Longitudinal Associations Between Early Parenting and Adolescent Allostatic Load: Examining

the Mediating and Moderating Role of Child Delay of Gratification

by

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A dissertation accepted and approved in partial fulfillment of the

requirements for the degree of

Doctor of Philosophy

In Prevention Science

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Summer 2023

DISSERTATION ABSTRACT

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Doctor of Philosophy in Prevention Science

Title: Longitudinal Associations Between Early Parenting and Adolescent Allostatic Load:
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Allostatic load (AL) is a representation of chronic wear and tear on the body due to prolonged exposure to stress. AL measures (e.g., blood pressure, cortisol) capture stress-related dysregulation across multiple physiological systems, which in turn contributes to future disease outcomes. Early contextual influences, such as parenting behaviors, can have a significant impact on the body's regulatory systems during younger years, with downstream effects on health outcomes later in development, making AL an important outcome to examine in relation to early parenting behaviors. This study utilized longitudinal data ($N = 1,364$) from the Study of Early Child Care and Youth Development (SECCYD) to examine the relationships between three early childhood parenting behaviors (i.e., supportive presence, respect for autonomy, and hostility) assessed at child age 24 and 36 months and adolescent AL, assessed at age 15. Further, given that early parenting behaviors can influence child self-regulation, and individual differences in self-regulation are linked to AL indicators, this study examined the role of delay of gratification, a behavioral measure of child self-regulation, as a mediator and moderator of the association between early parenting and adolescent AL. Analyses revealed that two early childhood parenting behaviors (i.e., supportive presence and hostility) were directly associated with adolescent AL, and all three parenting behaviors were associated with childhood delay of gratification. However, there was no significant association between childhood delay of gratification and adolescent AL, and childhood delay of gratification was not a significant mediator or moderator of the associations between early parenting behaviors and adolescent AL.

Future research should further examine the role of self-regulation using more comprehensive assessments, repeated over time from early childhood to adolescence. Other potential mediators and moderators (e.g., lifestyle factors) should also be examined to understand the mechanisms by which early parenting impacts AL in later years, and identify individuals who may be most at-risk for high AL.

ACKNOWLEDGMENTS

I would like to formally thank Dr. Atika Khurana for her mentorship throughout my graduate education. She has taught me how to be a better teacher, a better writer, and a better person. I hope I can shape the lives of future students the way she has shaped mine.

DEDICATION

For Doug, for always standing by me while I find my way, and being there for every step. I love you more. For my kids, your love has carried me in ways you will never know. Finally, for my dad, who taught me that I am smart enough to achieve whatever I set my mind to.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER 1

INTRODUCTION

Allostatic load (AL), broadly defined as chronic wear and tear on the body due to prolonged exposure to stress, has been linked to both physical and mental health problems, including cardiovascular disease, diabetes, anxiety, depression, and post-traumatic stress disorder (Doan, 2021). Unlike individual markers of stress-related dysregulation, AL combines several biomarkers of stress-related dysregulation into one composite, thus capturing dysregulation across multiple biological systems (i.e., neuroendocrine, metabolic, cardiovascular, and immune systems). As such, AL is regarded as a more reliable and better predictor of future morbidity and mortality than individual markers of stress-related dysregulation (Evans & Fuller-Rowell, 2013). Nevertheless, much of the research on AL has been conducted in adult populations with limited focus on younger years (Gruenewald et al., 2006). Recent studies have shown that AL can be measured reliably in children as young as 9-years-old (Doan, 2021), and childhood AL is predictive of long-term health outcomes, including cognitive functioning, behavioral problems, as well as future diabetes and hypertension (Brody et al., 2016; Doan, 2021). Given its role as a critical marker for early identification and prevention of chronic disease, it is important to understand how early contextual influences predict AL later in development. To that end, the present study examined how parenting behaviors in early childhood are associated with AL in adolescence using longitudinal data from the National Institute of Child and Human Development (NICHD) Study of Early Child Care and Youth Development (SECCYD) study.

Early parenting can play an influential role in shaping long-term health outcomes (Flaherty & Sadler, 2011) by promoting or inhibiting the development of healthy attachment (Feldman, 2010), self-regulation (Bernier et al., 2010), and physiological systems (e.g.,

hypothalamic-pituitary-adrenocortical, or HPA, axis) associated with stress regulation (Roisman et al., 2009). Indeed, families provide the earliest social relationships in life, contributing profoundly to the "competence, resilience, and well-being" of children (Basic Behavioral Science Task Force of the National Advisory Mental Health Council, 1996, p. 81). Both positive (e.g., parental sensitivity, emotional support) and negative parenting behaviors (e.g., intrusiveness, hostility), have been linked to behavioral and emotional health outcomes in children (Belsky et al., 2007; Feldman, 2010). Further, there is moderate stability in parenting behaviors from early childhood to adolescence (Else-Quest et al., 2011; Wang et al., 2013), indicating that patterns of early parenting may be enduring with lasting impact on child health outcomes. Parenting interventions, especially in early childhood, can be effective in promoting healthy parent-child relationships and child self-regulation (Bernier et al., 2010; Kok et al., 2022; Moilanen et al., 2018). To the extent that early parenting also influences adolescent AL, interventions designed to promote specific parenting behaviors in younger years can have a beneficial impact on future AL and health outcomes.

Prior research has documented associations between early parenting behaviors and AL markers later in development. For example, using the SECCYD data, one study found that low levels of maternal sensitivity assessed in the first three years of life were associated with lower adolescent awakening cortisol levels at age 15, indicative of stress-related dysregulation (Roisman et al., 2009). Using the same sample, Boyer and Nelson (2015) found that maternal and paternal sensitivity (assessed at 54 months and grade 1) was associated with lower blood pressure and higher awakening cortisol at age 15. Further, this relationship was fully mediated by increases in child social competence from grade 1 to grade 6.

Other studies using non-SECCYD samples have also reported parenting effects on AL markers. For instance, in a large cross-sectional study of nearly 5,000 middle and high school students, male adolescents who reported high maternal authoritarian parenting (defined as low responsiveness and high demandingness) and high paternal neglectful parenting (low responsiveness and low demandingness) had higher BMI scores, while female adolescents who reported their fathers modeling and encouraging healthy practices (e.g., healthy dietary intake and physical activity) had lower BMI scores (Berge et al., 2010). A recent systematic review also found that across studies, negative parenting behaviors (i.e., child maltreatment and harsh parenting) are associated with higher AL in adulthood, while positive forms of parenting (i.e., parenting warmth and emotional support) are associated with lower AL in adulthood (Guidi et al., 2021). However, much of prior research has either examined these associations cross-sectionally, or assessed early parenting using retrospective reports which can be inaccurate due to recall bias (Misiak et al., 2022). Only a few studies have examined the associations between early parenting and adolescent AL using longitudinal samples, with objective assessments of early parenting (e.g., Boyer & Nelson, 2015; Roisman et al., 2009), but these studies have tested early parenting effects on specific AL biomarkers (e.g., blood pressure, awakening cortisol) and not more comprehensive AL assessments that capture dysregulation across multiple physiological systems.

The present study advances current understanding of early parenting effects on adolescent AL by using the longitudinal SECCYD data to (a) examine the longitudinal associations between specific early parenting behaviors (assessed using observational coding of mother-child interactions) and adolescent AL, assessed using multiple biomarkers, and (b) assess the role of childhood delay of gratification – a behavioral measure of self-regulation – as a

potential mediator and moderator of early parenting associations with adolescent AL. Understanding how specific parenting behaviors in early childhood can impact adolescent AL and the underlying mechanisms of influence can enable the development of more targeted parenting interventions that can offset the risk for high AL later in development.

Allostatic Load

Allostasis represents the body's adaptive mechanism to maintain homeostasis during periods of stress (Beckie, 2012). AL theory suggests that exposure to chronic stress triggers physiological changes in the body that can lead to long term negative health outcomes (Carbone et al., 2022). More specifically, chronic stress exposure can lead to dysregulation of the HPA axis – the self-regulating system in the body responsible for managing how the body responds to stress (Boyer & Nelson, 2015). The HPA axis controls the primary stress hormones in the body (i.e., cortisol, epinephrine, norepinephrine, and DHEA-S). Repeated activation and deactivation of this mechanism due to acute and/or chronic stress exposure can cause wear and tear on physiological systems within the body, contributing to downstream dysregulation of multiple body systems, and subsequent disease or illness (McEwen & Stellar, 1993). Specifically, the primary dysregulation of the HPA axis, over time, leads to biological changes at the cellular level, contributing to secondary dysregulation of the inflammatory, metabolic, and cardiovascular systems, resulting in changes in biomarkers such as blood pressure, cholesterol, and glucose levels. This, in turn, contributes to tertiary physical disease outcomes and mental health disorders (Carbone et al., 2022; Juster et al., 2010).

