PARENTING BEHAVIOR AND CHILD INFLAMMATION: THE EFFECT OF PARENTAL ATTRIBUTIONS

by

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DISSERTATION ABSTRACT

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Title: Parenting Behavior and Child Inflammation: The Effect of Parental Attributions

The proximal family environment has been identified as a critical factor implicated in childhood illness, with research pointing to chronic inflammation as a mechanism by which early social stress engenders risk for poor mental and physical health. While research has documented that aversive parenting behavior exerts a measurable impact on biomarkers of children's stress responding and downstream chronic health problems, no research to date has explored the role that parental attributions may play in whether and how parenting processes effect children's health. The current study examines whether and how parents (a) think about and (b) behave toward their child influences levels of chronic inflammation in their young children.

Participants (parents and their 3 to 7-year-old children; N = 187) were drawn from a randomized clinical trial of Parent-Child Interaction Therapy (PCIT) for families involved with child welfare. Relationships among child inflammation concentrations and sociodemographic variables, risk variables, and parenting factors (e.g., attributions and behaviors) were explored to understand how elevations in CRP may emerge in early childhood, specifically in high-risk children exposed to adversity. Mediation analyses were used to test the hypothesis that parenting behavior would emerge as a mediator of parental attributions and child inflammation. To test a competing hypothesis, moderation

analyses were used to test if parental attributions instead served as a moderator of a relationship between parenting behavior and child inflammation.

Results indicated that child inflammation was not significantly related to sociodemographic variables, indices of adversity, parental attributions, or parenting behavior. However, the moderation model examining parental perceived control attributions as a moderator of harsh parenting behavior and child inflammation emerged at $p = .05$. In the presence of high perceived control attributions (i.e., parents attributing responsibilities of negative parent-child interactions to themselves, rather than their child) harsh parenting behavior was related to higher concentrations of child CRP ($p = .05$). The results of this study suggest that targeting attributions may offer a useful protective factor for children, however more research is needed to establish whether these social cognitive factors exert causal influences on children's immune development and downstream physical health into adulthood.

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CHAPTER I

INTRODUCTION

The foundations of good health can be established early in life. There is growing evidence that early experience has significant immediate effects in childhood (Bethell, Newacheck, Hawes, & Halfon, 2014; Graham-Bermann & Seng, 2005), and shapes health and well-being into adulthood (Anda et al., 2009; Dube et al., 2003). Indeed, the potential long-term impact of early adverse experiences in shaping adult physical health outcomes is well documented (Anda et al., 2009; Dube et al., 2003, Filetti et al., 1998), with studies showing that stressful family environments predict current and future health problems (Danese et al., 2011; Danese & Tan, 2014; Dube et al., 2003; Lanier et al., 2015; Lee, 2010; Rogosch, Dackis, & Cicchetti, 2011). Thus, promoting health and well-being during childhood by fostering positive family functioning has the potential to improve the immediate health of young children, and contribute to the development of physically healthy, emotionally resilient adults.

The proximal family environment has been identified as a critical factor implicated in childhood illness (Belsky, Bell, Bradley, Stallard, & Stewart-Brown, 2007; Priest et al., 2015; Wood & Miller, 2002), with research pointing to chronic inflammation as a mechanism by which early social stress engenders risk for poor mental and physical health (Byrne et al., 2017; Nelson et al., 2017; Slavich & Irwin, 2014). Efforts have therefore increased to identify risk and protective factors in family interactions that may contribute to increased levels of chronic inflammation in childhood. Research suggests that how parents (a) think about and (b) behave toward their child influences multiple facets of development (Repetti, Taylor, & Seeman, 2002; Shonkoff, Boyce, & McEwen,

2009). While research has documented that aversive parenting behavior exerts a measurable impact on biomarkers of children's stress responding and downstream chronic health problems (Miller, Brody, Yu, & Chen, 2014; Miller, Chen, & Parker, 2011), no research to date has explored the role that parental attributions may play in whether and how parenting processes effect children's health. Parents who hold negative, threat-sensitive views toward their children, and presume their child maintains high power and control in the parent-child relationship, are more likely to use harsh and abusive parenting strategies (Bugental et al., 1990; Bugental & Happaney, 2000; Crouch et al., 2017), and display negative emotionality toward their child (Mills & Rubin, 1990), and physiological arousal in challenging caregiving contexts (Bugental & Cortez, 1988; Wang, Deater-Deckard, & Bell, 2016).

The current study will examine whether and how observed parenting behavior and quality of parental attributions impact levels of chronic inflammation in their children. I posit that quality of a parent's behavior will relate directly to their child's level of chronic inflammation. Additionally, this study will examine if parental attributions interact with quality of parenting to affect child inflammation levels, or if attributions strengthen or weaken the effect of parenting behavior on their children's chronic inflammation. As such, I will test both mediation and moderation models regarding the associations between attributions, behavior, and children's chronic inflammation. Increased understanding of the relationships between parenting behavior, parental attributions, and child inflammation will provide insight into how parents' behavior and perceptions of their child converge to influence their children's health.

Chronic Inflammation

Research has implicated the immune response as a mechanism connecting early experience with later disease (Miller et al., 2010). Activity of the immune system is critical in accelerating disease states, as chronic stimulation of an immune response over time results in the development of numerous illnesses, including depression (Miller, Maletic, & Raison, 2009), coronary heart disease (Lippy, 2001; Miller, Stetler, Carney, Feedland, & Banks, 2002), and some cancers in adulthood (Adler et al., 1994). Indeed, chronic overactivity of the immune response increases susceptibility to illnesses over time (Bray & Cotton, 2003; Cohen et al., 2012), with initial biological disruptions beginning in childhood. Research has documented that family environments, specifically, influence children's immune activity (Cohen, 2004), with evidence pointing to inflammation as a mechanism that links adverse early family environments to disease (Byrne et al., 2017; Fagundes, Bennet, Derry, & Kiecolt-Glaser, 2011; Miller et al., 2011). Inflammation is a critical component of immune system functioning, as it is the body's response to pathogenic invasion or physical injury (Hänsel, Hong, Cámara, & Von Känel, 2010; Miller et al., 2011). The inflammatory response is stimulated by physical stressors (i.e., acute physical injury) as well as psychosocial stress (e.g., family conflict, child maltreatment [CM]; Byrne et al., 2017; Hennessy et al., 2004). When a stressor occurs, be it physical or psychosocial in nature, the body initiates a physiological reaction to mobilize toward the threatening stimulus (Cannon, 1929; Gunnar & Quevedo, 2007). The result is an upregulation of a "fight or flight" stress response, including increased heart rate, epinephrine, cortisol, and the later production of proinflammatory cytokines (Kuhlman, Chiang, Horn, & Bower, 2017; Miller et al., 2011). Increases in pro-

inflammatory cytokines in response to acute threat or injury are necessary to repair damaged cells and tissue (Punt & Owen, 2001). However, repeated stimulation of an inflammatory response results in chronic, low-grade levels of inflammatory biomarkers and are predictive of chronic disease (e.g., diabetes, cardiovascular disease; Kaplan & Frishman, 2001, autoimmune disorders; Abou-Raya & Abou-Raya, 2006; some forms of cancer; Antoni et al., 2006; Mantovani et al., 2008).

Parenting Behavior and Chronic Inflammation

One inflammatory cytokine that is particularly sensitive to psychosocial stress is C-reactive protein (CRP). CRP is a marker of chronic, low-grade inflammation that has been linked with negative childhood experiences (Miller et al., 2011) and is indicative of risk for multiple chronic diseases in adulthood (Bertoni et al., 2010; Brody, Yu, Beach, Kogan, Windle, & Philibert, 2014; Sesso et al., 2010). There is much evidence to support that severe disturbances in early caregiving are linked to high CRP levels in adulthood (Danese et al., 2007; Kiecolt-Glaser et al., 2011; Slopen et al., 2010), however the pathways for these associations are not well understood. For instance, experiences of maltreatment in early childhood have been retrospectively linked with elevated CRP levels in adulthood (Baumeister, Akhtar, Ciufolini, Pariante, & Modnelli, 2016; Coelho et al., 2015; Danese, Pariante, Capsi, Taylor, & Poulton, 2007; Slopen et al., 2015, Taylor et al., 2006), and have been found to prospectively predict elevated CRP in adolescents at age 12 (Danese et al., 2011), and in 18-year-old females (Baldwin et al., 2018). Further, parent-child disruptions (i.e., foster care placement, parental separation) during middle childhood, coupled with the accumulation of adverse events from birth to age 8 years, predicted elevated CRP levels at age 10 and persisted over time with increases in CRP

observed at age 15 (Baldwin et al., 2018). These studies suggest that more serious disruptions in family functioning early in life may enact lasting changes on the developing immune system to potentiate disease states in adulthood.

While previous research has established an association between severe forms of adverse family experiences in childhood and elevated CRP over time, there is conflicting evidence concerning how fine-grained forms of parent-child interactions may also influence inflammation. Child maltreatment encompasses a constellation of negative, harsh parenting behaviors (Belsky, 1993) that evoke stressful, physiologically heightened responses in children; thus, increased understanding of relationships among specific parental behaviors and child inflammation within a sample of child welfare-involved families may clarify how parenting influences health and disease states throughout development. Indeed, there is some evidence that negative, harsh parenting behavior affects inflammation levels in childhood, and this study will test the direct effects of negative parenting behavior on levels of inflammation in a high-risk sample of 3 to 7-year old children, in child welfare-involved families. Negative, harsh parenting styles can include high levels of vigilance and control (Brody et al., 2014) directed towards children, which, coupled with elevated levels of anger and low levels of emotional control, triggers physiological reactions (i.e., sympathetic-pituitary-adrenal [SAM] and HPA axis systems) in children that result in chronically elevated systemic inflammation (Brody et al., 2014). Research has supported the link between negative parenting and increased inflammation in childhood. For instance, in another study by Byrne and colleagues (2017), self-reported parenting style predicted levels of inflammation in children ages 8 to 11 years old, such that parents who endorsed poor monitoring behavior

had children with higher levels of inflammation and immune activation. Further, in a sample of low-income African American youth, exposure to harsh parenting was linked to higher levels of anger in 11-13-year-old adolescents, which was predictive of higher CRP levels as teenagers (Brody et al., 2014). These studies suggest that negative parenting behavior may be key in shaping physiological responding in children, as evidenced by levels of chronic inflammation.

Conversely, experiences of positive parenting in childhood appear to buffer later proinflammatory signaling as early as adolescence (Byrne et al., 2017). In a study of parents and their 10 to 12-year-old adolescents, observed positive parenting behavior during a conflictual task was found to be associated with lower adolescent CRP levels approximately two years later (Byrne et al., 2017). Miller and colleagues (2010) examined the effects of a family-oriented intervention in a sample of low-income parents and their 11-year old children, and found improvements in parenting quality (i.e., increases in nurturing parenting, coupled with decreases in harsh-inconsistent parenting) were associated with reductions in inflammation levels in their children assessed later at age 19. Taken together, these studies suggest that patterns of positive parent-child interactions may be significant enough to influence lasting changes in children's inflammatory responding.

Although these studies show significant links between parenting behaviors and CRP levels in adolescence, few investigations have explored how parenting affects child inflammation at an earlier stage of development. A small body of research has found links between attachment relationships and CRP during infancy and suggests that infants with disorganized attachments (marked by parents who display contradictory behaviors

such as warmth and responsiveness followed by avoidance, distress, or anger in response to their child's distress; Lyons-Ruth & Spielman, 2004) show greater CRP levels than securely attached infants (David et al., 2017; Measelle & Ablow, 2018; Measelle, David, & Ablow, 2017). Similarly, in a longitudinal examination of infants over 6 months, securely attached infants evidenced lower CRP levels over time (Nelson, Bernstein, Allan, & Laurent, 2019) suggesting that an infant-caregiver relationship characterized by high maternal warmth and predictability may influence biological processes responsible for pro-inflammatory responding as early as infancy. Of note, these investigations were drawn from one high-risk mother-infant participant sample, which somewhat limits generalizability of the findings.

