012). Exploring these potential effects could be accomplished using a cross-lagged model or nonrecursive structural model (Kline, 2005).

Finally, the present research literature on childhood stress focuses on psychosocial stress and impairment, and neglects the concomitant physiological dysfunction or even physical findings (e.g., pre-disease, disease states) experienced by children. Studies focusing on both psychosocial and physical outcomes are needed in order for researchers, developmentalists and clinicians to have a more complete picture of how children experience chronic stress and the health consequences of chronic stress.

**Conclusion**

The results of this study indicate that chronic stress experienced during middle childhood is related to allostatic load during adolescence. However, the relationship between the context of chronic stress and allostatic load was moderated by the sex of the research participant. Specifically, the relation between Home/Family stress and allostatic load was significant for males while the relation between Extrafamilial stress and allostatic load was significant for females. With regard to maternal sensitivity, no evidence was found that maternal sensitivity moderated the relationship between chronic stress and allostatic load.

There are clear research and potential clinical implications for these findings. First, as in this study, allostatic load is likely present in at least a subset of healthy, typically-developing children by the time they reach age 15 years. Whether these children go on to develop pre-disease or disease states is not yet clear, but is suggested by
literature of young adults and adults. Still, using the allostatic load framework to monitor children and adolescents for physiological dysregulation, pre-disease, or disease states may have utility in terms of health promotion and disease prevention.
REFERENCES


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### Table 1. Demographic Characteristics of the Sample (N = 699)

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>%</th>
<th>M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean income-to-needs ratio</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Under 2.0</td>
<td>160</td>
<td>23.08</td>
<td></td>
</tr>
<tr>
<td>2.0 or above</td>
<td>541</td>
<td>77.39</td>
<td>4.21 (3.22)</td>
</tr>
<tr>
<td>Maternal education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HS or less</td>
<td>198</td>
<td>28.32</td>
<td></td>
</tr>
<tr>
<td>Some college, 4-yr degree or more</td>
<td>501</td>
<td>71.67</td>
<td>14.41 (2.41)</td>
</tr>
<tr>
<td>Maternal minority status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-minority</td>
<td>544</td>
<td>78.11</td>
<td>-</td>
</tr>
<tr>
<td>Minority</td>
<td>155</td>
<td>22.22</td>
<td>-</td>
</tr>
<tr>
<td>Child sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>331</td>
<td>47.35</td>
<td>-</td>
</tr>
<tr>
<td>Female</td>
<td>368</td>
<td>52.64</td>
<td>-</td>
</tr>
<tr>
<td>Partner status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partner in home</td>
<td>521</td>
<td>74.53</td>
<td>-</td>
</tr>
<tr>
<td>Single at either third or fifth grades</td>
<td>178</td>
<td>25.46</td>
<td>-</td>
</tr>
</tbody>
</table>
Table 2. Descriptive Characteristics for Allostatic Load Variables (N = 699)