In recent years, AL has gained popularity as an effective measure to assess the effects of chronic stress in both children and adults (Evans & Fuller-Rowell, 2013). Given that the AL composite includes biomarkers of dysregulation across multiple biological systems

(neuroendocrine, metabolic, cardiovascular, and immune systems), it provides a more comprehensive assessment of stress-related dysregulation and is a more robust predictor of future health outcomes than individual biomarkers like BMI or blood pressure (Evans & Fuller-Rowell, 2013). Common biomarkers used to assess AL include heart rate, systolic blood pressure, diastolic blood pressure, hemoglobin A1c, cholesterol, waist-to-hip ratio, cortisol, and body mass index (Carbone et al., 2022). However, there is no clear consensus on which biomarkers are most relevant to include in an AL composite and which ones are developmentally appropriate for younger populations (Beckie, 2012). A recent review of 395 studies highlighted the wide variety of biomarkers that are utilized to calculate AL, with most studies including 6-14 biomarkers (Carbone et al., 2022). In particular, heart rate, systolic blood pressure, diastolic blood pressure, hemoglobin A1c, HDL cholesterol, C-reactive protein, waist-to-hip ratio, cortisol, body mass index, and total cholesterol were used in at least half of the studies included in the review, with an additional 30 biomarkers utilized less frequently. In another review study focused on adolescent AL, the most commonly used biomarkers included cortisol, epinephrine, norepinephrine, systolic blood pressure, diastolic blood pressure, and body mass index (Whelan et al., 2021).

Since stress hormones are thought of as primary markers of dysregulation, including at least one primary stress hormone in the assessment of AL is recommended (Whelan et al., 2021). Cortisol, the end product of the HPA axis, is one of the most commonly used biomarkers in AL assessments with children (Doan, 2021). In addition to utilizing stress hormones as biomarkers for primary dysregulation, it is important to include biomarkers indicative of secondary changes (e.g., blood pressure, cholesterol, BMI), which could lead to tertiary disease outcomes like cardiovascular disease (Carbone et al., 2022).

A factor analysis of AL measures by King et al. (2019) found that some of the best indicators to use for adolescent-specific AL include those that measure metabolic dysregulation (e.g., BMI, waist circumference, waist to hip ratio), suggesting that these measures may reflect the earliest clinical signs of elevated AL in adolescents. Additionally, previous research with pediatric populations emphasizes the importance of using age-specific anthropometric measures (e.g., BMI) due to the association of these measures with AL in early life (Calcaterra et al., 2019). Thus, utilizing a variety of biomarkers from multiple biological systems, including a primary stress hormone and measures of metabolic dysregulation, is important when assessing adolescent AL.

There is also no clear consensus in the field about designating high-risk criteria for AL markers. The risk criteria cut offs are more clearly defined for some measures (e.g., BMI) but not for others (e.g., waist to hip ratio). Majority of studies have used a high-risk quartile approach in which the sample distribution for any particular biomarker data is divided into quartiles and a "high risk" quartile is determined, typically the highest or lowest quartile, depending on the biomarker. For instance, the top quartile has been used as the risk cut-off for waist-to-hip ratio, whereas the lowest quartile is typically used as the risk cut-off for awakening salivary cortisol because in healthy children and adults, cortisol levels are supposed to be the highest in the first 30-40 minutes after awakening, gradually tapering off through the afternoon (Rogosch et al., 2011). Instead of quartiles, some studies have used the top or bottom 10% of the distribution, representing the group at highest risk, in order to capture the most clinically significant levels for some biomarkers such as blood pressure (Goldman et al., 2006; Hwang et al., 2014; Seeman et al., 1997). Additionally, when available, researchers have also used established clinical cutoffs to define high-risk (Carbone et al., 2022). For instance, BMI levels $\geq 85^{th}$ percentile for child age

and sex are commonly used as the high-risk cutoff, based on current guidelines for adolescent overweight or obesity (Centers for Disease Control and Prevention, 2022). Despite the different approaches used to define "high risk" cutoffs in AL assessment, measurements of AL are found to be reliable and valid predictors of future health outcomes (Evans & Fuller-Rowell, 2013).

Parenting Behaviors and Adolescent AL

The biological embedding of childhood adversity model proposes that early childhood exposure to stressors (e.g., parental hostility, neglect) becomes embedded in molecular pathways through frequent activation of stress hormones, exacerbating inflammatory responses already programmed into cells of the body, which contributes to chronic disease over time (Berens et al., 2017; Miller et al., 2011). In particular, chronic interpersonal stressors early in life, such as unsupportive parenting and maltreatment, can contribute to frequent and chronic activation of the stress response system leading to negative health outcomes (Blair et al., 2011; Miller et al., 2011; Roisman et al., 2009). This biological dysregulation beginning at an early age can lead to cascading negative mental and physical health outcomes over time (Repetti et al., 2002).

Positive parenting behaviors, on the other hand, can lead to healthy development of the body's stress response and self-regulatory systems. For example, in an intervention study to promote positive parenting and maternal responsiveness, children (ages 1-3 years) of mothers who were in the intervention group had lower levels of basal cortisol one year later as compared to the control group (Bakermans-Kranenburg, 2008). In another longitudinal study, where young adults (N = 756) retrospectively reported on parenting during childhood and adolescence, higher parental warmth, affection, and bonding was significantly associated with lower AL in adulthood (Carroll et al., 2013). Other studies with adult samples, using retrospective reports of parenting during childhood and adolescence, have similarly reported protective associations between

maternal warmth and emotional support, and multiple indicators of biological dysregulation (e.g., high blood pressure, impaired glucose control, abdominal adiposity, and lipid dysregulation) in adulthood (Slopen et al., 2016). Thus, there is evidence, though primarily from adult samples, that early parenting (positive and negative) can impact AL indicators across different physiological systems. There is also some evidence to suggest that the effects of early parenting on later AL may be mediated by parenting effects on child self-regulation (Compas, 2006; Roisman et al., 2009), however this mechanistic pathway has not been tested using longitudinal samples.

Parenting Behaviors and Child Delay of Gratification

There is a wealth of research documenting early parenting effects on the development of child self-regulation (Belsky et al., 2007; Chang et al., 2015; Landry et al., 2002). Self-regulation is broadly defined as the conscious regulation of one's thoughts, emotions, and behaviors in the service of a goal (Masten et al., 2010). Self-regulation develops rapidly during early childhood years, with heightened sensitivity to environmental impacts, including early parenting behaviors (Bernier et al., 2010; Fay-Stammbach et al., 2014). In general, positive parenting behaviors (e.g., supportive and sensitive parenting) have been linked to increases in self-regulation in early childhood years (Kok et al., 2022), while negative parenting behaviors (e.g., hostility) have been associated with self-regulation difficulties (Moilanen et al., 2010). Social cognitive theories posit that supportive parenting promotes children's self-regulation through respect for child's autonomy, positive emotional support and responsiveness, and modeling of emotional and behavioral regulation. Over time and with practice, children become better at regulating their own emotions and behaviors. Harsh parenting, on the other hand, undermines the development of self-regulation by making it harder for the child to regulate in relational contexts characterized

by heightened emotional stress, unpredictability, intrusiveness, and lack of emotional support and sensitivity to child's needs and autonomy (Fay-Stammbach et al., 2014). Parents can thus influence children's development of self-regulation through direct support of children's developing regulatory capacities, modeling well-regulated behaviors, and creating a predictable, emotionally secure, nurturing environment which facilitates the development of self-regulation.

Self-regulation is a multidimensional construct that has been assessed using a variety of measures across cognitive (e.g., executive functions), behavioral (e.g., delay of gratification, impulsivity), emotional (e.g., emotional reactivity/regulation), and temperamental (e.g., effortful control) domains. This study utilizes delay of gratification, a behavioral measure of self-regulation, commonly used with young children. Delay of gratification is typically assessed using a choice paradigm in a rewarding context. For instance, in the famous marshmallow task (Mischel & Ebbesen, 1970), children's delay of gratification is assessed by their ability to not give into the urge to have the instant reward of one marshmallow and instead wait for the larger reward of two marshmallows after a fixed delay (Duckworth, Tsukayama, et al., 2013).

Specific parenting behaviors such as supportive presence, defined as the parent's level of positive regard and encouragement of their child's efforts, are found to be positively associated with child delay of gratification. One cross-sectional study in preschool children ages 4-6 years (N = 102) found that higher self-reported maternal warmth and support was associated with better child delay of gratification (Von Suchodoletz et al., 2011). Another cross-sectional study in preschool children at age 3 (N = 258) found that greater maternal supportive presence (assessed using an observational task) was associated with better child delay of gratification (Kok et al., 2022). A longitudinal study using SECCYD data found that maternal sensitivity

(which included maternal supportive presence) assessed in the first three years of life is linked to greater delay of gratification at age 54 months (Razza & Raymond, 2013).