Nevertheless, these early studies of attachment relationships suggest a small linkage between the quality of parent-child relationships and inflammation levels may appear in infancy. However, there is a relative dearth of research examining the effect of parental behaviors on CRP during early childhood, specifically among children ages 3 to 7 years old. Do similar patterns of inflammatory responding appear in children who experience maladaptive parenting in the early childhood years? Several studies have examined links between experiences of maltreatment and concurrent inflammation in children; however, the results are inconclusive. For instance, Tyrka and colleagues (2015) examined relationships between adverse experiences and two biomarkers of inflammation, IL-1 β and CRP, in a sample of child welfare-identified, maltreated preschoolers. Results showed associations between number of concurrent and lifetime contextual stressors (including child maltreatment) and higher levels of IL-1β, but no significant associations with CRP. Similarly, in a sample of older 3 to 12-year-old child

welfare-involved children, exposure to child maltreatment was unrelated to current salivary CRP levels (Cicchetti, Handley, & Rogosch 2015). In a healthy, community sample of 5-year-old children, Riis and colleagues (2016) similarly found no associations between maternal psychological distress and their children's inflammation levels (measured by a composite of salivary IL-1β, IL-6, IL-8, and TNF-α). Although these studies examined parenting quality via parent self-report, research has not investigated how observed parenting behaviors may relate to concurrent inflammation levels in their preschool-aged children, specifically utilizing CRP via dried blood spots.

Early childhood is a salient time where biology is sensitive to disruptions (Measelle & Ablow, 2018), and research suggests that stressful environments experienced within a sensitive developmental window enact lasting changes in neuroendocrine (Giedd & Rapoport, 2010), immune (Boyce, 2016), and cardiovascular (Berenson, 2002) systems. By characterizing the patterns of inflammatory responding in children exposed to negative parenting styles, we can gain a deeper understanding of how parent-child relationships may influence disease vulnerability in their 3 to 7-year-old children. Further, studies have not extended beyond parenting behavior to investigate possible drivers of problematic parenting strategies (i.e., cognitive influences on parental behavior) and inflammation in children. Similarly, research has not examined the conditions under which parenting behavior be might be exacerbated in its impact on child inflammation. One important factor that influences parent-child interactions is parental attributions (Bugental, Blue, & Lewis, 1990; Crouch et al., 2017), or the kinds of cognitive schemas parents hold about their children. It is possible that the quality of attributions a parent holds about their child exerts a salient moderating effect on the

association between parenting and biomarkers of chronic inflammation in children. For instance, a parent who assumes their child misbehaves with intentional malice, or is to blame for negative parent-child interactions, may behave in a more reactive, harsh manner toward their child whereas a parent who views their child's misbehavior through a developmentally appropriate lens may respond with warmth and attention. In other words, a parent's behavior is generated based on their pre-existing "attributional style" (e.g., Bugental 1987). Alternatively, support may be observed for a mediation model in which parental attributions show a relation with child inflammation by influencing the quality of parenting displayed toward the child. For example, a parent's negative, threat-sensitive attributions about their child may cause the parent to engage in more negative parenting behaviors, which then relates to greater chronic inflammation in the child. The present study will therefore address two gaps in the current literature to clarify in a sample of child welfare families, (a) if parenting behavior relates directly to children's chronic inflammation during early childhood, and (b) whether parents' attributions moderate the strength of associations between quality of parenting and markers of inflammation in their 3 to 7-year-old children, or if parenting behavior mediates the association between attributions and inflammation levels in children.

Parental Attributions

The role of parental cognitions is critical in understanding how parent-child dynamics are developed and maintained, and may be key in influencing immune activity in children. Parental attributions can be defined as caregivers' interpretations and evaluations of their child and their child's behavior (Beckerman, van Berkel, Mesman, & Alink, 2017). Parents differ in their reactions to challenging caregiving situations; given

the same "difficult" child behavior, parents may respond in growth-promoting or risk-inducing ways based on their underlying cognitive schemas (Bugental et al., 1990). Research promotes the importance of caregivers' attributions in predicting family functioning (Bugental & Johnston, 2000) as studies have demonstrated that parents who think of their children in positive, developmentally sensitive ways tend to show greater warmth and responsiveness while parenting (Bugental, 1987; Hastings & Rubin, 1999). In contrast, parents who hold negatively biased cognitions toward their children show harsher, more abusive behavior in caregiving contexts (Bugental et al., 1990; Bugental & Happaney, 2000; Crouch et al., 2017). More specifically, parents who view their children as intentionally misbehaving, controlling, or purposefully acting with malice, are more likely to use abusive parenting behavior (Azar, 1988; Miller & Azar, 1996). Indeed, studies suggest that parents with documented histories of perpetrating child physical abuse are more likely to assume their child misbehaves with deliberate hostility and perceive themselves as victims of challenging child behavior (Azar, 1987; Azar & Twentyman, 1986; Bugental, Blue, & Cruzcosa, 1989; Larrance & Twentyman, 1983).

Research also suggests that parents with low perceived power act differently with children whose behavior patterns could be interpreted as posing a threat (Bugental & Happaney, 2004; Martorell & Bugental, 2006). For example, parents who see themselves as having less power than their children are thought to hold threat-sensitive caregiving schemas (Bradley & Peters, 1991), in that hard-to-manage child behavior is seen as a threatening and intentional. Parents who view their child in negative, threat-sensitive ways may presume their child is manipulative, bothersome, or blameworthy (Bugental & Cortez, 1988). These negative attributions have been found to be important predictors of

disciplinary actions (Milner 1993, 2003), with research showing that children who are identified as "difficult" are more likely to be recipients of harsher, more abusive parenting than do their "easier" siblings (Bugental, 1990). Further, parents who use harsh and abusive behaviors were more likely to explain difficult interactions with their child as a result of child motivation (i.e., the child being stubborn or manipulative; Bugental, 1990). These studies place importance on parental attributions as key in influencing parenting behavior.

While there is strong evidence to suggest that type of attributions a parent holds about their child are linked to the ways in which they behave toward him/her/them (Bugental 1990; Bugental & Cortez, 1998; Crouch et al., 2017; Slep & O'Leary, 1998), the directionality of this relationship remains unclear. In theory, a direct causal effect exists between beliefs and subsequent behaviors (e.g., a previously held belief determining later action; Goodstadt & Hejelle, 1972). Thus, social-cognitive research historically identified attributions as antecedents that shape parenting behavior (Bugental, 1987; Kelley & Michela, 1980). For example, in a study of low-risk mothers and toddlers, Slep & O'Leary (1998) found that mothers' attributions toward their children's misbehavior determined their observed disciplinary actions, providing support for the notion that attributions drive parenting behavior. Similarly, Nix and colleagues (1999) found that maternal hostile attributions predicted child conduct problems, but that much of this effect was mediated by harsh parental discipline. Taken together, these studies provide some evidence that parental attributions promote certain parenting tendencies.

Conversely, there is also evidence that attributions are poor predictors of caregiving behavior (Becker & Krug, 1965; Bugental, 1987), and that attributions may be

best understood as moderating the effect of parenting practices on their children's functioning (Bugental 1987; Goodnow, 1985; Snyder, Cramer, Afrank, & Patterson, 2005). In other words, certain attributional styles may sensitize parents to challenging child behavior and others may buffer against the effect of maladaptive disciplinary actions on child outcomes (Bugental 2000; Snyder et al., 2005). Parental attributions can be conceptualized as cognitive "rules" that guide behavior (Hayes & Ju, 1995) and, once established, inform parents' behavior regardless of situational or developmental considerations of their child's behavior (Miller & Prinz, 2003; Snyder et al., 2005). Indeed, research suggests that parents who view child misbehavior as intentional, or stemming from child traits, are more unwilling to change their discipline strategies (Miller & Prinz, 2003). Alternatively, parents who hold "high perceived control" attributions (i.e., attribute difficult child behavior to factors over which the parent has control), or who simply lack hostile attributions toward their child, may be more sensitive to situational or developmental variations and more flexible in their parenting behaviors (Bugental, 1987; Hastings & Rubin, 1999). Some research supports the moderating effect of parent attributions on child outcomes. In a study of low-income parents and their kindergarten-aged children, parents' problematic discipline practices predicted child behavior problems, but this effect was exacerbated when parents attributed hostile intent to their child's misbehavior (Snyder et al., 2005). Similarly, Bugental Blue, & Cruzcosa (1989) found that mothers who maintained "low perceived control" attributions (i.e., perceiving themselves to be victim of child behavior) were more likely to be abusive and display high coercive parenting behavior compared to mothers who did not have this attributional style. These studies support the notion that attributions may operate as a

moderating factor, where parents' attributional style provides a context in which parenting behavior affects child outcomes (Bugental, 1987). The current study will examine both potential models of parenting behavior and parental attributions in relation to children's chronic inflammation levels. First, a mediation model will be examined, where negative attributions are associated with more aversive parenting behavior, which are then linked to greater child inflammation. Alternately, a moderation model will be tested to examine if parental attributions serve as a moderator of associations between quality of parenting and children's inflammation, such that the presence of more negative attributions exacerbates the negative effect of harsh parenting on elevations in child inflammation. Likewise, positive attributions would be expected to buffer (or weaken) the negative effect of harsh parenting on child outcomes.

Current Study

The present study will test two competing hypotheses to determine whether parental attributions and parenting behavior together, or individually, relate to child inflammation. Given mixed findings about whether parental attributions drive parenting behavior, or whether parental attributions strengthen or weaken parenting behavior, the current study seeks to clarify which of two pathways best represents associations with children's outcomes. Despite the evidence that negative parenting behavior may "get under the skin" to influence child biology (e.g., Miller et al., 2011), little is known about how attributions might drive parenting behavior to influence child inflammation, or if attributions might exacerbate or buffer the effect of parenting behavior on child inflammation levels. Previous research has identified attributions as a driver of parenting behavior (Nix et al., 1999; Slep & O'Leary, 1998); thus, it is possible that parental

attributions influence parenting behavior, which in turn impacts child inflammation. More specifically, parents who hold negative attributions toward their child may demonstrate less positive parenting behavior in caregiving contexts. Children of parents who perceive them negatively may have greater levels of chronic inflammation, but this effect may occur through the experience of negative parenting behavior (see figure 2 for the hypothesized mediation model).

Conversely, research has also identified attributions as a potential moderator between parenting behavior and other child outcomes (i.e., child conduct problems; MacKinnon-Lewis et al., 1994). It may be the case that children of parents who demonstrate more negative, harsh parenting behavior will evidence higher levels of chronic inflammation, but this effect will be exacerbated in the presence of negative, threat-sensitive, controlling parental attributions (see figure 2 for the hypothesized moderation model). Research on the moderating effect of parental attributions has documented that parents who possess pre-existing negative beliefs about their children are more vulnerable to difficult parent-child interactions, exacerbating behavioral and emotional reactions toward their children and resulting problematic child behavioral and emotional outcomes (Bugental & Johnston, 2000; Hastings & Rubin, 1999). However, there are no studies to date that examine how parenting behavior and attributions interact to affect child stress physiology (i.e., chronic inflammation). The current study will address this gap with the aim of understanding how parental attributions and parenting behavior relate to child inflammation. Thus, there are three research questions and associated hypotheses:

Research question 1: Does observed parenting behavior relate directly to child inflammation?