<table>
<thead>
<tr>
<th>Variable</th>
<th>M for study sample</th>
<th>SD</th>
<th>Actual Range</th>
<th>Cutoff for Risk $\geq 75$ percentile</th>
<th>N at Risk</th>
<th>Percent of Sample at Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>113.74</td>
<td>11.68</td>
<td>83.33 – 173.00</td>
<td>$\geq 120$</td>
<td>180</td>
<td>25.75</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>62.44</td>
<td>6.01</td>
<td>40.67 – 90.00</td>
<td>$\geq 66.33$</td>
<td>184</td>
<td>26.32</td>
</tr>
<tr>
<td>Waist-to-hip ratio (cm)</td>
<td>0.77</td>
<td>0.06</td>
<td>0.64 – 1.04</td>
<td>$\geq 0.80$</td>
<td>195</td>
<td>27.89</td>
</tr>
<tr>
<td>Total skinfold (mm)</td>
<td>27.34</td>
<td>12.01</td>
<td>7.00 – 91.50</td>
<td>$\geq 33.50$</td>
<td>178</td>
<td>25.50</td>
</tr>
<tr>
<td>Body mass index (kg/m$^2$)</td>
<td>22.76</td>
<td>4.65</td>
<td>15.05 – 44.23</td>
<td>$\geq 24.53$</td>
<td>174</td>
<td>24.89</td>
</tr>
<tr>
<td>Cortisol, mean waking level (µg/dl)</td>
<td>0.36</td>
<td>0.17</td>
<td>0.02 – 1.11</td>
<td>$\geq 0.46$</td>
<td>177</td>
<td>25.32</td>
</tr>
</tbody>
</table>
Table 3. Correlations among the Continuous Allostatic Load Variables (N = 699)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Body mass index (kg/m²)</th>
<th>Waist-to-hip ratio (mm)</th>
<th>Total Skinfold (cm)</th>
<th>Systolic Blood Pressure (mmHg)</th>
<th>Diastolic Blood Pressure (mmHg)</th>
<th>Cortisol (µg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Body mass index (kg/m²)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Waist-to-hip ratio (mm)</td>
<td>.52***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Total skin fold (cm)</td>
<td>.79***</td>
<td>.30***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Systolic blood pressure (mmHg)</td>
<td>.43***</td>
<td>.40***</td>
<td>.26***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Diastolic blood pressure (mmHg)</td>
<td>.13***</td>
<td>.10</td>
<td>.16***</td>
<td>.50***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Cortisol, average waking level (µg/dl)</td>
<td>-.10**</td>
<td>-.04</td>
<td>-.01</td>
<td>-.05</td>
<td>-.01</td>
<td></td>
</tr>
</tbody>
</table>

Note: * = p < .05, ** = p < .01, *** = p < .001
<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>M for study sample</th>
<th>SD</th>
<th>Actual Range</th>
<th>Cutoff for Risk</th>
<th>N at Risk</th>
<th>Percent of Study Sample at Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean income-to-needs ratio</td>
<td>693</td>
<td>4.19</td>
<td>3.22</td>
<td>0.13 – 25.85</td>
<td>≤ 2.0</td>
<td>160</td>
<td>23.08%</td>
</tr>
<tr>
<td>Maternal education</td>
<td>699</td>
<td>14.41</td>
<td>2.41</td>
<td>7 - 21</td>
<td>≤ 12 years</td>
<td>198</td>
<td>28.32%</td>
</tr>
<tr>
<td>Partner status</td>
<td>698</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>Ever single at G3 or G5</td>
<td>171</td>
<td>24.49%</td>
</tr>
<tr>
<td>Minority status</td>
<td>699</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>Anything other than white, non-Hispanic</td>
<td>155</td>
<td>22.22%</td>
</tr>
<tr>
<td>Home chaos (Third grade only)</td>
<td>682</td>
<td>19.01</td>
<td>3.18</td>
<td>15 - 29</td>
<td>≥ 21</td>
<td>183</td>
<td>26.83%</td>
</tr>
<tr>
<td>Mean depressive symptoms (Third &amp; fifth grades)</td>
<td>698</td>
<td>9.04</td>
<td>7.71</td>
<td>0 – 52</td>
<td>&gt;12.5</td>
<td>178</td>
<td>25.50%</td>
</tr>
</tbody>
</table>
Table 5. Correlations among the Home/Family Continuous Variables (N = 699)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Income-to-needs ratio</th>
<th>Maternal education</th>
<th>Partner status</th>
<th>Minority status</th>
<th>Home chaos</th>
<th>Maternal depression</th>
</tr>
</thead>
<tbody>
<tr>
<td>M Income-to-needs ratio</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal education</td>
<td>.51***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pa Partner status</td>
<td>-.30***</td>
<td>-.25***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minority status</td>
<td>-.30***</td>
<td>-.31***</td>
<td>.24***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>M Home chaos</td>
<td>-.17***</td>
<td>-.08*</td>
<td>-.03</td>
<td>-.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Maternal depressive symptoms</td>
<td>-.30***</td>
<td>-.19***</td>
<td>.17***</td>
<td>.11**</td>
<td>.40***</td>
<td></td>
</tr>
</tbody>
</table>

Note: * = p < .05, ** = p < .01, *** = p < .001
Table 6. Descriptive Characteristics for Extrafamilial Stressors