Another positive parenting behavior that has consistently been linked to child self-regulation is respect for autonomy (Bernier et al., 2010). This parenting behavior reflects respect and support of the child's individuality, allowing the child to actively participate in problem-solving and completing tasks, with less parental intrusion. Using the SECCYD data, Bindman et al. (2015) found that maternal autonomy support in the first three years of life was positively associated with child's ability to delay gratification at 54 months. Further, in a different longitudinal sample, Bernier et al (2010) found that maternal respect for autonomy at 15 months of age was predictive of stronger child executive functions (associated with self-regulation) at both 18 months and 26 months of age.

Negative forms of parenting, such as hostility (reflecting rejection and blaming the child for mistakes), model dysregulated behaviors for the child, compromising the child's ability to develop self-regulatory skills (Moilanen et al., 2018). Previous research has found that parental hostility can create a negative coercion cycle in which harsh parenting leads to poor child self-regulation, which in turn leads to more harsh parenting, continuing to impede the development of child self-regulation over time (Scaramella & Leve, 2004). In a longitudinal study of early adolescents (age 12), harsh parenting (assessed at age 12) was associated with lower adolescent self-regulation (assessed concurrently and 1 year later) while nurturing, responsive parenting was associated with higher self-regulation (Brody & Ge, 2001). In another study including 109 children from predominantly white, middle- to high-income families, maternal lack of responsiveness in early childhood (ages 1-3 years) was associated with difficulties with delaying gratification at age 5 (Rodriguez et al., 2005). The findings reviewed above suggest that both

positive and negative parenting behaviors in early childhood are linked to the development of self-regulation, including delay of gratification. To the extent that childhood delay of gratification is linked to adolescent AL, it may be a potential mechanism by which early parenting impacts adolescent AL.

Mechanisms Linking Early Parenting to Adolescent AL

Given prior research demonstrating moderate stability of parenting behaviors from early childhood into adolescence (Else-Quest et al., 2011; Wang et al., 2013), examining early parenting behaviors in relation to adolescent AL allows for the earliest glimpse at how parenting behaviors may impact adolescent AL. There are two potential mechanisms through which early parenting can impact adolescent AL. First, parenting may directly influence adolescent AL through biological programming that happens in younger years through early attachment in supportive and nurturing relationships (Miller et al., 2011). For example, parenting behaviors that promote a stressful rather than supportive family environment can trigger alterations in the endocrine and autonomic nervous systems and subsequent chronic inflammation (Compas, 2006; Miller et al., 2011).

A second mechanism may be through the link between early parenting and child self-regulation (Moilanen & Rambo-Hernandez, 2017), such that individuals with better self-regulation may be able to handle stress more effectively (Evans & Fuller-Rowell, 2013), leading to lower AL. Typically, individuals respond to stress by initiating coping mechanisms in an effort to maintain control over their emotions and behaviors, and regulate physiologic responses to the stressor (Compas, 2006). However, chronic cumulative stress can disrupt the HPA system responsible for managing that stress, compromising the ability to self-regulate (Evans, 2003; Evans & Kim, 2013).

Individual differences in self-regulation in childhood have been linked to specific biomarkers of AL (e.g., BMI) (Robson et al., 2020), making it an important mediator to consider in the relationship between early parenting and AL. Multiple longitudinal studies have found delay of gratification to be linked to AL outcomes such as overweight, obesity, BMI, and stress (Duckworth, Tsukayama, et al., 2013; Shoda et al., 1990). Using the SECCYD data, Duckworth, Tsukayama, et al., 2013 found that greater ability to delay gratification at age 4 was associated with a lower BMI at age 15. Using a different sample of similar ages, Shoda et al. (1990) found that greater delay of gratification in preschool is associated with better coping skills and lower stress levels in adolescence ten years later. Thus, delay of gratification may be an important mediator to examine in understanding the association between early parenting and adolescent AL. However, no study to date has examined this mechanistic pathway of influence using a longitudinal sample with objective assessments of early parenting and comprehensive AL composite.

Moderating Effects of Child Delay of Gratification

Individual differences in self-regulation can also operate as a moderator of contextual effects, such as early parenting, on later AL and health outcomes (Brody et al., 2013; Dich et al., 2015a). Children with better self-regulation may be able to handle stress more effectively and may be buffered against the negative impacts of hostile parenting (Kryski et al., 2013). There is also some research to suggest that the protective effects of positive parenting may be stronger for children with weakness in self-regulation, as they have more room to grow and can benefit more from supportive parenting in being able to manage their emotional reactions (Song et al., 2018). For instance, one study of 239 children found that negative emotionality associated with stress exposure (e.g., negative life events) at age nine predicted higher levels of AL at age 17, but only

for children who had lower levels of self-regulation (assessed using the delay of gratification task) (Dich et al., 2015a). Children who were better able to delay gratification were less likely to experience high AL associated with negative emotionality. In a cross-sectional study of 160 preschool children (age 3), negative parenting, assessed using an observational measure of both mothers and fathers, was associated with a stronger cortisol response in children when encountering a laboratory stressor, but only for children with lower ability to delay gratification (Kryski et al., 2013). Other review studies have similarly found that children who are low in effortful control, and high in impulsivity and frustration are more susceptible to adverse consequences of negative parenting behaviors, such as the association between parental control and externalizing behaviors (Kiff et al., 2011). Given these findings related to differential susceptibility, it is possible that children who have difficulty delaying gratification may experience stronger negative impacts of hostile parenting on adolescent AL, and conversely may benefit more from positive parenting, than children with stronger delay of gratification skills. Thus, this study examined whether delay of gratification operated as a moderator of the longitudinal associations between early parenting and adolescent AL.

Present Study

This study utilized longitudinal data from the NICHD SECCYD study to address the following research questions:

Research Question 1: Is there an association between early parenting behaviors (assessed at 2-3 years of age) and adolescent AL (assessed at age 15)?

 Hypothesis 1: Early parenting behaviors are expected to be significantly associated with adolescent AL. In particular, positive parenting behaviors (i.e., supportive presence and respect for autonomy) are expected to be negatively associated with adolescent AL, and negative

parenting behavior (i.e., hostility) is expected to be positively associated with adolescent AL. See
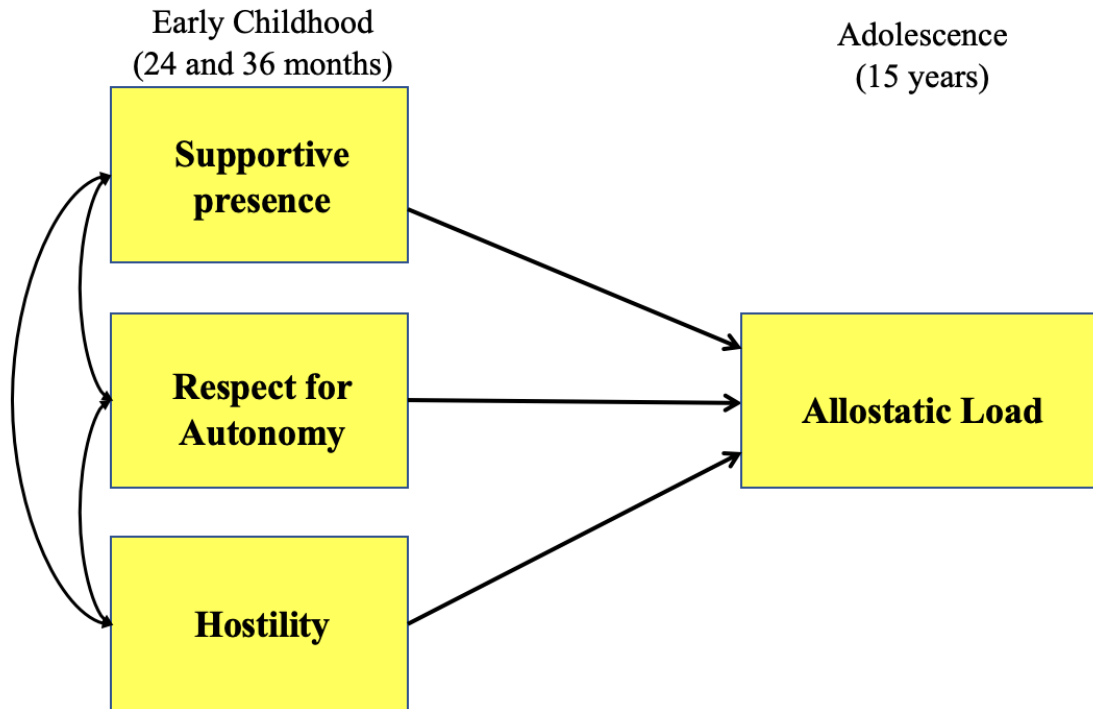
Fig. 1 for hypothesized model.