Hypothesis 1: Parent's observed harsh/controlling behavior will be associated with greater child inflammation. Conversely, observed positive parenting behavior will be associated with lower levels of child inflammation.

Research question 2: Does parenting behavior mediate the association between parental attributions and child inflammation?

Hypothesis 2: Negative parental attributions will be associated with more harsh parenting behavior, which in turn will be linked to higher levels of child inflammation.

Research question 3: Do parental attributions moderate associations between parenting behavior and child inflammation?

Hypothesis 3: Associations between negative harsh parenting behavior and greater chronic inflammation in children will be stronger in the presence of negative, threat-sensitive parental attributions and low perceived parental control attributions. Associations between positive parenting and children's CRP levels will weaken or not change in the presence of positive parental attributions and high perceived parental control attributions.

An additional exploratory aim of the current study is to address an important gap in the current understanding of how chronic inflammation is distributed in high-risk children ages 3 to 7-years-old and associations with children's sociodemographic characteristics and indices of risk. As reviewed above, there are very few studies examining chronic inflammation in early childhood and its' relationship with family relational quality, and the studies that do exist have produced mixed findings. There is a

similar lack of research on how elevations in CRP may relate to experiences of environmental risk in general (e.g., low family income or poverty, child maltreatment) in young children who have experienced adversity. Again, the studies that have examined these associations have reported inconsistent results (Cicchetti et al., 2015; Tyrka et al., 2015), and the same is true for studies examining associations between CRP concentrations and demographic variables (i.e., child age, sex, and anthropomorphic measurements) in high-risk children during the early childhood years (Broyles et al., 2012; Cook et al., 2000). In sum, the current research evidence base on how CRP 1) appears in high-risk, 3 to 7-year-old children in relation to experiences adversity; and 2) how CRP may vary based on children's individual, demographic characteristics, is significantly limited. Thus, this study will examine the following variables to understand any associations that may exist with child CRP concentrations.

Child age and sex. Ample evidence suggests that CRP concentrations increase with age (Cook et al., 2000; Ford et al., 2003; Wener, Duam, & McQuillan, 2000). Similarly, child sex was included as a possible covariate given some evidence to suggest CRP levels may differ between male and female children, with females showing higher CRP concentrations (Cook et al., 2000, Ford et al., 2003). However, some studies examining at-risk children suggest no differences in CRP concentrations between males and females (ages 8-11 years; Cicchetti et al., 2015; ages 3- 5 years; Tyrka et al., 2015).

Waist Circumference (WC). Previous research has established that adiposity is highly related to CRP levels in children (Cook et al., 2000). As childhood-onset obesity has emerged as a public health priority due to its increased prevalence and status as a precursor to chronic health conditions (Cook et al., 2000; Ford et al., 2003; Visser et al.,

2001), CRP has been identified as an early-detectable marker of the association between obesity in childhood and cardiovascular disease in adulthood. Although body mass index (BMI) is a measurement of obesity commonly included as a covariate of CRP, it has been argued that waist circumference (WC) is a more meaningful measure of weight status as it reflects central obesity, thus reflecting risk factors for metabolic syndrome (i.e., abdominal obesity and its association with impaired glucose tolerance, dyslipidemia, and high blood pressure; Elks & Francis, 2010; Nakamura et al., 2008).

Ethnicity. There is some evidence to suggest that CRP concentrations vary by ethnicity group, with Mexican American/Latino children showing higher CRP levels than Black and White-identified children (Dowd, Zajacova, & Aiello, 2010; National Health and Nutrition Examination Survey, 2000). However, some studies have examined associations between CRP and ethnicity by comparing only White to Non-White children (Broyles et al., 2012; Slopen et al., 2011), or have found no associations between child ethnicity and CRP levels (Broyles et al., 2012; Cicchetti et al. 2015).

Family Income. Poverty or low family income has been found to robustly associate with CRP levels starting in later childhood (Broyles et al., 2012; Miller & Cole, 2012; Slopen et al., 2012) and into adulthood (Danese et al., 2011; Taylor et al., 2006). One study reported an association between greater socioeconomic disadvantage and salivary CRP in high-risk infants (David et al., 2017). There are some studies that do not show such association, however (Cook et al., 2000; Tyrka et al., 2015), leaving the CRP-income link in early childhood unclear.

Adverse Childhood Experiences/ Child Maltreatment Status. Childhood adversity, including experiences of abuse and/or neglect, have been found to associate with CRP

levels in infancy (David et al., 2017; Measelle & Ablow, 2018; Nelson et al., 2017) and adolescence (Howe et al., 2010; Murasko, 2008). Conversely, other studies have observed no relationships among early adversity and children's CRP concentrations. (Cicchetti et al., 2015; Cook et al., 2000, Tyrka et al., 2015). The current study will explore relations among CRP concentrations and experiences of adversity to clarify these inconsistencies.

CHAPTER II

METHOD

Participants

Participants were drawn from a randomized clinical trial of Parent-Child Interaction Therapy (PCIT) for families involved with child welfare, entitled the Coaching Alternative Parenting Strategies project (NIDA R01DA036533) with 205 parents and children. Families were eligible for inclusion in the larger study if their children were 3 to7-years-old at the time of study entry. Parents were biological parents who were the main custodial caregiver of the child, who resided in the same home setting with the child at least 50% of the time and spoke English fluently. Families were excluded from the larger study if: the target age child was in foster care, living with a caregiver or adult who had perpetrated sexual abuse (PCIT is contra-indicated in such cases), or could not complete the assessment procedures due to a severe developmental, medical, or physical disorder. Participating parent-child dyads included in the current study are 205 families who completed Wave-1 preintervention assessments in the clinical trial. Approximately 18 parents reported their child was administered medications producing anti-inflammatory effects (i.e., Benadryl, Zyrtec, Ibuprofen, among others; Assanasen & Naclerio, 2002; El-Shaaraway, El-Hakim, & Sameeh, 2006; Takeda et al., 2003) in the 24 hours prior to their CRP blood draw. These cases were subsequently removed from the analytic sample due to the possible confounding effect on CRP. Additionally, approximately 35 children refused or were unable to provide whole blood spot samples, leaving 170 children with valid CRP data. Two children had CRP concentrations greater than 10mg/L and were excluded as well, as values above 10mg/L

indicate acute infection (Snodgrass et al., 2007), leaving a total of 150 children with complete CRP data that were included in the analyses.

Descriptive statistics for the sample demographic variables are presented in Table 1. Participants included in the current study were 187 high-risk parent-child dyads. The majority of parents were mothers (n = 163; 87.2%) with 24 fathers participating in the study (12.8%). The average parent age was M = 32.30 years (SD = 6.48). Most parents were single (n = 88, 42.7 %). The participant sample somewhat reflected the racial/ethnic composition of the Pacific Northwest community from which it was drawn. The majority of parents (n = 133, 71.5%) and children (n = 110, 58.6%) identified as White, 35.9% (n = 65) of children and 19.9% (n = 37) of parents identified as Multiracial, and 3.2% (n = 6) of children and 2.7% (n = 5) identified as Hispanic American/Latina. Child ages ranged from 3 to 7 years (M = 4.74, SD = 1.42). There were slightly more males (n = 105) than females (n = 82), and most children (n = 65) were in preschool or head start, followed by not in school (n=40), kindergarten (n = 38) and second grade (n=27).

Regarding the socioeconomic characteristics of the current sample, parents ranged in yearly household income from $700- $90,000 per year, with the family who earned $90,000 as an outlier (median yearly income = $14,400). Approximately 33 parents did not report yearly income (n = 154). Nearly half of parents enrolled in the study were unemployed (n = 101, 54.6%) and 82 parents (43.9%) reported receiving Temporary Assistance for Needy Family (TANF/Welfare). Other forms of financial assistance for the participant sample included utilization of food stamps (n = 156; 83.4%), receipt of Women Infants and Children (n = 78; 41.7%), and 86 parents (46.0%) reported their child receives free lunch/food assistance. Per parental report, the average number of people

living in home was 4.26 (*SD* = 2.07), and the average number of family members supported by the reported income was 3.55 (*SD* = 1.35), thus placing this participant sample as living predominantly below the federal poverty line (U.S. Department of Health & Human Services, 2020).

Other risk factors that characterize the current sample as high-risk are parental education, Adverse Childhood Experiences (ACEs) exposure, and child maltreatment status. The majority of parents reported a relatively low average education level, with most parents' highest education completed equating to just under a high school diploma. Parents self-reported adverse childhood experiences (per the parent ACEs questionnaire) ranged from 0-10 ACEs, and on average parents reported experiencing 5.2 (*SD* = 2.72). ACEs. Child ACEs ranged from 0-8 adverse experiences, with the average child experiencing 3.5 (*SD* = 1.95) ACEs. 74.3% of children (*n* = 139) included in the current study were exposed to maltreatment (based on review of families' child welfare records, which are coded using the National Child Abuse and Neglect Data System; NCANDS Child File Codebook, 2019).

Table 1

Sociodemographic Characteristics of Sample

Variable name	*N*	*M*	*SD*	Range	Skew
Child age (in years)	187	4.74	1.42	3-8	0.259
Child ACEs (possible range 0-10)	187	3.50	1.96	0-8	0.208
Parent age (in years)	187	32.30	6.48	18-64	0.930
Parent ACEs (0-10)	187	5.22	2.71	0-10	-0.242
Yearly household income (in dollars)	154	17786.7 *Mdn: 14400*	12993.86	700-90000	1.99
Number of people living in home	185	4.27	2.07	2-15	2.05

Variable name	*N*	Percent Endorsed
Child Ethnicity		
European American/White	110	58.6%
Hispanic American/Latina	6	3.2%
African American/Black	3	1.6%
Native American/Alaskan Aluet	1	0.5%
More than one race/ethnicity	65	34.9%
Unknown	2	1.1%
Parent Ethnicity		
European American/White	133	71.5%
Hispanic American/Latina	5	2.7%
African American/Black	3	1.6%
Pacific Islander	3	1.6%
Native American/Alaskan Aleut	2	1.1%
More than one race/ethnicity	37	19.9%
Unknown	4	1.6
Child Sex		
Male	105	55.9%
Female	82	44.1%
Parent Sex		
Male	24	12.9%
Female	163	87.1%
Child grade in school		
Not in school	40	21.5%
Preschool/Head start	65	34.4%
Kindergarten	38	20.4%
First grade	17	9.1%
Second Grade	27	14.5%
Parental Education		
No high school	6	3.3%
Partial high school	26	14.0%
Graduated high school/GED	94	50.0%

Table 1, Continued.

Parental Education		
Technical/vocational certificate	28	15.1%
Associates degree/junior college	22	11.8%
Bachelor's degree	9	4.8%
Graduate degree	2	1.1%
Parent Marital Status		
Married	30	
Living together	21	56.3%
Separated	19	17%
Divorced	19	10.2%
Widowed	1	0.5%
Single	88	47.3%
Other	9	4.8%
Parent Employment Status		
Unemployed	102	54.6%
Part time temporary/seasonal	6	3.2%
Part time stable employment	32	17.3%
Full time temporary/seasonal employment	6	3.2%
Full time stable employment	41	21.6%
Child Maltreatment Status	187	
Maltreatment Indicated	139	74.3%
No Maltreatment	48	25.6%

Note. ACEs= Adverse Childhood Experiences.

Procedure

All procedures used in this study were approved by the University of Oregon and State of Oregon Department of Human Services (DHS) Institutional Review Boards. Recruitment and assessment procedures began in 2015. Parent-child dyads were invited to participate due to involvement with the Oregon Child Welfare and were recruited via a DHS liaison at the CAPS study. For parents without legal custody who retained physical custody of their child, parent and DHS caseworker legal consent was obtained for child participation in the study.