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>M for study sample</th>
<th>SD</th>
<th>Actual Range</th>
<th>Cutoff for Risk</th>
<th>N at Risk</th>
<th>Percent of Study Sample at Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean maternal report of neighborhood safety</td>
<td>698</td>
<td>8.27</td>
<td>1.30</td>
<td>2.33 – 10.00</td>
<td>≤ 7.58</td>
<td>172</td>
<td>24.64%</td>
</tr>
<tr>
<td>(third &amp; fifth grades)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child report of neighborhood safety</td>
<td>678</td>
<td>3.73</td>
<td>0.92</td>
<td>1.00 – 5.00</td>
<td>≤ 3.1429</td>
<td>177</td>
<td>26.11%</td>
</tr>
<tr>
<td>(third grade only)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child’s feelings toward school</td>
<td>681</td>
<td>3.49</td>
<td>0.34</td>
<td>2.05 – 4.00</td>
<td>≤ 3.30</td>
<td>188</td>
<td>26.90%</td>
</tr>
<tr>
<td>(fifth grade only)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>School attachment</td>
<td>678</td>
<td>3.47</td>
<td>0.50</td>
<td>1.60 – 4.00</td>
<td>≤ 3.20</td>
<td>216</td>
<td>31.85%</td>
</tr>
<tr>
<td>(sixth grade only)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean peer victimization</td>
<td>693</td>
<td>1.81</td>
<td>0.68</td>
<td>1.00 – 5.00</td>
<td>≥ 2.13</td>
<td>201</td>
<td>29.00%</td>
</tr>
<tr>
<td>(third &amp; fifth grades)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean loneliness</td>
<td>698</td>
<td>27.09</td>
<td>7.57</td>
<td>16.00 – 54.50</td>
<td>≥ 31.50</td>
<td>184</td>
<td>26.36%</td>
</tr>
<tr>
<td>(third &amp; fifth grades)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 7. Correlations among the Continuous Extrafamilial Stressors

<table>
<thead>
<tr>
<th>Variable</th>
<th>Maternal report of neighborhood safety (third &amp; fifth grades)</th>
<th>Child report of neighborhood safety (third grade only)</th>
<th>Child’s feelings toward school (fifth grade only)</th>
<th>School attachment (sixth grade only)</th>
<th>Mean peer victimization (third &amp; fifth grades)</th>
<th>Mean loneliness (third &amp; fifth grades)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal report of neighborhood safety (third &amp; fifth grades)</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child report of neighborhood safety (third grade only)</td>
<td>.30***</td>
<td>--</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child’s feelings toward school (fifth grade only)</td>
<td>.11**</td>
<td>.18***</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>School attachment (sixth grade only)</td>
<td>.13***</td>
<td>.11**</td>
<td>.32***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean peer victimization (third &amp; fifth grades)</td>
<td>-.19***</td>
<td>-.31***</td>
<td>-.26***</td>
<td>-.23***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean loneliness (third &amp; fifth grades)</td>
<td>-.20***</td>
<td>-.37***</td>
<td>-.40***</td>
<td>-.28***</td>
<td>.43***</td>
<td></td>
</tr>
</tbody>
</table>

Note: ** = p < .01, *** = p < .001
Table 8. Correlations between Continuous Allostatic Load Variables and Continuous Family Risk Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Income-to-needs ratio</th>
<th>Maternal education</th>
<th>Partner status</th>
<th>Minority status</th>
<th>Home chaos</th>
<th>Maternal depressive symptoms</th>
<th>Home/Family Stress Index (0 – 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index (kg/m²)</td>
<td>.20***</td>
<td>.13***</td>
<td>.13***</td>
<td>.09*</td>
<td>.06</td>
<td>.10*</td>
<td>.21***</td>
</tr>
<tr>
<td>Waist-to-hip ratio (cm)</td>
<td>.07</td>
<td>.10**</td>
<td>.05</td>
<td>.01</td>
<td>.05</td>
<td>.04</td>
<td>.10**</td>
</tr>
<tr>
<td>Total skin fold (mm)</td>
<td>.14***</td>
<td>.10**</td>
<td>.07</td>
<td>.04</td>
<td>.04</td>
<td>.08*</td>
<td>.13***</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>.03</td>
<td>-.03</td>
<td>.05</td>
<td>.05</td>
<td>.00</td>
<td>.02</td>
<td>.03</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>-.05</td>
<td>-.03</td>
<td>.01</td>
<td>-.01</td>
<td>-.05</td>
<td>-.01</td>
<td>-.04</td>
</tr>
<tr>
<td>Cortisol (µ/dl)</td>
<td>-.04</td>
<td>-.01</td>
<td>-.01</td>
<td>-.01</td>
<td>-.04</td>
<td>-.02</td>
<td>-.03</td>
</tr>
<tr>
<td>Allostatic Load Index (0 – 5)</td>
<td>.11**</td>
<td>.08*</td>
<td>.09*</td>
<td>.05</td>
<td>.01</td>
<td>.07†</td>
<td>.13***</td>
</tr>
</tbody>
</table>