Figure 1. Hypothesized Model Examining Direct Effects of Early Parenting Behaviors on Allostatic Load (AL). Direct effects of early parenting behaviors on adolescent AL will be tested together in the same model as well as separately in individual models. The effects of the following covariates will be accounted for: child sex, child race/ethnicity, family income to needs ratio, maternal depressive symptoms, maternal negative life events, maternal education. These pathways are omitted for clarity.

Research Question 2: Does childhood delay of gratification (assessed at 54 months) mediate the

associations between early parenting behaviors and adolescent AL?

Hypothesis 2: Childhood delay of gratification is expected to be positively associated

with positive parenting behaviors (i.e., supportive presence, respect for autonomy) and

negatively associated with negative parenting behaviors (i.e., hostility). Further, delay of

gratification is expected to be negatively associated with adolescent AL, such that children with

better ability to delay gratification will have lower adolescent AL. Finally, childhood delay of gratification is expected to mediate the association between all three early parenting behaviors and adolescent AL. See Fig. 2 for hypothesized model.
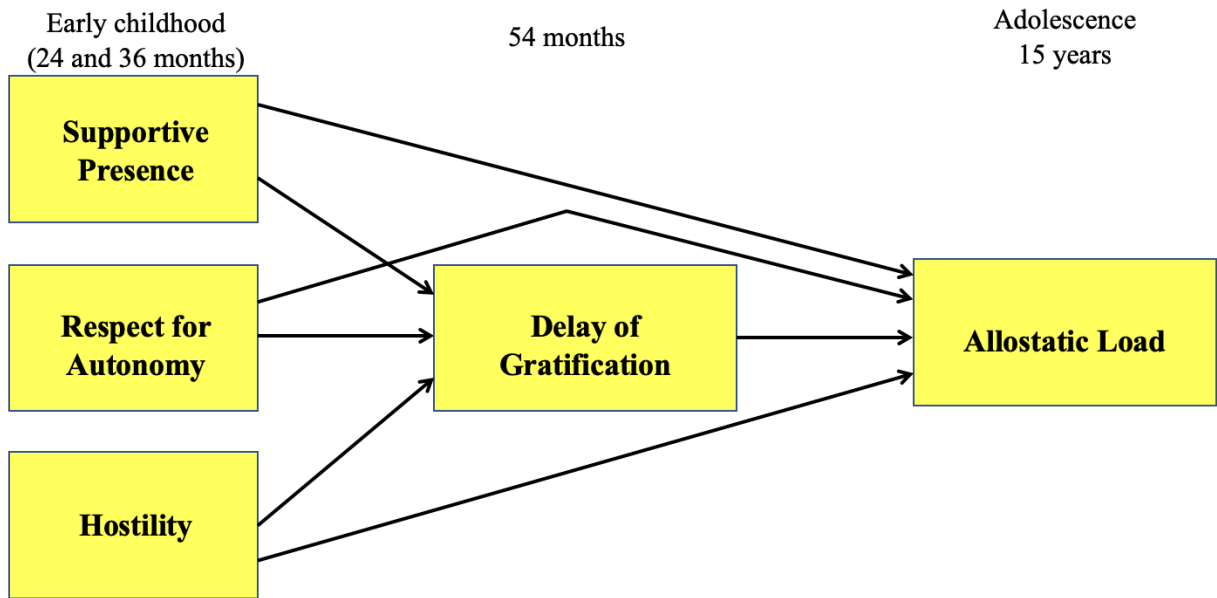
Figure 2. Hypothesized Model Examining Direct and Indirect Effects of Early Parenting Behaviors on Adolescent Allostatic Load as Mediated by Child Delay of Gratification. The effects of the following covariates on the mediator and outcome variables will be accounted for: child sex, child race/ethnicity, family income to needs ratio, maternal depressive symptoms, maternal negative life events, maternal education. These pathways are omitted for clarity. The direct and indirect effects will be tested in separate models for each of the three parenting behaviors.

Research Question 3: Does childhood delay of gratification moderate the associations between early parenting behaviors and adolescent AL?

Hypothesis 3: Childhood delay of gratification is expected to moderate the impact of early parenting behaviors on adolescent AL, such that among children who exhibit greater difficulties with delaying gratification, the protective effects of positive parenting behaviors, and

risk enhancing effects of negative parenting behaviors will be more pronounced as compared to

children who are better able to delay immediate gratification. See Fig. 3 for hypothesized model.
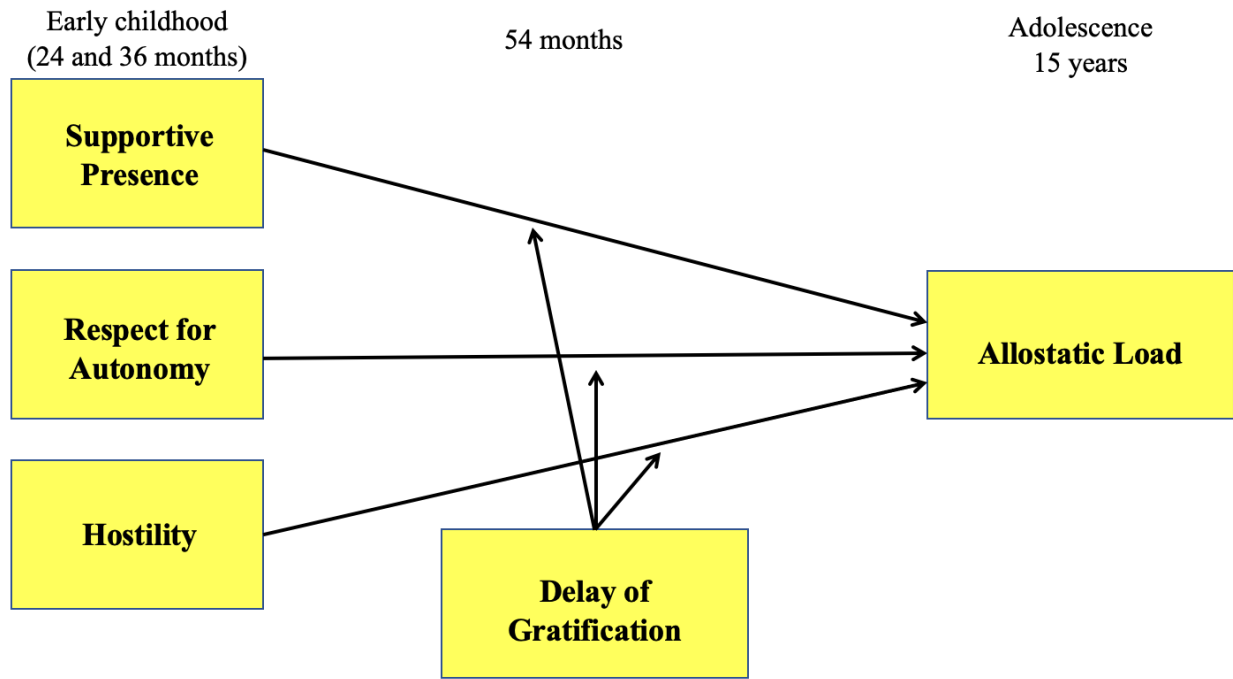
Figure 3. Hypothesized Model Examining the Moderating Effect of Child Delay of Gratification on the Association Between Early Parenting Behaviors and Adolescent Allostatic Load. The effects of the following covariates will be accounted for: child sex, child race/ethnicity, family income to needs ratio, maternal depressive symptoms, maternal negative life events, maternal education. These pathways are omitted for clarity. The interaction effects will be tested in separate models for each of the three parenting behaviors and child delay of gratification.

CHAPTER 2

METHODS

Sample Description

This study analyzed data from the SECCYD study, which was a four-phase longitudinal study conducted from 1991 to 2008 by the NICHD examining child development and childcare experiences from infancy (1-month) to mid-adolescence (age 15). This data set is uniquely suited to answer the current study questions for the following reasons: (1) it is a longitudinal study spanning 15 years, covering developmental periods from early childhood to adolescence, (2) it includes observational coding of parenting behaviors at multiple time points during development, (3) it includes task-based assessment of child self-regulation using the delay of gratification task at kindergarten age, when individual differences in child self-regulation can be reliably assessed (Whitebread, 2021) and have strong predictive utility (Montroy et al., 2016), and (4) it includes a comprehensive range of biomarkers for AL assessment that were assessed by trained research staff. Overall, not only does this data set contain all the variables of interest, assessed over time from early childhood to adolescence; it also includes highly objective, multi-method assessments of these variables.

Initially, 3,015 eligible mothers were recruited from 24 designated hospitals across 10 U.S. cities. To be included in the study, mothers had to be at least 18 years of age, English speaking, and have no reported history of substance abuse. In addition, their infant had to be healthy with no disease or disability at birth. The Phase I (1991-1994) sample included 1,364 mothers; 65% had a high school degree or more, 11% had not completed high school, 14% were from single-parent households. Infant demographics included 52% male and 80.4% non-

Hispanic White, 12.9% African American, 1.6% Asian, 0.4% American Indian/Eskimo/Aleutian, and 4.7% other.