For the larger clinical trial from which data for this study were drawn, all parents and children were invited to participate in pre- and post-PCIT intervention assessments held approximately 12-months apart. Each assessment was comprised of two laboratory visits scheduled one week apart, and used a similar protocol: completion of demographic and psychosocial questionnaires, cognitive performance tasks, and bio-behavioral assessments, all of which were conducted by a team of three trained research assistants (see Nekkanti et al., under review, for full study protocol). The current study focuses on data collected during the preintervention (i.e., Wave 1) assessment at visits 1 and 2.

Upon arrival to the laboratory, voluntary informed consent was obtained from parents (and caseworkers if relevant), and parent-child dyads underwent a series of anthropomorphic measurements (i.e., height, weight, waist circumference). Parents and children were then fitted with disposable electrodes for electrocardiogram (ECG) and impedance cardiogram (ICG), which were used to monitor participants' cardiac physiology during resting baseline, all tasks, and post-task recoveries at each visit.

Parent-child dyads then completed the PCIT Dyadic Assessment Protocol (Eyberg & Funderburk, 2011), a joint interaction task that was video-recorded while cardiac physiology was continuously monitored. Observations of parenting behaviors during this task were used in the current study. The PCIT dyadic assessment protocol consists of three 5-minute parent-child interactions: a 5-minute Child-Led Play task, where parents are instructed to follow their child's lead in play; a 5-minute Parent Led Play task during which parents lead the play and instruct their child to play by their rules; and a 5-minute Clean Up task, where parents are instructed to direct their child in cleaning up toys without parental assistance. Parents were given an earbud and walkie-talkie for assessors

to provide instructions for the joint interaction tasks. Video recordings of the PCIT dyadic assessment were transcribed and coded at a later date by a team of trained coders using the Dyadic Parent-Child Interaction Coding System-IV (DPICS-IV; Eyberg, Chase, Fernandez, & Nelson, 2013; see pp 27 for details regarding the DPICS-IV coding procedure).

Dyads completed another joint task, then were given a short break and snack prior to transitioning to individual tasks, which included a battery of cognitive performance and self-regulatory tasks. No data from these individual tasks for parents or children were used in the current study.

Families returned for a second laboratory visit approximately one week after their initial assessment. Children completed the remaining cognitive, executive functioning, and self-regulatory tasks individually while parents answered questionnaires that assessed demographic information, environmental risk, and child behavior. Parents also completed the Parental Attribution Test (PAT; Bugental et al.,1989) and Structural Analysis of Social Behavior (SASB)-Intrex Questionnaire (Benjamin, 1974) which assessed parental attributions. Whole blood spots were then collected from parents and children to assess metabolic and immune markers. For each consenting participant, five full drops of whole blood were collected by sterile lancet fingerstick on Whatman 903TM filter paper cards. The filter paper was dried at room temperature for 4 to 24 hours, then transferred to plastic Ziplock bags with desiccant, stored in a small in-lab freezer, then periodically transferred in batches to long-term storage in a padlocked, -80˚C freezer. Blood spots were assayed using enzyme-linked immunosorbent assay (ELISA; see pg. 26 for detailed information regarding CRP processing procedures). Parents were compensated for their

time at each visit, were offered paid taxi services or reimbursed for transportation, and children were given a small prize.

Measures

Sociodemographics. Parents completed a brief demographic questionnaire to assess child age, child sex, parent age, parent sex, parent highest completed grade level, child education level, race/ethnic background, romantic relationship status. A questionnaire assessing child health history, including current medication regimen was administered to parents. Parents also reported on their family's socioeconomic status, including yearly income, access to federal income assistance, number of family members in home and approximate number of family members supported by their income. Child age and sex were included in the analyses as covariates based on extant prior research confirming their associations with CRP concentrations in children (Cook et al., 2000; Elks & Francis, 2010; Ford et al., 2003; Nakamura et al., 2008; Riis et al., 2016; Wener, Duam, & McQuillan, 2000).

Adverse Childhood Experiences (ACEs). Parents' and children's experiences of adversity before the age of 18 years were assessed via the 10-item Adverse Childhood Experiences Survey (ACEs; Felitti et al., 1998). Parents reported on the number of events experienced early in life (e.g., psychological, physical, or sexual abuse; exposure to family violence, parental substance abuse, mental illness, or incarceration; Felitti et al., 1998) that can result in trauma or chronic stress responses. A higher number of ACEs has been robustly linked to have been shown to relate to negative health outcomes (Anda et al., 1999; Dube et al., 2003; Felitti et al., 1988)

Child Waist Circumference (WC). Child participants' waist circumference (WC) measurements were assessed by trained research assistants by wrapping a tape measurer around the child's waist, immediately above the iliac crest (i.e., the wide part of the pelvic girdle, just above the belly button). Research assistants first felt for hip bone placement on child participants to ensure an accurate measurement, and then recorded circumference in centimeters. A fractional rank of WC by child age and sex was then employed to standardize WC to the child participant sample, and was included in the analyses as a covariate based on prior research confirming its association with CRP (Cook et al., 2000; Elks & Francis, 2010; Ford et al., 2003; Nakamura et al., 2008; Riis et al., 2016; Wener et al., 2000).

Child Chronic Inflammation. Inflammation levels were assessed in whole-blood spots using high-sensitivity enzyme-linked immunosorbent assays (ELISA) to assay C-Reactive Protein (CRP) levels. A 3.2-mm circular punch from each participating child's dried blood spot (DBS) card will be eluted overnight in 250 µl assay buffer. CRP levels are then assessed by ELISA according to the protocol for DBS validated in McDade et al. (2004). CRP remains stable in dried blood spots for at least 5 days at room temperature or 14 days at 4°C and stable for years at −80°C. Next, serum equivalents were calculated using the following algorithm based on the serum-blood spot regression: serum (high-sensitivity CRP) = 1.38 * (blood spot CRP value) – 0.97 (McDade et al. 2007). Observations with values above 10 mg/L indicate frank infection (e.g., Snodgrass et al., 2007) requiring removal from statistical analysis, whereas values below 10 mg/L have been shown to index chronic low-grade inflammation associated with cardiovascular and metabolic risk (Pearson et al., 2003).

Parenting Behaviors. The Dyadic Parent-Child Interaction Coding System-IV (DPICS-IV; Eyberg, Chase, Fernandez, & Nelson, 2013) is a behavioral coding system used to observationally-code parenting behaviors during caregiver-child interactions. The DPICS-IV has been used extensively to track parent skill acquisition in Parent-Child Interaction Therapy (PCIT; Eyberg & Robinson, 1982), however its utility extends to evaluating other parenting interventions and research objectives (Nelson & Olsen, 2018). The detailed behavioral coding definitions are reported in the DPICS-IV manual (Eyberg et al., 2013). Data included for coding was drawn from transcriptions and video recordings of parent-child interactions during the PCIT Dyadic Interaction Assessment described above. Trained DPICS coders coded each 5-minute joint interaction tasks (Child-Led play, Parent-Led play, and Clean-Up), and recorded all parent verbalizations.

The DPICS-IV coding scheme identifies well-defined parent verbalizations into the following categories: Labeled Praise, which includes specific, positive evaluations of a child's appropriate or pro-social behavior (e.g., "thank you for sharing your toys"); Unlabeled Praise, which includes positive evaluations of child behavior that is not specific (e.g., "good job!"); Reflections, which involve restating or paraphrasing a child's pro-social verbalizations (e.g., child "I have a blue block." Parent: "… a blue block"); and Behavior Descriptions, or narrating the child's prosocial play (e.g., "you're putting the green block on top of your tower"). These specific skills reflect parents' positive, warm, attentive behavior toward their child. Several codes also reflect a parent's negative behaviors toward their child, and include: Negative Talk, which describes a critical statement or negative evaluation of their child (e.g., "you're drawing an ugly picture"); Direct Command, which describes a clearly stated order, demand, or direction that is

specific to direct the child's behavior (e.g., "put your toys away now"); Indirect Command, which describes an order or demand that can be interpreted as an optional or implied (e.g., "can you put the toy away now?").

For the purposes of the current study, the frequency of parents' total positive skills, which included Labeled Praise, Unlabeled Praise, Behavior Description, and Reflections, were summed across Child Led, Parent Led, and Clean Up joint interaction tasks to generate a composite score of Positive/Warm parenting behavior, referred to as DPICS-IV Positive Parenting in this document. These positive parent verbalizations were summed across all three tasks based on the notion that positive parenting could occur regardless of the context generated by each play scenario. Negative parent verbalizations, including Negative Talk, Direct Command, and Indirect Command, were summed during the Child Led play segment only, and Negative Talk codes during the Child Led, Parent Led, and Clean Up interactive tasks also were included to characterize Harsh/Controlling parenting behavior. It was determined that Direct and Indirect Commands reflected 'negative' parental behavior only during the Child Led task, as this procedure required parents to allow their child to lead the play and follow their choice of activity. Negative Talk verbalizations were summed across the three interactive tasks based on the notion that negative evaluations of the child constitute negative parenting, regardless of context. This combination of negative verbalizations is referred to as DPICS-IV Harsh Parenting.

For exploratory purposes, DPICS-IV proportion counts were also used to capture an additional conceptualization of positive and harsh parenting behavior. Positive parenting using proportional counts of DPICS-IV codes will be referred to as DPICS-IV Positive Proportional Parenting, and harsh parenting using proportional counts of DPICS-

IV codes will be referred to as DPICS-IV Harsh Proportional Parenting. Each measure (DPICS-IV Positive and Harsh Proportional Parenting) included the same combination of verbalizations used to construct DPICS-IV Positive and Harsh Parenting scores, described above (i.e., DPICS-IV Positive Parenting included Labeled Praise, Unlabeled Praise, Reflections, Behavior Descriptions; DPICS-IV Harsh Parenting included Direct Commands, Indirect Commands, Negative talk) across the same interactive play tasks, however they were calculated as a proportion of total parent verbalizations uttered across tasks. The use of proportion scores was considered based on the possibility that the amount of positive versus negative parenting behavior that occurred during the parent-child interactive tasks would only be relevant in relation to the amount of parental verbalizations overall.

DPICS coders were trained undergraduate and graduate level research assistants who completed 20 hours of training, including thorough study of the DPICS-IV coding manual, coding practice via videos and worksheets, and comparing coding to master-coders. Training occurred in weekly meetings and completion of weekly coding homework assignments, and coders continued to meet regularly to maintain 80% inter-rater reliability. All coders were blind to participants' assessment wave and condition group. Reliability coding was completed on 20% (n = 89) of study families and 84% inter-rater reliability was achieved. Of families coded for reliability, 30% (n = 27) were coded for consensus. The DPICS-IV has been used within many clinical populations and has been used broadly to assess changes in relationship quality and parenting resulting from treatment (Eyberg, Nelson, Ginn, Bhuiyan, & Boggs, 2013; Thomas & Zimmer-Gembeck, 2011). The DPICS-IV has gone through psychometric study and refinement

since the first published edition (Eyberg & Robinson, 1983), and has been normed on parents of varying genders (Eyberg et al., 2013) and races/ethnicities (Eyberg et al., 2013; McCabe, Yeh, Argote, & Liang, 2010).

Parental Attributions. Parental attributions were operationalized via two self-report questionnaires. First, the *Parent Attribution Test* (PAT; Bugental et al., 1989) is a 26-item measure of caregivers' perceived control (or power) in influencing caregiving outcomes. Parents are asked to rate the importance of potential causes of success or failure in hypothetical caregiving situations. A composite score is generated that conceptualizes a parent's perceived control within the parent-child relationship (i.e., the degree of power or control maintained by the parent versus the degree control attributed to the child; Bugental, 2011).