Note: + p = .052, * = p < .05, ** = p < .01, *** = p < .001
Table 9. Correlations between Continuous Allostatic Load Variables and Continuous Extrafamilial Risk Variables

<table>
<thead>
<tr>
<th></th>
<th>Mother report of neighborhood safety</th>
<th>Child report of neighborhood safety</th>
<th>Feelings about school</th>
<th>School attachment</th>
<th>Victimization by peers</th>
<th>Loneliness</th>
<th>Extrafamilial stress index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass Index (kg/m²)</td>
<td>-.17***</td>
<td>-.13***</td>
<td>-.07</td>
<td>-.06</td>
<td>.08*</td>
<td>.07</td>
<td>.11**</td>
</tr>
<tr>
<td>Waist-to-hip ration (cm)</td>
<td>-.06</td>
<td>-.05</td>
<td>-.13***</td>
<td>-.10*</td>
<td>.03</td>
<td>.00</td>
<td>.07</td>
</tr>
<tr>
<td>Total Skinfold (mm)</td>
<td>-.11**</td>
<td>-.12**</td>
<td>.00</td>
<td>.04</td>
<td>.07</td>
<td>.08*</td>
<td>.06</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>-.03</td>
<td>.02</td>
<td>-.14***</td>
<td>-.10*</td>
<td>-.01</td>
<td>-.04</td>
<td>.01</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>-.02</td>
<td>.01</td>
<td>.04</td>
<td>-.03</td>
<td>-.03</td>
<td>-.01</td>
<td>.02</td>
</tr>
<tr>
<td>Cortisol (µ/dl)</td>
<td>.12**</td>
<td>.01</td>
<td>.06</td>
<td>.04</td>
<td>.00</td>
<td>.04</td>
<td>-.02</td>
</tr>
<tr>
<td>AL Index (0-5)</td>
<td>-.10*</td>
<td>-.04</td>
<td>-.12**</td>
<td>-.04</td>
<td>.03</td>
<td>.10</td>
<td>.05</td>
</tr>
</tbody>
</table>

* = p < .05, ** = p < .01, *** = p < .001
Table 10. Hierarchical Regression Analysis Examining the Relationship of Chronic Stress (during Middle Childhood) to Allostatic Load at Age 15

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Step 1</th>
<th></th>
<th>Step 2</th>
<th></th>
<th>Step 3</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$B$</td>
<td>$\beta$</td>
<td>$B$</td>
<td>$\beta$</td>
<td>$B$</td>
<td>$\beta$</td>
</tr>
<tr>
<td>Sex</td>
<td>.37</td>
<td>.13***</td>
<td>.37</td>
<td>.13***</td>
<td>.37</td>
<td>.13***</td>
</tr>
<tr>
<td>Home/Family Risk Index</td>
<td>--</td>
<td>--</td>
<td>.11</td>
<td>.12**</td>
<td>.03</td>
<td>.03</td>
</tr>
<tr>
<td>Extrafamilial Risk Index</td>
<td>--</td>
<td>--</td>
<td>.02</td>
<td>.02</td>
<td>.10</td>
<td>.11</td>
</tr>
<tr>
<td>Sex x Home/Family (centered)</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>.16</td>
<td>.11*</td>
</tr>
<tr>
<td>Sex x Extrafamilial Risk Index (centered)</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>-.16</td>
<td>-.11*</td>
</tr>
<tr>
<td>Adjusted $R^2$</td>
<td>.02</td>
<td></td>
<td>.03</td>
<td></td>
<td>.04</td>
<td></td>
</tr>
<tr>
<td>$R^2$ change</td>
<td>.02</td>
<td></td>
<td>.02</td>
<td></td>
<td>.01</td>
<td></td>
</tr>
<tr>
<td>$F$ change</td>
<td>12.63***</td>
<td></td>
<td>5.70**</td>
<td></td>
<td>2.91*</td>
<td>$p = .055$</td>
</tr>
</tbody>
</table>