Participating families and children were assessed on parenting and child outcomes of interest in this study, almost annually from birth to age 15. Phase I included assessments conducted from birth to 3 years. Phase II (1995-1999) included assessments conducted from age 3 through 1st grade (child age 7 years), Phase III (2000-2004) included assessments conducted from 2nd grade through 6th grade (child age 12 years), and Phase IV (2005-2007) included assessments conducted from 7th grade through 9th grade (child age 15 years). There was 26% attrition across the waves ($N = 1226$, 1061, and 1009 across the last three phases respectively). Attrition across the waves was higher for African American participants and those from lower SES (Wang et al., 2013). A detailed description of the data collection procedures and instruments can be found in the study manual (US Department of Health and Human Services, 2009). This study used data from all four phases. Assessments of early parenting behaviors were from Phase I and II; covariates were from Phase I, II, and III; childhood delay of gratification assessment was from Phase II; and adolescent AL assessments was from Phase IV of the study.

Measures

Predictor variables

Early parenting behaviors. Three parenting behaviors, namely supportive presence, respect for autonomy, and hostility, were assessed using observational coding of the semi-structured Mother–Child Interaction Task. The task was administered at the 10 data collection sites at multiple time points with task- and age-appropriate modifications. It included a variety of games and teaching activities that were potentially entertaining and frustrating, creating an environment requiring mother and child to collaborate and demonstrate teaching abilities,

learning abilities, and emotional regulation. The interactions were recorded and then coded at a central location using 7-point rating scales. Coders were blind to all information about the dyad. Inter-rater reliability was monitored throughout the coding period with intraclass correlations ranging from 0.84 to 0.91 (Wang et al., 2013). This study used the 24-month and 36-month assessments to capture parenting during the "early childhood" developmental stage. Earlier assessments of the mother-child interaction task were not included in the present study because the same parenting constructs (i.e., supportive presence, respect for autonomy, and hostility) were not assessed during infancy. For each parenting behavior, the two assessments (24 months and 36 months), coded on a scale from 1-7, were standardized and then averaged to create a continuous score.

Supportive presence reflected the mother's positive regard and emotional support for the child. High scores demonstrated mothers who acknowledged the child's accomplishments and provided encouragement and confirmation of the child's actions. Mothers low on this scale were passive, aloof, and uninvolved, rarely providing supportive cues to the child (Mintz et al., 2011).

Respect for autonomy was assessed based on mother's sensitivity, respect and support for the child's perspective and individuality (Bindman et al., 2015; Wang et al., 2013). High scores reflected mother's acknowledgment of their child's opinions and actions as an important part of the child's individuality. Low scores indicated intrusive interactions, denying the autonomy of the child, and not respecting the child as a partner in the interaction (Wang et al., 2013).

Hostility scores demonstrated mother's discounting or rejection of the child, or expression of anger towards the child. High scores indicated mothers who outwardly rejected the child, blamed the child for mistakes, or explicitly demonstrated that they did not support the child emotionally. Low scores reflected that, regardless of the level of support for the child, the

mother did not blame or reject the child, or make statements demonstrating a lack of emotional support for the child (Wang et al., 2013).

Given prior literature suggesting that concurrent/proximal parenting may be a stronger predictor of delay of gratification than early parenting (Conway, 2020), the effects of the 54-month assessments of these parenting behaviors on delay of gratification and AL, along with the early parenting assessments, were tested to evaluate both early and proximal parenting associations. Stability pathways were included in the model, such that any significant association of parenting at 54 months reflected unique effects of parenting at 54 months controlling for parenting in early childhood.

Outcome variable

Allostatic load (AL). A composite index for AL was created using the following seven variables assessed at age 15 by trained research staff: 1) systolic blood pressure, 2) diastolic blood pressure, 3) waist-to-hip ratio, 4) triceps skinfold measurement, 5) subscapular skinfold measurement, 6) BMI, and 7) awakening salivary cortisol. Measurement protocols for all variables were standardized across all study sites. Cut-off criteria for high risk levels of each variable were established based on prior research or using clinical cut-offs when available. Each variable was recoded as a binary variable (0 = high risk criteria not met, 1 = high risk criteria met). The score on the seven binary (0/1) variables was then added, resulting in a composite score ranging from 0-7. Due to a low number of scores in the higher categories, the upper category was collapsed to 5+, resulting in a range of 0-5 ($M = 1.48$, $SD = 1.56$).

Blood pressure. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) measurements were taken by trained research staff at lab visits at age 15. Blood pressure was measured via blood pressure cuff (DINAMAP Pro 100; GE Healthcare) and stethoscope five

times in the right arm while the adolescent was seated. Measures were taken at 1-minute intervals. The last three measures were averaged to create the average systolic blood pressure and average diastolic blood pressure. Values in the top decile of the sample distribution were considered meeting the high-risk cutoff according to previously published research (Goldman et al., 2006; Hwang et al., 2014; Seeman et al., 1997) and were coded as 1.

Waist-to-hip ratio. Two measurements each of waist circumference and hip circumference were taken by trained research staff using a flexible anthropometric tape applied without pressure. If both measurements were within 1.5 cm of each other, an average of the two measurements was taken. If the measurements differed by more than 1.5 cm, a third measurement was taken. If those two measurements were within 1.5 cm, an average of those two measurements was taken. If they were not within 1.5 cm of each other, an average was taken of the two closest measurements. After establishing the average waist circumference and average hip circumference, waist-to-hip ratio was calculated by dividing the average waist circumference by the average hip circumference. Values in the top quartile were used as the risk cut-off and were coded as 1.

Skinfold measurements. Trained research staff obtained measurements of the triceps skinfold and subscapular skinfold on the right side of the body using Lange Skinfold Calipers (Cambridge Scientific Industries, Inc). Of the three values, if the first two measurements were identical, that value was used. If the second two measurements were identical, that value was used. Otherwise, the two closest values of the three values were averaged and used as the respective skinfold measurement (triceps and subscapular). Values in the top quartile were used as the risk cut-off.

Body mass index (BMI). Adolescent height and weight were measured by trained research staff. BMI was calculated using the measured height and weight (BMI = weight (kg)/height (m)2). For body mass index (BMI), high risk-cut off was defined based on clinical guidelines of $\geq 85^{th}$ percentile for child age and sex, for adolescent overweight or obesity (Centers for Disease Control and Prevention, 2022). Thirty-one percent of the sample was coded in the high-risk category (i.e., $\geq 85^{th}$ percentile for child age and sex) for this variable.

Awakening salivary cortisol. Adolescents and their parents received instruction on proper protocol for saliva collection. Adolescents collected saliva samples upon awakening on three consecutive school days using Salivette (Sarstedt, Numbrecht, Germany). An average cortisol value was calculated from the three daily values collected. Because lower levels of awakening cortisol are indicative of severe or enduring stress levels, values in the lowest quartile were used for the risk cut-off (Roisman et al., 2009).

Mediator and Moderator Variable

Delay of gratification. At 54 months, children completed a modified version of the classic marshmallow test (Mischel & Ebbesen, 1970). Trained research staff presented the child with a treat and told them they would be alone with the treat for 7 minutes. The child was given the option to wait to eat the treat until the researcher returned, at which point they would receive an additional treat as a reward for waiting, or they could eat the treat before the researcher returned and not be rewarded with an additional treat. The number of seconds the child waited to eat the treat was recorded. Majority of the sample (53%) waited the full 7 minutes to eat the treat. Given the right censoring of distribution, the scores were re-coded to the following categories (<20 seconds, 20 seconds – 2 minutes, 2 minutes – 7 minutes, 7 minutes), similar to the approach used by prior studies (Watts et al., 2018) (*M* = 3.03, *SD* = 1.19).

Covariates

Child Sex. Prior studies have noted differences in parenting behaviors (Williams et al., 2002), as well as AL scores based on child sex (Misiak et al., 2022), therefore, child biological sex was included as a covariate in the model. Child sex was assessed based on mother report at the 1-month assessment (52% male, 48% female).

Child Race-Ethnicity. Significant variations have been observed for different racial-ethnic groups in terms of AL markers (Beckie, 2012), therefore child race-ethnicity was included as a covariate. Due to the limited diversity, race-ethnicity was dichotomized as non-Hispanic White (80.4%) and non-White (19.6%). The non-White category included 12.9% African American, 1.6% Asian, 0.4% American Indian/Eskimo/Aleutian, and 4.7% other.