Multiple-dimensional scaling and factor analyses (Bugental et al., 1989) employed on the PAT revealed four primary components of parental attributions: (a) outcomes that are controllable by adults (e.g., adult effort), (b) outcomes that are not controllable by adults (e.g., adult illness), (c) outcomes that are controllable by children (e.g., child stubbornness) and (d) factors that are uncontrollable by the child (e.g., child having a "bad day"). Factor 1 and Factor 2 (reverse scored) are summed to create an Adult Control Over Failure (ACF) composite. Factor 3 and Factor 4 (reverse scored) are summed to create a second composite score, Child Control Over Failure (CCF). High ACF scores indicate parents perceive themselves to have a high degree of control over outcomes of challenging parent-child interactions, while high CCF scores indicate parents attribute outcomes of challenging parent-child dynamics as controlled by their child. A total score (Perceived Control Over Failure [PCF]) is created by subtracting the

CCF composite score from the ACF composite score, thereby measuring the perceived balance of power. PCF scores (i.e., perceived balance of control over caregiving failure) was used as the variable of parents' attributional style in the current study as a continuous variable (Bugental, 2011; Martorell & Bugental, 2006) given that previous research has linked low perceived control over failure in caregiving with more controlling, coercive discipline and increased caregiving problems (i.e., physical abuse; Bondy & Mash, 1999; Bugental et al., 1989). Negative or low PCF scores (referred to as "Low Perceived Control") suggest that parents perceive themselves to have less control than their children over negative caregiving outcomes (Bugental et al., 1989). Conversely, higher scores indicate that parents perceive themselves to have more control than their children (referred to as "High Perceived Control"). PCF scores are referred to as "Control Attributions" in the current study to conceptualize the degree of perceived control parents maintain over negative outcomes of hypothetical parent-child interactions.

The PAT has been found to be relatively immune to social desirability biases (Lovejoy, Verda, & Hays, 1997) and remains uninfluenced by preconceived self-representations. Bugental (1993) reported initial alpha coefficients within a sample of middle-class mothers as: Child controllable $a = .66$; Child uncontrollable $a = .40$; Adult controllable $a = .71$; Adult uncontrollable $a = .85$. Test-retest stability of the PAT was reported to be $r = .63$ in a sample of middle-class mothers over two-months. The PAT demonstrates good construct validity as research has consistently shown linkages between low perceived power and negative response patterns by parents, and an increased likelihood of abusive behavior (Bugental & Hapney, 2004; Bugental & Lin, 2001; Martorell & Bugental, 2006).

The second measure used to assess parental attributions is The *Structural Analysis of Social Behavior Intrex Questionnaire* (SASB-Intrex short form; Benjamin, 1974), a 16-item self-report questionnaire that elicits parents' perceptions of how their child acts toward him/her. The SASB characterizes dyadic interpersonal behavior as well as intrapsychic representations, and is comprised by three circumplex surfaces (see Figure 1; SASB simplified model). Each circumplex is defined by the orthogonal dimensions of affiliation (which describes communications on a continuum ranging from loving to hostile) and interdependence (which describes differentiated and autonomy-granting communications to enmeshed, controlling dynamics). Figure 1 provides the eight behavioral combinations of affiliation and interdependence of the simplified SASB model (Benjamin, 1996). Transitive interpersonal responses (labeled in bold; see Figure 1) are communications focused on other, and describe behaviors initiated by one person toward another that are to, for, or about the other person. Intransitive responses (underlined labels; see Figure 1) are communications focused on the self in reaction to other, and describe individual reactions toward another person. In the current study, questions regarding child transitive behavior toward parent were used as the measure of positive and negative parental attributions. Examples of transitive responses on the SASB-Intrex questionnaire include *[my child] likes me and tries to see my point of view; [my child] tells me my ways are wrong and I deserve to be punished; To make sure things turn out right, [my child] tells me exactly what to do and how to do it* . Caregivers enrolled in the study rated the16-items on a scale ranging from 0 (*never/not at all*) to 100 (*always/perfectly*) in increments of 10, indicating how well each statement described their views toward their child. Depending on a parent's unique combination of reported

perceptions toward their child, parents' scores will be categorized into one of eight "clusters" on each surface of the SASB circumplex. In the current study, scores in clusters 1-5 (blaming/criticizing) and 1-6 (controlling/managing) were used to capture parental attributions of their child as negative and controlling, and were summed to create a composite score. Higher scores on the summed SASB cluster 1-5 and 1-6 scores indicate a greater parental perception of their child as hostile and controlling within the parent-child relationship. In the current study, the summed SASB cluster 1-5 and 1-6 will be referred to as "Negative Attributions" to characterize the degree of hostile/controlling perceptions parents hold toward their children.

Additionally, a measure of positive attributions from the SASB Intrex questionnaire was utilized in current study. Each set of ratings of the SASB Intrex questionnaire produces a behavioral profile that comprises two-dimensional scores: a summary weighted affiliation score and weighted autonomy score. The Weighted Affiliation score, referred to as "Positive Attributions" in the current study, reflects the degree of warmth and affiliation a parent perceives their child to demonstrate toward them, thus capturing positive/warm attributions a parent holds toward their child. Higher Weighted Affiliation scores indicate a parent perceive their child as highly warm and affiliative while negative to low scores indicate their child is perceived as less or not warm and affiliative. One-month test-retest reliability for individual cluster profiles is high at $M = .87$ (Benjamin & Cushing, 2000). The factor structure of the SASB circumplex has been shown to conform to the two orthogonal dimensions of affiliation and autonomy (Pincus, Gurtman, & Ruiz, 1998).

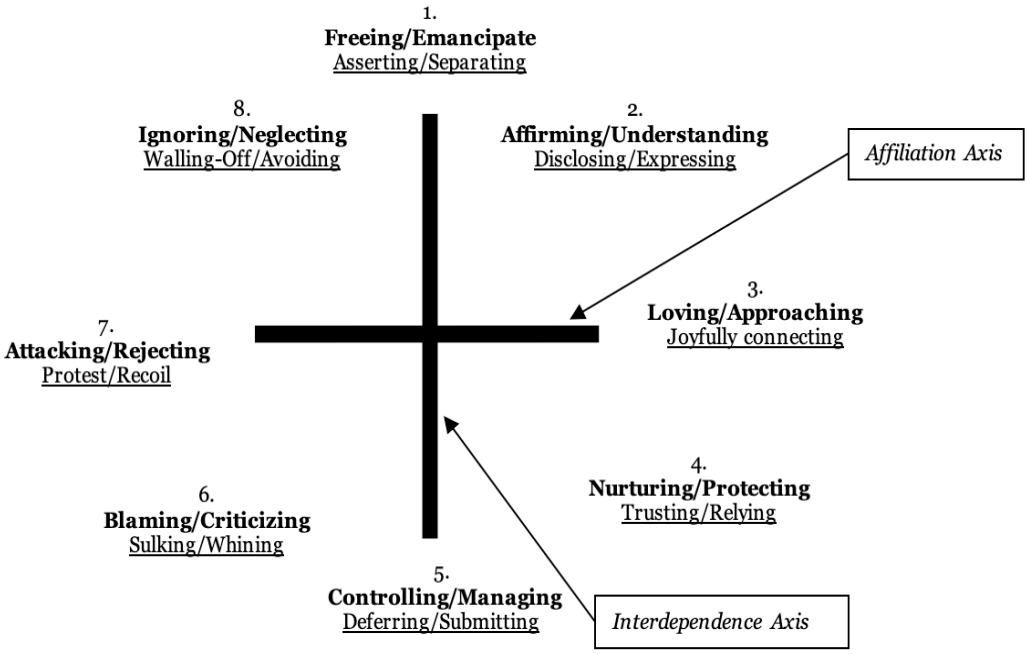


Figure 1. Structural Analysis of Social Behavior (SASB) simplified cluster model. The affiliation axis is the x-axis and the interdependence axis is the y-axis. Labels in **bold print** describe proto-typical parenting behaviors directed toward another person (i.e., child) and are the focus in the present study. Labels in underline print describe proto-typically child-like actions in response to the other (intransitive).

Analytic Strategy

All data analyses were conducted using IBM SPSS version 26.0 (IBM corp, 2019). Data were inspected to assure they met the assumptions for the planned data analyses and screened for patterns of missingness using Little's Missing Completely at Random (MCAR). Preliminary descriptive statistics for all variables and covariates were explored. Associations between CRP and risk variables (e.g., ACEs, child maltreatment status, income), demographic information (e.g., child age, sex, ethnicity, waist circumference), and main study variables of interest (e.g., parental attributions, parenting

behavior) were examined in depth to characterize patterns of CRP concentrations in the study sample. The lack of research on how CRP presents in high-risk children ages 3 to 7 years old remains a significant gap in the current literature; therefore, to address the exploratory aim of the present study, a thorough investigation of relationships that may exist between CRP elevations (or lack of elevations) and key variables in the participant sample was performed.

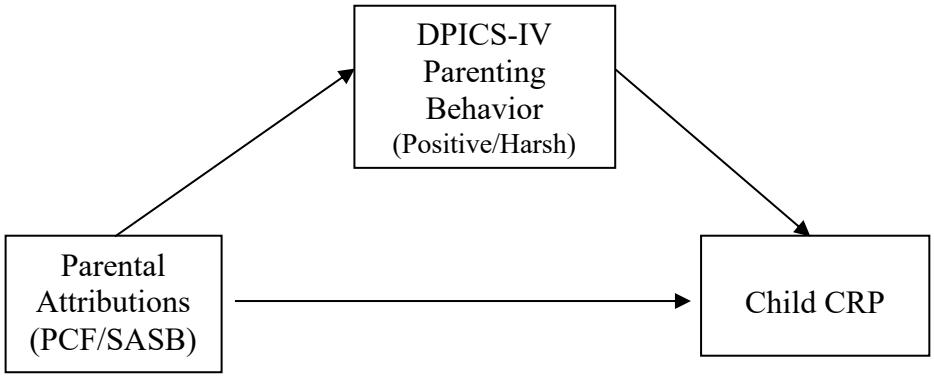
To address the first research question, "Does observed parenting behavior correlate directly with child chronic inflammation?" linear regression analyses were employed with DPICS-IV Positive and Harsh Parenting behaviors predicting child CRP levels while controlling for empirically driven covariates (i.e., child age, sex, waist circumference).

To address the second research question, "Does parenting behavior mediate associations between parental attributions and child inflammation?" a mediation analysis was performed using a regression-based PROCESS approach (Hayes & Preacher, 2013) Multicollinearity diagnostics were used to determine if values were within an acceptable range for conducting this procedure. Four separate mediation models were run, which included PCF Control Attribution scores, SASB Positive Attribution scores, and SASB Negative Attribution scores as the independent, continuous variables, and child CRP as the continuous, dependent outcome variable of interest. The DPICS-IV Positive Parenting and DPICS-IV Harsh Parenting scores served as the mediator variables. Covariates were entered. A bootstrapping procedure was used (Preacher & Hayes, 2008) with parameter estimates based on 10,000 bootstrapped samples. Confidence intervals for the indirect effect were computed with intervals excluding zero indicating statistically significant

indirect effects with $p < .05$ (Shrout & Bolger, 2002). See Figure 2 for the hypothesized mediation models.