Note: * = $p < .05$, ** = $p < .01$, *** = $p < .001$
Table 11. Hierarchical Regression Analysis Examining the Relationship of Chronic Stress to Allostatic Load and Testing Maternal Sensitivity as a Moderator of that Relationship

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Step 1</th>
<th></th>
<th>Step 2</th>
<th></th>
<th>Step 3</th>
<th></th>
<th>Step 4</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>β</td>
<td>B</td>
<td>β</td>
<td>B</td>
<td>β</td>
<td>B</td>
<td>β</td>
</tr>
<tr>
<td>Sex</td>
<td>.38</td>
<td>.14**</td>
<td>.37</td>
<td>.13**</td>
<td>.37</td>
<td>.13**</td>
<td>.39</td>
<td>.14***</td>
</tr>
<tr>
<td>Mean Maternal Sensitivity</td>
<td>.03</td>
<td>.04</td>
<td>.02</td>
<td>.03</td>
<td>.02</td>
<td>.03</td>
<td>.02</td>
<td>.03</td>
</tr>
<tr>
<td>Home/Family Risk Index</td>
<td>--</td>
<td>--</td>
<td>.11</td>
<td>.11**</td>
<td>.11</td>
<td>.11**</td>
<td>.10</td>
<td>.11*</td>
</tr>
<tr>
<td>Extrafamilial Risk Index</td>
<td>--</td>
<td>--</td>
<td>.02</td>
<td>.02</td>
<td>.02</td>
<td>.02</td>
<td>.03</td>
<td>.03</td>
</tr>
<tr>
<td>Mean Maternal Sensitivity (centered) x Home/Family (centered)</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>.00</td>
<td>.01</td>
<td>.03</td>
<td>.08</td>
</tr>
<tr>
<td>Mean Maternal Sensitivity (centered) x Extrafamilial Risk Index (centered)</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>.00</td>
<td>.01</td>
<td>.01</td>
</tr>
<tr>
<td>Sex x Maternal Sensitivity x Home/Family</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>-.06</td>
<td>-.10</td>
</tr>
<tr>
<td>Sex x Maternal Sensitivity x Extrafamilial</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>.01</td>
<td>.02</td>
</tr>
<tr>
<td>Adjusted R²</td>
<td>.02</td>
<td>.03</td>
<td>.02</td>
<td>.02</td>
<td>.02</td>
<td>.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R² change</td>
<td>.02</td>
<td>.02</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F change</td>
<td>5.28**</td>
<td>4.10**</td>
<td>.02</td>
<td>1.08</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: * = p < .05, ** = p < .01, *** = p < .001
Table 12. Hierarchical Regression Analysis Examining the Relationship of Chronic Stress (during Middle Childhood) to Obesity Index at Age 15