Family income to needs ratio. Income to needs ratio, an indicator of family socioeconomic status (SES), was included as a covariate given its associations with parenting behaviors (Roubinov & Boyce, 2017) and AL (Repetti et al., 2002). Family income to needs ratio was calculated by dividing mother's self-reported family income by the federal poverty threshold for the size of the family. The two assessments of family income to needs ratio, from child age 1 month and 36 months, were averaged to create the family income to needs ratio score used as a model covariate ($M = 3.14$, $SD = 2.59$).

Maternal depressive symptoms. Maternal depressive symptoms were assessed using the Center for Epidemiological Studies Depression Scale (Hart et al., 2022). The scale included 20 depression symptoms for which mothers were asked to report the frequency with which they experienced those symptoms over the past week. Mothers completed the measure at child age 1 month, 15 months, 24 months, and 36 months. Maternal depressive symptoms in early childhood were calculated as an average of the 4 assessment time points and this average score was

included as a model covariate given prior evidence of positive associations between maternal depressive symptoms and adolescent AL (Nelson et al., 2021) ($M = 10.05$, $SD = 7.02$).

Maternal negative life events. Mothers completed the Life Experiences Survey (Sarason et al., 1978) at the 54 months, grade 3, and grade 5 assessment time points. This 57-item questionnaire asks respondents to identify from a list of events (routine happenings to major and catastrophic events) those that have happened to them in the past year and the impact it had on them on a scale from +3 = very positive, 0 = neutral, to –3 = very negative. This study focused on the number of negative life events endorsed by the mothers. Because the data were right-skewed, scores were re-grouped into the following five categories: 1 = *no life events*, 2 = *1–2 life events*, 3 = *3–5 life events*, 4 = *6–8 life events*, and 5 = *9 or more life events* (Duckworth, Kim, et al., 2013). The three assessments at 54 months, grade 3, and grade 5 (each ranging from 1-5) were averaged to create the final negative life events score, included as a model covariate ($M = 2.53$, $SD = 0.93$).

Maternal education. Given that maternal education is associated with parenting behaviors, above and beyond family SES (Carr & Pike, 2012; Neitzel & Stright, 2004), maternal education level reported by mothers at child age 1 month was included as a covariate. Maternal education was categorized based on prior research (NICHD Early Child Care Research Network, 2001) as follows: 1 = *< 12th grade*, 2 = *high school graduate*, 3 = *some college*, 4 = *bachelor's degree*, 5 = *graduate/professional degree*) ($M = 3.08$, $SD = 1.18$).

Because these covariates have been linked to child delay of gratification and AL (Duckworth, Kim, et al., 2013; Lamm et al., 2018; Razza & Raymond, 2013; Silverman, 2003), the effect of the covariates was regressed on the mediator, child delay of gratification, and adolescent AL.

Analytic Plan

Prior to model testing, the data were evaluated for assumptions for discrete dependent variable models, including independence among observations and multicollinearity, using bivariate associations (e.g., correlations), histograms, and density plots. Descriptive statistics were evaluated for all variables. Path analysis using *Mplus* v8 (Muthén & Muthén, 2017) was used to test the main research questions. Given the count nature of the outcome variable, zero-inflated negative binomial regression was used when regressing allostatic load on the parenting and delay of gratification variables.

Missing Data and Estimation

Patterns and distributions of missingness were evaluated to see whether the data were missing at random. Multiple imputation was used to account for missing data. Twenty imputed datasets were utilized in the analysis and the pooled results across these twenty datasets are reported. Attrition across the study period was higher for African American participants and participants with lower family income to needs ratio (Wang et al., 2013). Both of these variables were included as covariates in the analyses. Accounting for missing data, the sample size for the regression models was 1,364.

Main Analyses

Significance tests were examined at $\alpha < .05$. Point-biserial correlation coefficients (r_{pb}) were used to evaluate bivariate associations between continuous and dichotomous variables. Bivariate associations among continuous variables were evaluated with Pearson *r* as the correlation coefficient. The effect size of correlation coefficients was interpreted according to Cohen's (1988) conventions: small = .10, moderate = .30, large = .50.

To address RQ1, the effects of all three early parenting variables were tested when included together in the same model, accounting for their covariances, and then individually in separate models to generate unadjusted results. RQ2 was evaluated by testing the effects of the three parenting behaviors on adolescent AL in separate models and including child delay of gratification as a mediator, to test for indirect effects. For RQ3, the effects of delay of gratification as a moderator were tested using joint product interaction terms for each of the parenting variables and the delay of gratification variable. Interaction terms were included in the model one at a time and tested for significance.

CHAPTER 3

RESULTS

Descriptive statistics and bivariate correlations between study variables are found in

Table 1. Although all three early parenting behaviors were significantly correlated with each

other (range = .50 – .63), multicollinearity was not an issue (VIF range = 1.04 – 2.57). Early

maternal supportive presence and respect for autonomy were positively correlated (r = .56) and

both supportive presence and respect for autonomy were negatively correlated with early

maternal hostility (r = -.53 and r = -.63, respectively). Early maternal supportive presence and

respect for autonomy were positively correlated with child delay of gratification at 54 months (r

= .27 and .28, respectively) while early maternal hostility was negatively correlated with child

delay of gratification at 54 months (r = -.25). All three early parenting behaviors were correlated

with adolescent AL, with supportive presence and respect for autonomy negatively correlated

with adolescent AL (r = -.18 and -.17, respectively) and hostility positively correlated with

adolescent AL (r = .17). Child delay of gratification at 54 months was also negatively correlated

with adolescent AL (r = -.11).

Table 1. Bivariate Correlations of Study Variables.

		1	2	3	4	5	6	7	8	9	10	11	12	13	14
1	EC Support														
2	EC Autonomy	.56***													
3	EC Hostility	-.53***	-.63***												
4	54m Support	.49***	-.40***	-.39***											
5	54m Autonomy	.41***	.46***	-.35***	.72***										
6	54m Hostility	-.34***	-.41***	.40***	-.61***	-.64***									
7	54m DoG	.27***	.28***	-.25***	.28***	.27***	-.20***								
8	Adolescent AL	-.18***	-.17***	.17***	-.16***	-.16***	.12**	-.11**							
9	Child Sex	.08**	.14***	-.04	-.02	.04	-.001	.06	-.07						
10	Child Race/Ethnicity	-.28***	-.33***	.26***	-.27***	-.28***	.21***	-.26***	.10**	.02					
11	Income to Needs Ratio	.35***	.27***	-.25***	.24***	.24***	-.15***	.24***	-.17***	.03	-.21***				
12	Maternal Depressive Sx	-.27***	-.30***	.24***	-.22***	-.21***	.21***	-.19***	.12**	-.01	.18***	-.27***			
13	Maternal NLE	.11***	.11***	.11***	.12***	.10**	-.09**	-.01	.06	-.02	-.13***	-.01	.17***		
14	Maternal Education	.40***	.38***	-.33***	.36***	.32***	-.24***	.27***	-.18***	.04	-.22***	.51***	-.33***	.10***	
	N (count)	1214	1214	1214	1040	1040	1040	961	699	1364	1364	1342	1363	1154	1363
	Mean / %	-.0018	-.0096	.0065	.000	.000	.000	3.03	1.48	48%	19.6%	3.14	10.05	2.53	3.08
	SD	.84	.85	.84	1.00	1.00	1.00	1.19	1.56			2.59	7.02	.93	1.18

Note. *p<0.05, **p<0.01, ***p<0.001. EC = early childhood, Support = supportive presence, Autonomy = respect for autonomy, 54m = 54 months, DoG = delay of gratification, AL = allostatic load, Sx = symptoms, NLE = negative life events. Child sex was dummy coded such that male was the omitted category. Child race-ethnicity was dummy coded such that non-Hispanic White was the omitted category. The three parental variables are standardized composite measures.

Results from the Little's MCAR Test, $X^2 = 82.86$, df = 23, p = .000, suggested that the data were not missing completely at random (MCAR), however, multiple imputation is appropriate to use when data are not MCAR, and provides unbiased estimates (Enders, 2022).

Regression output from the direct effects model, including all three early parenting behaviors in the same model accounting for their covariances, revealed no significant associations between any of the parenting behaviors and adolescent AL (see Table 2). None of the model covariates (i.e., child sex, child race-ethnicity, family income to needs ratio, maternal depressive symptoms, maternal negative life events, maternal education) had a significant effect on adolescent AL.

Next, the effects of the three early parenting behaviors on adolescent AL were tested in separate models. Both supportive presence and hostility had a significant direct effect on adolescent AL, controlling for model covariates. Specifically, children whose mothers displayed greater supportive presence in early childhood had lower levels of AL in adolescence (B = -0.10, SE = 0.05, ß = -0.08, p = .048), compared to children whose mothers displayed lower levels of supportive presence in early childhood (see Table 2). Further, children whose mothers displayed high levels of hostility in early childhood were more likely to have higher AL in adolescence (B = 0.10, SE = 0.05, ß = 0.08, p = .04), compared to those whose mothers displayed lower levels of hostility (see Table 2). Maternal respect for autonomy in early childhood was not significantly associated with adolescent AL. None of the model covariates had a significant effect on adolescent AL. Even though maternal education was negatively associated with adolescent AL, the association was not significant (B = -0.07, SE = 0.04, p = .05).