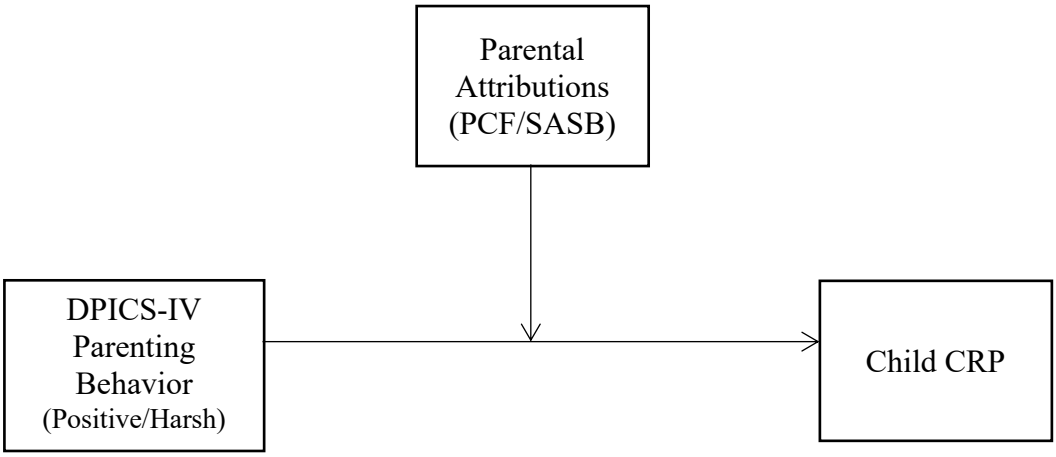
To address the third, competing, research question, "Do parental attributions moderate associations between parenting behavior and child inflammation?" a moderation analysis was performed using the PROCESS macro for SPSS (version 23; Hayes & Preacher, 2013) in four separate models. The independent, continuous variable was DPICS-IV parenting behavior scores, categorized into two levels (i.e., DPICS-IV Positive Parenting scores and DPICS-IV Harsh Parenting scores; see figure 3), and the continuous outcome variable of interest was child CRP levels. The continuous moderator variables were PCF Control Attribution scores, SASB Negative Attribution scores, and SASB Positive Attribution scores. Empirically-informed covariates were entered (i.e., child age, sex, waist circumference). Predictor variables and the interaction terms were grand-mean centered in all analyses. If analyses showed that the interaction was significant, the main effects were not interpreted. A significant interaction between the predictor variables (DPICS-IV Positive/Harsh Parenting scores) and moderator variables (PCF Control Attribution scores, SASB Negative and Positive Attribution scores) would indicate a significant moderation effect; thus, main effects of parenting behavior on child CRP would not be examined. Significant moderation effects were probed by testing conditional effects of DPICS-IV Positive or Harsh Parenting Scores on child CRP levels, at one standard deviation below the mean, at the mean, and one standard deviation above the mean. See figure 3 for the hypothesized moderation models.

Figure 2. Hypothesized mediation model representing the relationship between parental attributions and child CRP, mediated by parenting behavior.



Note. SASB = Structural Analysis of Social Behavior; PCF = Perceived Control over Failure; DPICS-IV = Dyadic Parent-child Interaction Coding System IV; CRP = C-Reactive Protein.

Figure 3. Hypothesized moderation model representing the relationship between parenting behavior and child CRP levels, moderated by parental attributions.



Note. SASB = Structural Analysis of Social Behavior; PCF = Perceived Control over Failure; DPICS-IV = Dyadic Parent-child Interaction Coding System IV; CRP = C-Reactive Protein.

CHAPTER III

RESULTS

Descriptive Statistics

Missing Data. Missing data analyses were conducted using Little's missing completely at random (MCAR) test. Missingness met the assumptions for missing completely at random for all variables, χ^2 (486) = 425.76, p = .98. χ^2 and *t*-tests were utilized to explore differences among CRP missingness and key demographic variables (i.e., child age and sex, parent age and sex, child maltreatment status, child ACEs). No significant differences were found, suggesting no meaningful patterns of missingness in child CRP.

Descriptive Statistics for Child CRP. Raw child CRP concentrations ranged from .00 to 19.35mg/L (*M*= .80, *SD* = 2.39). Two values fell above 10mg/L, which indicate the presence of acute infection (Snodgrass et al., 2007), and as noted above, were thus removed from the sample. CRP was found to be positively skewed (ϒ= 4.60, *SE* = .20); therefore, outliers were winsorized and a natural logarithm transformation was applied to CRP scores prior to analyses to correct for positive skew (ϒ = 1.84, *SE* = .20). Transformed CRP concentrations ranged from 0-1.56 (*M*=.32; *SD* = .37). See figures 4 and 5 for histograms of raw and transformed CRP concentrations.

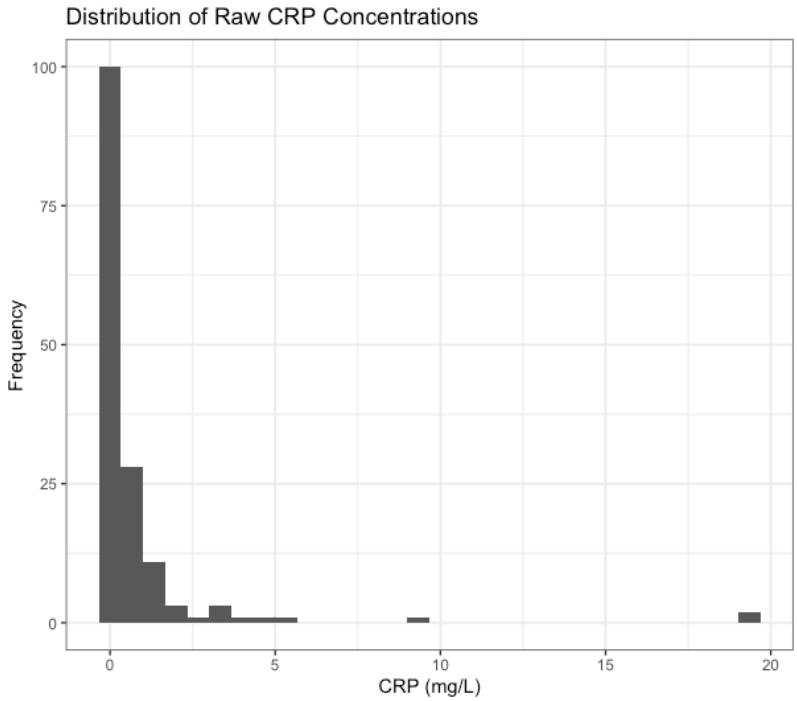


Figure 4. CRP = C-Reactive Protein. Frequency distribution of raw child CRP values. CRP concentrations >3mg/L indicate chronic inflammation.

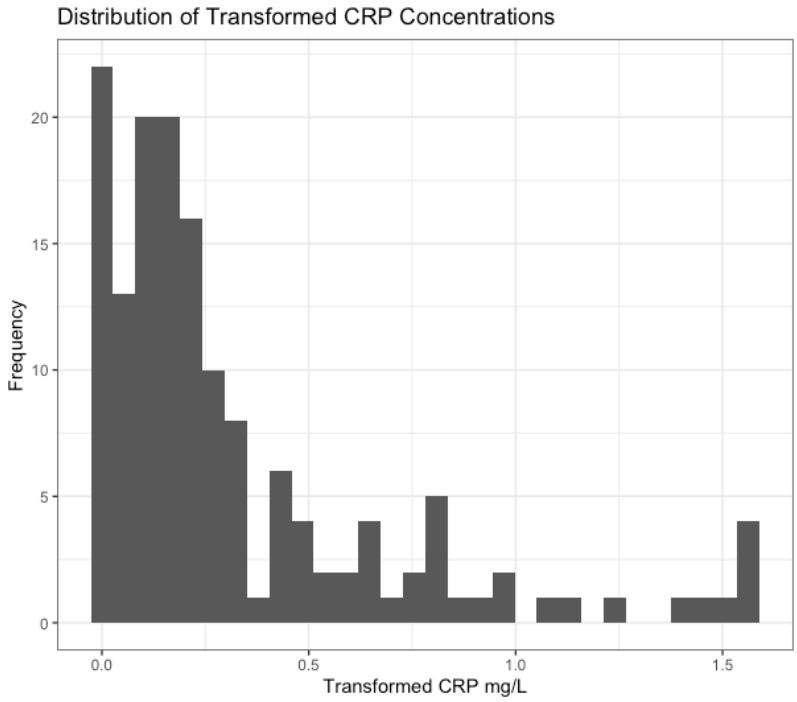


Figure 5. Frequency distribution of transformed child CRP values. CRP = C-Reactive Protein.

Descriptive Statistics for Parenting Behavior and Attributions. DPICS-IV Positive Parenting scores ranged from 36 to 179 (*M* = 102.8, *SD* = 30.11*)* and were normally distributed. DPICS-IV Harsh Parenting scores, however, were slightly positively skewed (ϒ = 2.28, *SE* = .18), thus a natural logarithm transformation was applied (ϒ = .85, SE = .18) and these transformed values were used in the main analyses. SASB Positive Attribution scores, SASB Negative Attribution scores, and PCF Control Attribution scores were normally distributed. Table 2 presents descriptive statistics for raw and transformed CRP values, DPICS-IV Positive Parenting scores, DPICS-IV raw and transformed Harsh Parenting scores, SASB Positive and Negative Attribution scores, and PCF Control Attribution scores.

Table 2. *Descriptive Statistics for Child CRP, Parenting Behavior, Parental Attributions***.**

Variable name	*M*	*SD*	Range
Raw Child CRP	0.80	2.39	0-19.35
Transformed Child CRP	0.32	0.37	0-1.56
DPICS-IV Positive Parenting	102.82	30.11	36-179
DPICS-IV Raw Harsh Parenting	12.46	10.37	0-71
DPICS-IV Transformed Harsh Parenting	3.26	1.29	0-4.28
SASB Positive Attributions	147.42	52.11	-85.12-210
SASB Negative Attributions	71.41	46.06	0-200
PCF Control Attributions	0.85	0.91	-1.83-3.83

Note. DPICS-IV= Dyadic Parent-Child Interaction Coding System-IV; higher values for DPICS-IV Harsh Parenting scores indicate a higher frequency of summed harsh parenting behaviors toward child. Higher values for DPICS-IV Positive Parenting scores indicate a higher frequency of summed positive parenting behaviors toward child. CRP =

C-Reactive Protein; higher values indicate greater concentration of CRP in dried blood spots. SASB= Structural Analysis of Social Behavior; higher values of SASB Positive Attribution scores indicate greater presence of warm/affiliative attributions parents hold toward child. Higher values of SASB Negative Attribution scores indicate greater presence of negative attributions parents hold toward child. PCF Control Attributions = Perceived Control over Failure; higher values indicate parents perceive themselves as maintaining a greater degree of control than their child in the parent-child relationship (High Perceived Control), lower values indicate parents perceive their child as holding more control (Low Perceived Control).

Characterizing Relations Among Child CRP Scores, Sociodemographic, and Parenting Factors. Bivariate correlations were examined prior to main analyses to assess associations among predictor variables and potential covariates (i.e., child age, child sex, child waist circumference). Parent age, parent sex, child ACEs, parent ACEs, and yearly household income were also examined to explore relationships among CRP and contextual risk factors. Table 3 presents Pearson's correlation coefficients, which illustrate that higher SASB Positive Attribution scores correlated negatively with DPICS-IV Harsh Parenting scores ($r(174)= -.19$, $p < .05$) and SASB Negative Attributions ($r(183) = -.24$, $p < .01$). Older parents displayed more positive parenting behavior (DPICS-IV Positive Parenting scores; $r(176)= .17$, $p < .05$) and had younger children ($r(185) = -.16$, $p < .05$). Children with higher reported ACEs were older ($r (185) = .22$, $p < .01$), and had parents with more self-reported ACEs ($r(185) = .30$, $p < .01$). Last, yearly income negatively correlated with parent ACEs ($r(153) = -.16$, $p < .05$), suggesting that parents who reported greater exposure to adversities in childhood also reported lower yearly income.