<table>
<thead>
<tr>
<th>Block 1</th>
<th>B</th>
<th>SE</th>
<th>t</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>.24</td>
<td>.08</td>
<td>.12**</td>
<td>3.11</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Block 2</th>
<th>B</th>
<th>SE</th>
<th>t</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>.23</td>
<td>.08</td>
<td>.11**</td>
<td>3.07</td>
</tr>
<tr>
<td>Home/Family stress</td>
<td>.13</td>
<td>.03</td>
<td>.18***</td>
<td>4.51</td>
</tr>
<tr>
<td>Extrafamilial stress</td>
<td>.01</td>
<td>.03</td>
<td>.02</td>
<td>.37</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Block 3</th>
<th>B</th>
<th>SE</th>
<th>t</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>.23</td>
<td>.08</td>
<td>.11**</td>
<td>3.10</td>
</tr>
<tr>
<td>Home/Family stress</td>
<td>.07</td>
<td>.04</td>
<td>.10</td>
<td>1.72</td>
</tr>
<tr>
<td>Extrafamilial stress</td>
<td>.06</td>
<td>.04</td>
<td>.08</td>
<td>1.46</td>
</tr>
<tr>
<td>Home/Family stress (centered) x Sex</td>
<td>.11</td>
<td>.06</td>
<td>.11*</td>
<td>1.98</td>
</tr>
<tr>
<td>Extrafamilial stress (centered) x Sex</td>
<td>-.09</td>
<td>.06</td>
<td>-.09</td>
<td>1.61</td>
</tr>
</tbody>
</table>

Note: * = p < .05, ** = p < .01, *** = p < .001
Table 13. Hierarchical Regression Analysis Examining the Relationship of Chronic Stress (during Middle Childhood) to Obesity Index at Age 15

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SEB</th>
<th>β</th>
<th>t</th>
<th>ΔR²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Block 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.02</td>
</tr>
<tr>
<td>Sex</td>
<td>.24</td>
<td>.09</td>
<td>.12**</td>
<td>2.73</td>
<td></td>
</tr>
<tr>
<td>Mean Maternal sensitivity</td>
<td>.02</td>
<td>.02</td>
<td>.04</td>
<td>.81</td>
<td></td>
</tr>
<tr>
<td>Block 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.03</td>
</tr>
<tr>
<td>Sex</td>
<td>.23</td>
<td>.09</td>
<td>.11**</td>
<td>2.71</td>
<td></td>
</tr>
<tr>
<td>Mean Maternal sensitivity</td>
<td>.01</td>
<td>.02</td>
<td>.02</td>
<td>.40</td>
<td></td>
</tr>
<tr>
<td>Home/Family stress</td>
<td>.13</td>
<td>.03</td>
<td>.18***</td>
<td>3.85</td>
<td></td>
</tr>
<tr>
<td>Extrafamilial stress</td>
<td>.01</td>
<td>.03</td>
<td>.03</td>
<td>.35</td>
<td></td>
</tr>
<tr>
<td>Block 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.00</td>
</tr>
<tr>
<td>Sex</td>
<td>.24</td>
<td>.09</td>
<td>.12**</td>
<td>2.72</td>
<td></td>
</tr>
<tr>
<td>Mean Maternal sensitivity</td>
<td>.01</td>
<td>.02</td>
<td>.01</td>
<td>.29</td>
<td></td>
</tr>
<tr>
<td>Home/Family stress</td>
<td>.13</td>
<td>.03</td>
<td>.18***</td>
<td>3.80</td>
<td></td>
</tr>
<tr>
<td>Extrafamilial stress</td>
<td>.01</td>
<td>.03</td>
<td>.02</td>
<td>.43</td>
<td></td>
</tr>
<tr>
<td>Home/Family stress (centered) x Mean Maternal Sensitivity (centered)</td>
<td>-.08</td>
<td>.20</td>
<td>-.02</td>
<td>-.40</td>
<td></td>
</tr>
<tr>
<td>Extrafamilial stress (centered) x Mean Maternal Sensitivity (centered)</td>
<td>.13</td>
<td>.18</td>
<td>.03</td>
<td>.72</td>
<td></td>
</tr>
</tbody>
</table>

Note: * = p < .05, ** = p < .01, *** = p < .001
APPENDIX B

FIGURES

Figure 1. N at Each Level of AL

![Bar chart showing N at each level of AL.](image)

N = 699

Figure 2. N at Each Level of Home/Family Stress

![Bar chart showing N at each level of Home/Family Stress.](image)

N = 699
Figure 3. N at Each Level of Extrafamilial Stress

\[ N = 699 \]

Figure 4. Relationship between Home/Family Stress and Allostatic Load

\[ N = 699 \]
Figure 5. Relationship between Extrafamilial Stress and Allostatic Load

N = 699