Table 2. Regression Estimates of Direct Effects of Early Parenting Behaviors on Adolescent Allostatic Load.

	B(SE)	β	*p* value
Direct Effects from Combined Early Parenting Model			
Supportive Presence	-0.06 (0.06)	-0.05	.266
Respect for Autonomy	0.00 (0.06)	0.00	.997
Hostility	0.08 (0.05)	0.06	.161
Child Sex (Female=1)	-0.12 (0.07)	-0.06	.079
Child Race-Ethnicity	0.059 (0.10)	0.02	.574
Family Income to Needs Ratio	-0.04 (0.03)	-0.16	.105
Maternal Depressive Symptoms	0.002 (0.01)	0.01	.740
Maternal Negative Life Events	0.08 (0.04)	0.07	.085
Maternal Education	-0.07 (0.04)	-0.08	.087
Direct Effects: Supportive Presence Only			
Supportive Presence	**-0.10 (0.05)**	**-0.08**	**.048**
Child Sex	-0.12 (0.07)	-0.06	.069
Child Race-Ethnicity	0.08 (0.10)	0.03	.466
Family Income to Needs Ratio	-0.05 (0.03)	-0.12	.103
Maternal Depressive Symptoms	0.002 (0.01)	0.02	.638
Maternal Negative Life Events	0.07 (0.04)	0.07	.109
Maternal Education	-0.07 (0.04)	-0.09	.054
Direct Effects: Respect for Autonomy Only			
Respect for Autonomy	-0.07 (0.06)	-0.06	.219
Child Sex	-0.12 (0.07)	-0.06	.092
Child Race-Ethnicity	0.08 (0.10)	0.03	.470
Family Income to Needs Ratio	-0.05 (0.03)	-0.13	.067
Maternal Depressive Symptoms	0.00 (0.01)	0.02	.600
Maternal Negative Life Events	0.07 (0.04)	0.06	.122
Maternal Education	-0.08 (0.04)	-0.09	.047
Direct Effects: Hostility Only			
Hostility	**0.10 (0.05)**	**0.08**	**.038**
Child Sex	-0.13 (0.07)	-0.07	.051
Child Race-Ethnicity	0.07 (0.10)	0.03	.498
Family Income to Needs Ratio	-0.05 (0.03)	-0.12	.075
Maternal Depressive Symptoms	0.00 (0.01)	0.02	.624
Maternal Negative Life Events	0.07 (0.04)	0.07	.091
Maternal Education	-0.07 (0.04)	-0.09	.060

Note. Values significant at *p* <.05 are highlighted in bold.

To evaluate the effects of proximal parenting behaviors, models including both early (24- and 36-month) and proximal (54 month) parenting behaviors, were tested for each of the three parenting behaviors. There were no significant associations between the 54-month parenting variables and adolescent AL in any of the three models. Including the 54-month parenting variables caused the effects of early parenting behaviors (i.e., supportive presence and hostility) to become non-significant. See Table 3. Given the non-significant associations of proximal (54-month) parenting behaviors, RQ 2 (mediation) and RQ 3 (moderation) were tested with the individual early parenting behaviors only.

Table 3. Examining Direct Effects of Early and 54-month Parenting on Adolescent Allostatic Load.

	B(SE)	β	*p* value
Supportive Presence			
Early Childhood Supportive Presence	-0.08 (0.06)	-0.07	.159
54-month Supportive Presence	-0.05 (0.04)	-0.05	.309
Child Sex	-0.13 (0.07)	-0.07	.060
Child Race-Ethnicity	0.06 (0.11)	0.03	.556
Family Income to Needs Ratio	-0.05 (0.03)	-0.12	.101
Maternal Depressive Symptoms	0.00 (0.01)	0.02	.671
Maternal Negative Life Events	0.07 (0.04)	0.07	.089
Maternal Education	-0.07 (0.04)	-0.08	.090
Respect for Autonomy			
Early Childhood Respect for Autonomy	-0.04 (0.06)	-0.04	.463
54-month Respect for Autonomy	-0.06 (0.04)	-0.06	.142
Child Sex	-0.12 (0.07)	-0.06	.087
Child Race-Ethnicity	0.06 (0.11)	0.02	.588
Family Income to Needs Ratio	-0.05 (0.03)	-0.13	.068
Maternal Depressive Symptoms	0.00 (0.01)	0.02	.646
Maternal Negative Life Events	0.07 (0.04)	0.07	.107
Maternal Education	-0.07 (0.04)	-0.08	.075
Hostility			
Early Childhood Hostility	0.09 (0.05)	0.08	.086
54-month Hostility	0.018 (0.04)	0.018	.666
Child Sex	-0.13 (0.07)	-0.07	.050
Child Race-Ethnicity	0.07 (0.11)	0.03	.528
Family Income to Needs Ratio	-0.05 (0.03)	-0.12	.074
Maternal Depressive Symptoms	0.00 (0.01)	0.02	.653
Maternal Negative Life Events	0.07 (0.04)	0.07	.083
Maternal Education	-0.07 (0.04)	-0.09	.063

Results from the mediation models revealed that there was a significant direct effect of supportive presence (in early childhood) on child delay of gratification ($B = 0.19$, SE $= 0.05$, $p =$ <.001), but the effect from child delay of gratification to adolescent AL was not significant ($B = -0.03$, SE $= 0.04$, $p = .34$). The total indirect effect of supportive presence on adolescent AL, as mediated by child delay of gratification, was also not significant ($B_{indirect} = -0.01$, SE $= 0.01$, $p = .37$). Early childhood respect for autonomy ($B = 0.20$, SE $= 0.06$, $p = .001$) and hostility ($B = -0.19$, SE $= 0.06$, $p = .001$) were also significant predictors of child delay of gratification at 54 months, but the pathway from child delay of gratification to adolescent AL was not significant. The total indirect effect of respect for autonomy ($B_{indirect} = -0.01$, SE $= 0.01$, $p = .35$) and hostility ($B_{indirect} = -0.01$, SE $= 0.01$, $p = .40$) on adolescent AL, as mediated by child delay of gratification, was not significant.

For RQ3, no significant interaction effect was found between any of the three early parenting variables and child delay of gratification. The interaction effect of child delay of gratification with supportive presence ($B_{int} = -0.03$, SE $= 0.04$, $p = .48$), respect for autonomy ($B_{int} = -0.03$, SE $= 0.03$, $p = .26$), and hostility ($B_{int} = 0.03$, SE $= 0.03$, $p = .24$) was not significant. None of the interaction terms were retained in the final model.

CHAPTER 4

DISCUSSION

The purpose of this study was to examine whether positive and negative parenting behaviors in early childhood are associated with AL in adolescence, either directly or indirectly through early parenting effects on children's ability to delay gratification (an indicator of self-regulation). The moderating effect of individual differences in children's ability to delay gratification on the potential associations between early parenting behaviors and adolescent AL was also tested. Understanding the role of malleable contextual factors, such as parenting, early on in development can help with designing preventive interventions that can offset the risk for high AL and future morbidity and mortality. Further, identifying moderators of these relationships can help identify individuals who are most at-risk for high AL and might benefit from targeted interventions.

The first hypothesis that parenting behaviors in early childhood would be significantly associated with adolescent AL was partially supported. Of the three parenting behaviors, only supportive presence and hostility were significantly associated with adolescent AL, with high supportive presence associated with lower adolescent AL and high hostility associated with higher adolescent AL. Much of the existing research examining parenting effects on children's stress regulation has focused on younger years, with no developmental studies that have examined early parenting associations with AL outcomes in adolescence. Studies that have examined early parenting associations with AL in later years have relied on retrospective reports of early parenting which are limited in terms of their accuracy. The current study makes a unique contribution by examining the associations of early parenting (assessed using observational coding of mother-child interactions) with adolescent AL (assessed using a comprehensive range

of biomarkers) in a longitudinal sample that was followed from birth to 15 years. These findings have relevance for prevention efforts aimed at reducing risk for adolescent AL. Prior intervention studies have found that promoting sensitive parenting and positive discipline in early childhood can lower basal cortisol secretion in children at risk for externalizing behaviors (Bakermans-Kranenburg, 2008). Thus, to the extent that current findings are replicated in future studies, interventions promoting supportive presence (e.g., providing encouragement and supportive cues to the child, providing positive emotional responses to the child) and reducing hostility (e.g., blaming or rejecting the child, withholding emotional support) may be able to reduce AL risk by promoting better stress regulation among children.