Figures 6-20 present associations observed between raw child CRP concentrations and key demographic variables (i.e., child sex, age, waist circumference, ethnicity), and main study variables of interest (i.e., DPICS-IV Harsh and Positive Parenting scores,

SASB Positive and Negative Attribution scores, PCF Control Attribution scores). No significant correlations were found between child CRP values and child age ($r(148) = -.03$, $p = .71$; see figure 6), waist circumference ($r(148) = .14$, $p = .09$, see figure 9), ACE exposure ($r(148) = .02$, $p = .78$; see figure 12), DPICS-IV Harsh Parenting scores ($r(141) = .03$, $p = .6$, see figure 16) or DPICS-IV Positive Parenting scores ($r(141) = -.04$, $p = .62$, see figure 17), SASB Positive Attribution scores ($r(147) = .11$, $p = .12$, see figure 19), SASB Negative Attribution scores ($r(147) = -.13$, $p = .11$, see figure 18), or PCF Control Attribution scores ($r(146) = -.08$, $p = .35$, see figure 20).

Independent samples *t*-tests were used to assess differences between child sex, child ethnicity, parent sex, and child CRP levels (using transformed CRP values). *T*-tests were performed in an exploratory manner to understand associations among child CRP and sample characteristics. No significant differences among sociodemographic variables of interest and child CRP concentrations were found. For instance, the independent samples *t*-test of child sex and CRP concentration indicated no significant difference between males and females $t(148) = .72$, $p = .48$, $d = .12$); figure 7 shows CRP scores in females ($M = .35$, $SD = .40$) and males ($M = .30$, $SD = .36$). Figure 8 presents the null associations observed among child CRP values and age for males and females.

As child ACEs were not significantly correlated with child CRP levels (see Figure 12), ACEs were instead dichotomized into two groups (1 = less than 4 ACEs; 2 = greater than 4 ACEs) to explore if child CRP differed among children with lower ACE exposure versus those with higher ACE exposure. An independent samples *t*-test showed no significant difference $t(148) = .61$, $p = .55$. Figures 12 present the null associations between mean child CRP concentrations and ACEs, and Figure 13 shows general trends

(although nonsignificant) among mean child CRP concentrations and ACE exposure among males and females.

An independent samples *t*-test was also used to assess a difference between child maltreatment status and CRP levels. No significant difference was found in CRP levels between children with documented instances of maltreatment and those with no maltreatment history ($t(148) = -1.45$, $p = .15$., $d = .07$). Figure 11 present the mean CRP scores for children with maltreatment histories (($M = .30$, $SD = .37$) and those without documented maltreatment ($M = .40$, $SD = .38$). Last, figure 14 presents mean child CRP levels for ethnicity group; no differences were found among ethnicity group and mean child CRP levels.

Retained covariates. No significant correlations emerged between covariates and child CRP; however, child age ($r(150) = -.03$, $p = .71$), waist circumference (WC) ($r(149) = .14$, $p = .09$), and sex $t(148) = .72$, $p = .48$, $d = .12$), were retained in the main study analyses as covariates given prior theory and research demonstrating their importance (see analytic plan, pg. 35; Cook et al., 2000; Elks & Francis, 2010; Ford et al., 2003; Nakamura et al., 2008; Riis et al., 2016; Wener et al., 2000). In the current study, a fractional rank of WC by child age and sex was employed to standardize WC to the study sample. Raw WC measurements ranged from 20cm to 86 cm ($M = 55.55$, $SD = 7.48$).

Table 3

Bivariate Correlations among Demographic Variables, Parenting Behavior, Parental Attributions, and child CRP.

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Child age	--										
2. Parent age	1.63*	--									
3. Child ACEs	.22**	.11	--								
4. Parent ACEs	.13	.06	-.30**	--							
5. Child WC	.04	.07	.01	.01	--						
6. Yearly income	-.01	-.03	-.14	-.17*	-.01		--				
7. DPICS-IV Harsh Parenting	-.06	.05	.02	.14	-.07	-.13					
8. DPICS-IV Positive Parenting	-.01	.17*	-.01	.65	.36	.92	.73	--			
9. SASB Negative Attributions	.04	.10	.10	.07	-.03	-.08	-.04	.09	--		
10. SASB Positive Attributions	.00	-.01	-.05	.09	-.02	.11	-.19*	.14	-.24**	--	
11. PCF Control Attributions	.05	-.05	.08	.10	.03	.07	-.03	-.05	.09	.03	--
12. Child CRP	-.03	-.10	.02	.04	.14	.13	.04	-.04	-.13	.11	-.08

Notes. ACEs= Adverse Childhood Experiences, WC= Waist Circumference, DPICS-IV= Dyadic Parent-Child Interaction Coding System-IV, SASB = Structural Analysis of Social Behavior; PCF= Perceived Control over Failure (Control Attributions), CRP= C-Reactive Protein (transformed values). $^{**}p < 0.01$, $^{*}p < 0.05$, two-tailed significance.

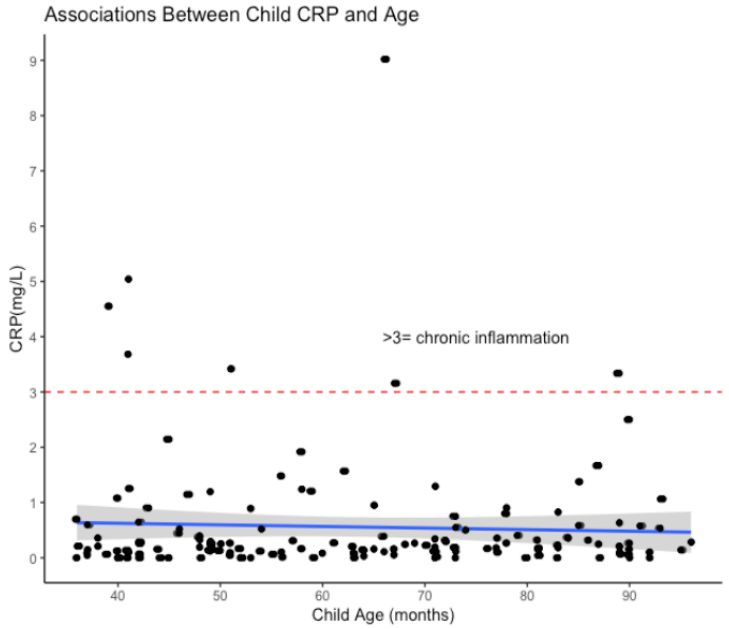


Figure 6. CRP = C-reactive protein. Association between child CRP and child age in months. CRP values >3 mg/L indicate chronic inflammation.

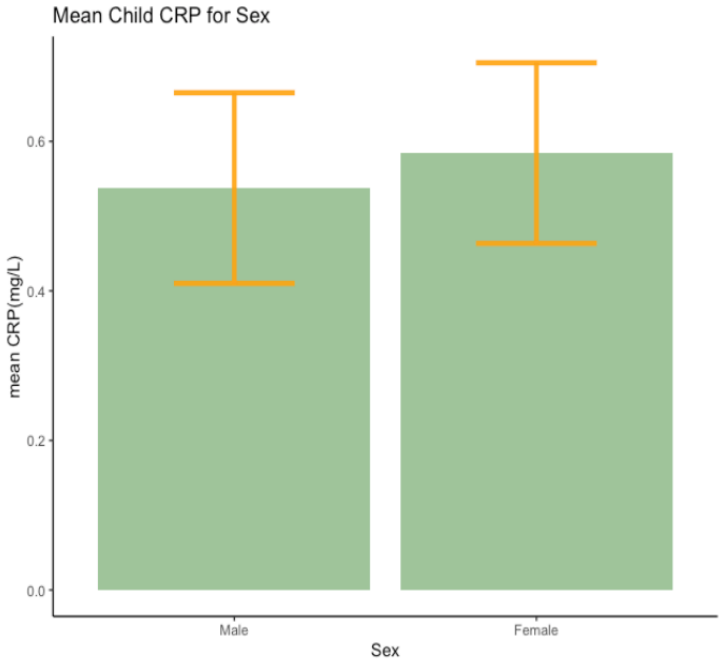


Figure 7. Mean child CRP for males and females. Standard error bars for means represented in orange.

Figure 8. Associations between child CRP concentrations and child age, categorized by sex. CRP concentrations >3mg/L indicate chronic inflammation.

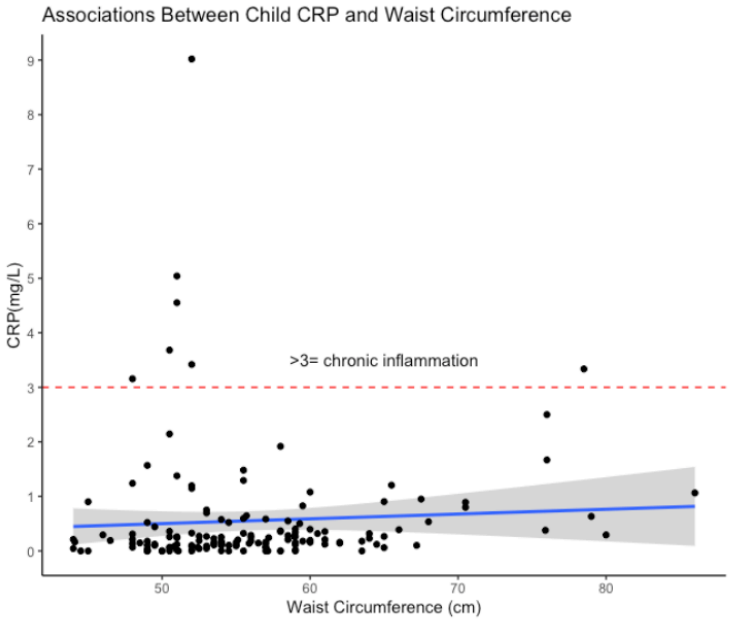


Figure 9. Associations between child CRP concentrations and raw waist circumference (cm) measurements. CRP concentrations > 3mg/L indicate chronic inflammation.

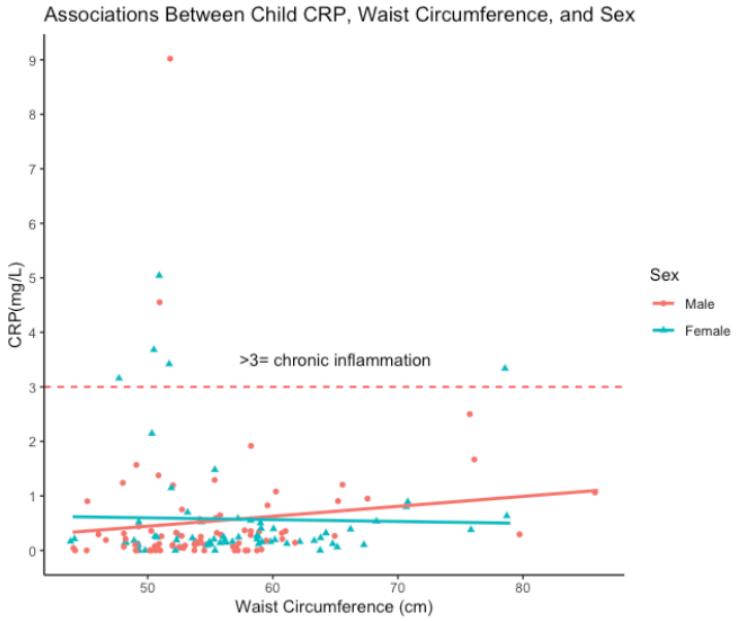


Figure 10. Associations between child CRP concentrations and waist circumference (cm), categorized by sex. CRP concentrations >3mg/L indicate chronic inflammation.