Respect for autonomy did not emerge as a significant predictor of adolescent AL. It is possible that some parenting behaviors matter more than others in terms of predicting future AL risk. Supportive presence is more closely aligned to the sensitive and nurturing aspects of parenting that have been linked to emotion and stress regulation, and hence it was a stronger predictor of adolescent AL than respect for autonomy. It is also possible that respect for autonomy is more relevant for the development of self-regulation (Bernier et al., 2010), or promotes other potential mediators, such as lifestyle factors (physical activity, dietary intake) that might be linked to adolescent AL. Future research should examine the effects of respect for autonomy in relation to other potential mediators in the link between early parenting and adolescent AL.

Exposure to harsh and negligent forms of parenting has been linked to stress dysregulation. For example, chronic interpersonal stress, such as hostile parenting, experienced early in life can lead to frequent activation of stress hormones and dysregulation of the HPA axis, as demonstrated through changes in cortisol levels (Blair et al., 2011; Miller et al., 2011;

Roisman et al., 2009), putting children at risk for secondary changes in biomarkers (e.g., blood pressure, cholesterol, glucose level) and tertiary changes in terms of risk for chronic disease (Berens et al., 2017; Repetti et al., 2002). Prior research using the SECCYD data has found that exposure to hostile forms of parenting between ages 4-11 was associated with greater cardiovascular risk at age 15 (e.g., higher blood pressure, BMI, and adiposity) (Niu et al., 2018). The current study adds to this evidence base by documenting associations between both positive and negative forms of parenting in early childhood and AL in adolescence.

Although the associations between early child parenting behaviors with adolescent AL were relatively small in magnitude, these associations are relevant in that they were present over a long follow up period of approximately 13 years and were significant controlling for important covariates including family income-to-needs ratio, maternal depressive symptoms, maternal education level and negative life events. Further, parenting behaviors and adolescent AL were assessed using objective methods; as such the associations observed are less likely to be conflated due to shared method variance or reporter bias.

Prior studies comparing timing of effects have found that recency of stress exposure can have a stronger impact on stress regulatory outcomes than early stressors (Grant et al., 2004). The current study evaluated early and more proximal parenting (at 54 months) on adolescent AL and found that the effects of early parenting were stronger and more robust than proximal parenting. This finding suggests that early parenting behaviors may matter more than proximal parenting behaviors in influencing the development of AL. This is consistent with the biological embedding of childhood adversity model which suggests that exposure to adverse conditions during the most rapid and foundational periods of development lead to the most significant biological changes (e.g., metabolic dysregulation, dysregulation of the HPA axis) (Berens et al.,

2017). Thus, the influence of early parenting behaviors is more critical as it may become embedded in biological pathways in a way that impacts AL outcomes long-term (Berens et al., 2017).

The second hypothesis that individual differences in children's ability to delay gratification (an indicator of self-regulation) would mediate the effect of early parenting on adolescent AL was not supported. This may be related to the inability of a single assessment of delay of gratification (as opposed to repeated assessments that measure change over time) to predict future AL. It could also be that delay of gratification captures only one dimension of self-regulation (i.e., behavioral regulation in the context of a reward), and other measures (e.g., emotion regulation), or a more comprehensive assessment of self-regulation across domains (cognitive, behavioral, emotional) may be needed to fully test the mediation hypothesis.

While previous research supports early parenting as a significant predictor of individual differences in children's ability to delay gratification (Von Suchodoletz et al., 2011) and, in turn, delay of gratification is a significant predictor of specific AL markers such as BMI (Duckworth, Tsukayama, et al., 2013), it is possible that delay of gratification is not a strong predictor of more comprehensive AL assessments, such as the one used in the present study which includes additional biomarkers such as blood pressure or cortisol. Other mediators such as attachment patterns and biological programming (e.g., brain functioning, HPA axis) should also be explored in future research to better understand the mechanisms by which early parenting impacts AL in adolescence.

Parenting behaviors also tend to have moderate stability, as such it may be the case that over the years parenting interacts with other lifestyle factors (e.g., diet, physical activity) and contextual influences (e.g., exposure to environmental risks) to impact adolescent AL (Katz et

al., 2012; Niu et al., 2018). Positive parenting can operate as a buffer protecting children against the negative impacts of environmental stressors (e.g., racial discrimination, neighborhood risk) that are known to be associated with higher AL (Evans et al., 2007). Future research should examine other potential mechanisms by which early parenting may impact adolescent AL.

Consistent with prior research documenting early parenting effects on child self-regulation (Moilanen & Rambo-Hernandez, 2017; Morris et al., 2007), all three early parenting behaviors assessed in this study were significantly associated with child delay of gratification in the expected directions. These results suggest that parenting interventions that focus on promoting positive parenting behaviors like supportive/sensitive caregiving, and reducing coercive, hostile parent-child interactions can help improve children's ability to delay gratification, an important indicator of self-regulation. (Bernier et al., 2010; Kok et al., 2022; Moilanen et al., 2018).

The third hypothesis that individual differences in children's ability to delay gratification would moderate the association of early parenting with adolescent AL was not supported. While interaction effects between early parenting and other dimensions of child self-regulation (e.g., effortful control) on externalizing behaviors have been reported in previous research (Kiff et al., 2011) as well as interaction effects between early negative parenting and delay of gratification on AL-related outcomes (i.e., cortisol levels) (Kryski et al., 2013), there was no significant interaction effect found in this study. One possible reason for the lack of interaction effect could be that delay of gratification, which captures the behavioral dimension of self-regulation in a rewarding context, does not operate as a moderator, but other dimensions that capture emotional or cognitive aspects of self-regulation might be more relevant. For example, previous research has shown child emotionality to moderate the relationship between maternal responsiveness and

adolescent AL (Dich et al., 2015b). While additional measures of child self-regulation (i.e., parent and teacher reports of child self-control) were available, self-reported measures can be biased, hence this study utilized the more objective, behavioral measure of delay of gratification. Future studies should test the moderation hypothesis using other measures of self-regulation (e.g., emotion regulation, effortful control).

CHAPTER 5

LIMITATIONS

This study has the following limitations which should be considered. First, it is possible that factors in parental life history (e.g., trauma) or in utero could negatively impact a child's stress response system and AL. Information on these variables was not available to account for their effects in the current analyses. In addition, genetic effects could not be controlled for and could account for the associations observed between parenting behaviors and child outcomes. Second, the findings reflect longitudinal associations between early parenting and adolescent AL. Given the study design, causal claims cannot be made. The associations observed were also small in effect size, which may be attributable to the long follow-up period, range of confounding variables controlled for in the analyses, and measurement issues for some of the variables (e.g., delay of gratification). Third, this study used a single measure of delay of gratification to assess child self-regulation. While recent studies have found the effects of delay of gratification to be highly sensitive to contextual variables (Kidd et al., 2013; Watts et al., 2018), this measure was selected because it is a more objective assessment than teacher or mother reports of child self-regulation. Additionally, the data set did not contain repeated assessments of delay of gratification, thus limiting our ability to test for transactional models between parenting and child delay of gratification, even though past research has found evidence for bidirectional associations between parenting and child self-regulation in younger years (Kiss et al., 2014). Relatedly, previous research has found that high AL can lead to poor self-regulation (Evans, 2003), suggesting the possibility of a bidirectional relationship between self-regulation and AL. It was not possible to test these associations with the current data set as AL was only assessed at the age 15 assessment.

Fourth, for the three parenting behaviors, even though the coders were blind to information about the mother-child dyads, and inter-rater reliability was monitored, there is still possibility of potential bias in the observational coding of parenting behaviors. Fifth, in relation to the AL outcome variable, although seven of the most common biomarkers used in previous research were utilized, there is no measure of immune function in the current data set (e.g., albumin, C-reactive protein, white blood cell count), which is regarded as an important indicator of stress adaptation (Whelan et al., 2021). Additionally, due to the lack of clinical high-risk cutoffs for the majority of biomarkers used in the composite AL variable, use of top/bottom quartiles could lead to quantifying some individuals as high risk even though they may not necessarily be at-risk. Finally, the SECCYD sample was predominately white and socioeconomically advantaged, so the findings may not be generalizable to families from diverse racial-ethnic backgrounds or families experiencing poverty or other significant forms of adversity.

CHAPTER 6

CONCLUSION

The current findings reveal that early parenting behaviors can have significant, long-lasting effects on adolescent AL, observed over a 13-year follow-up period, using objective multi-method assessments, and controlling for a range of confounding variables. Prior intervention studies have found that supportive and sensitive parenting behaviors can promote better stress regulation (Bakermans-Kranenburg, 2008) and healthy behaviors (e.g., dietary intake, physical activity) in children, thereby decreasing the risk of unhealthy outcomes later in life (Brody et al., 2019). Interventions that promote positive parenting behaviors such as supportive presence and decrease hostile forms of parenting may also help to reduce the risk of higher AL in adolescence.

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