Figure 11. CRP = C-Reactive Protein. Mean child CRP concentrations for children with documented maltreatment histories (n = 139) and without maltreatment (n = 48). Standard error bars for means represented in orange.

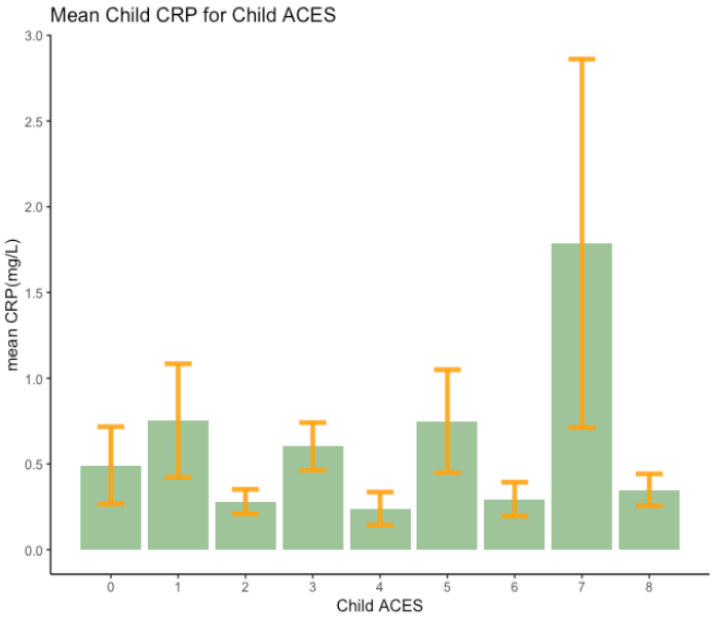


Figure 12. CRP = C-Reactive Protein; ACEs = Adverse Childhood Experiences. Mean child CRP concentrations for number of lifetime ACEs. Standard error bars for means represented in orange.

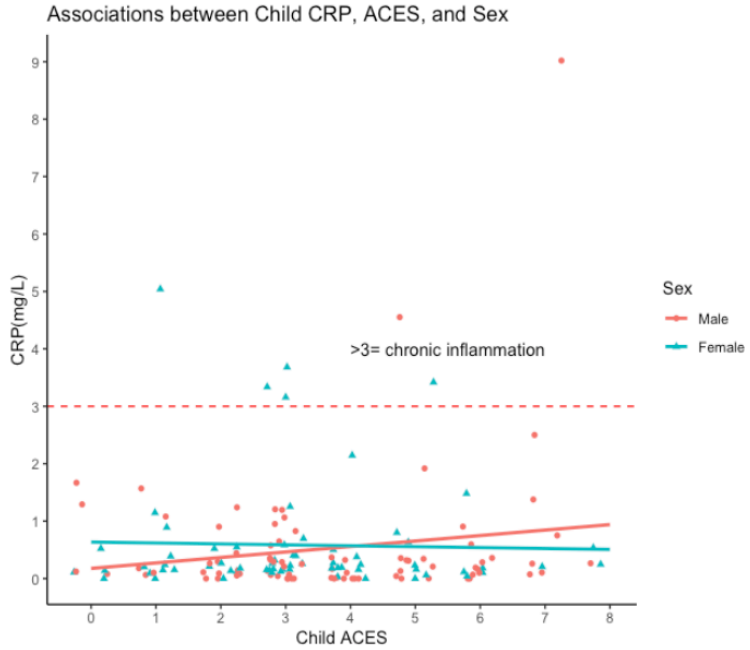


Figure 13. CRP = C-Reactive Protein; ACEs = Adverse Childhood Experi ences. Associations between child CRP and ACEs, categorized by child sex.

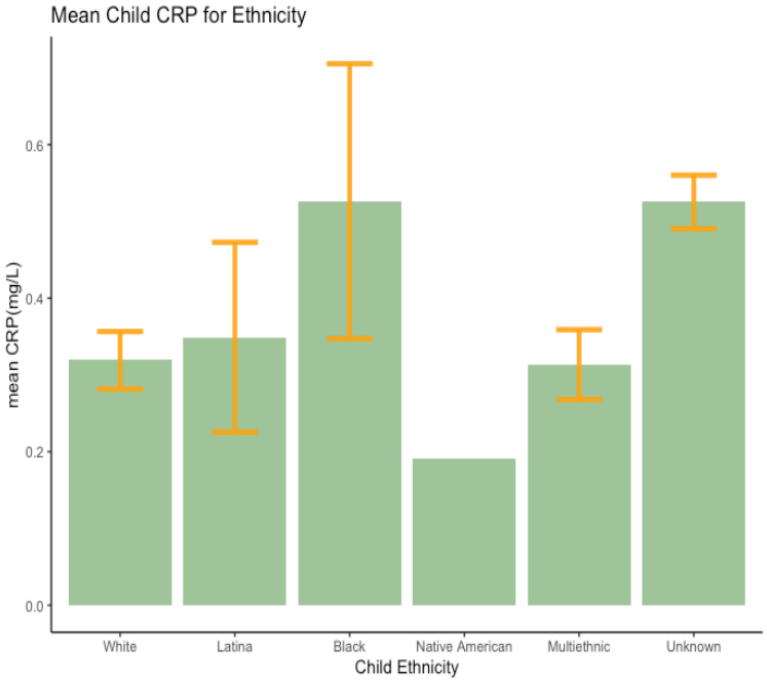


Figure 14. Mean child CRP for ethnicity group. Standard error is represented in orange. Six ethnicity groups were reported: White/ European American (n = 88); Hispanic American/Latino (n = 5); African American/Black (n = 3); Native American (n = 1); More than one ethnicity ("multiethnic"; n = 51); Unknown (n= 2).

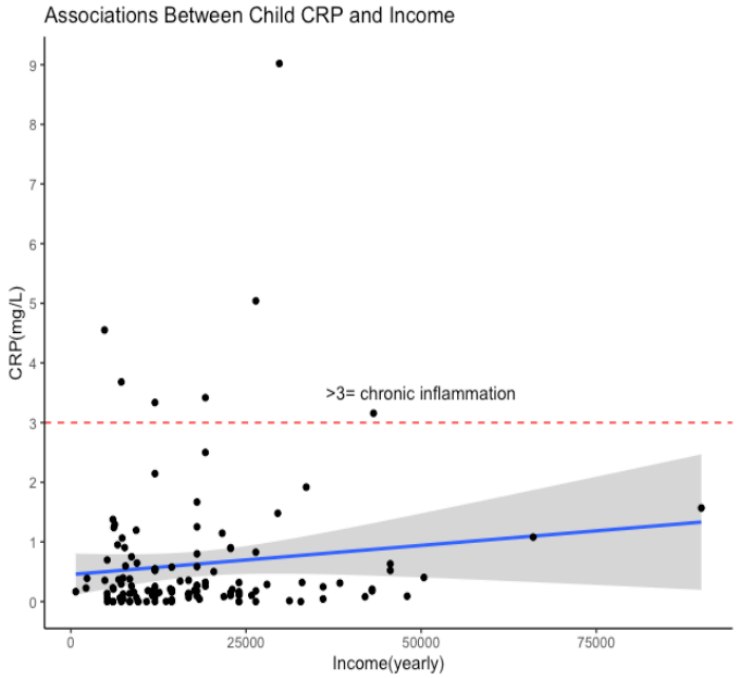


Figure 15. CRP = C-Reactive Protein. Association between child CRP and yearly income.

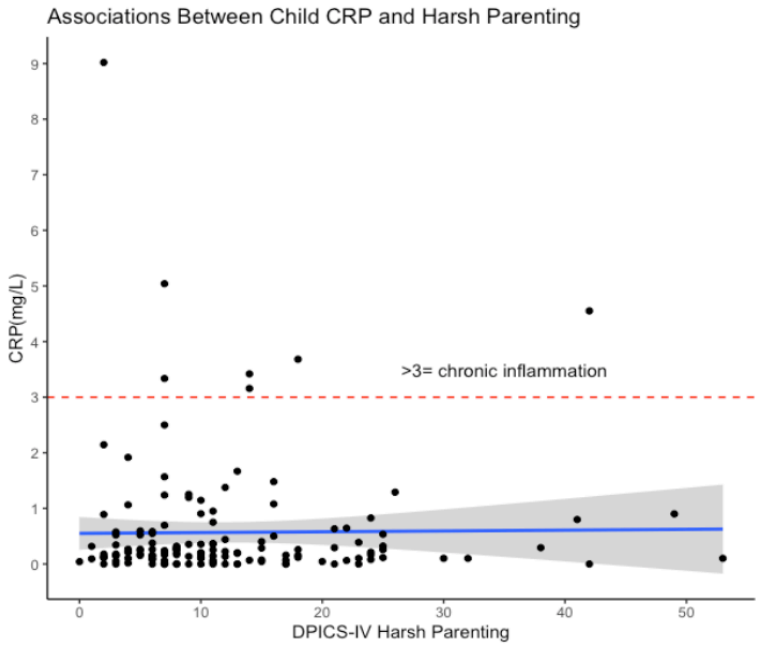


Figure 16. DPICS-IV = Dyadic Parent-Child Interactive Coding System IV. DPICS-IV Harsh Parenting reflects the frequency of observed negative verbalizations (i.e., direct commands, indirect commands, negative talk) parents demonstrate toward their child in joint play scenarios. Higher scores indicate greater frequency counts of observed negative/harsh parenting behaviors.

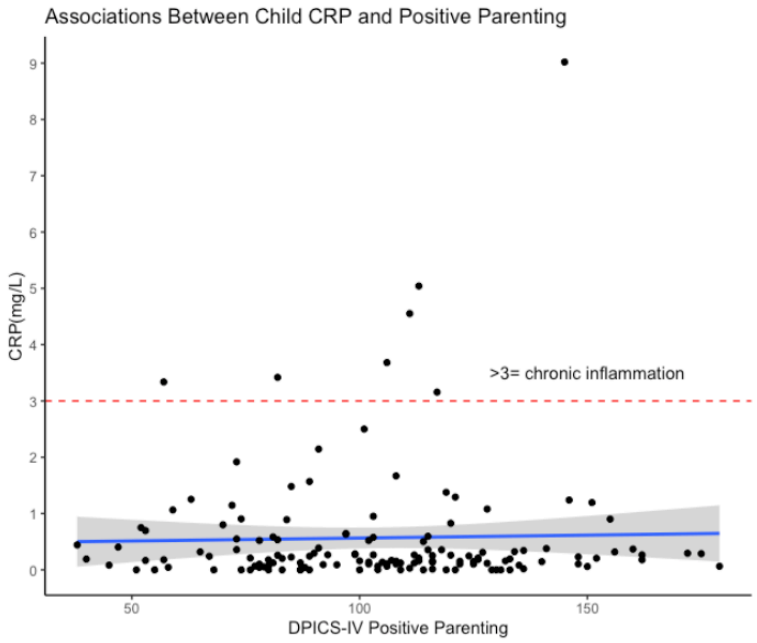


Figure 17. Associations between child CRP and Positive Parenting. DPICS-IV Positive Parenting reflects the frequency of observed positive verbalization (i.e., labeled praise, unlabeled praise, reflections, behavior descriptions) parents demonstrate toward their child during joint play scenarios. Higher scores indicate greater frequency counts of observed positive parenting behaviors.

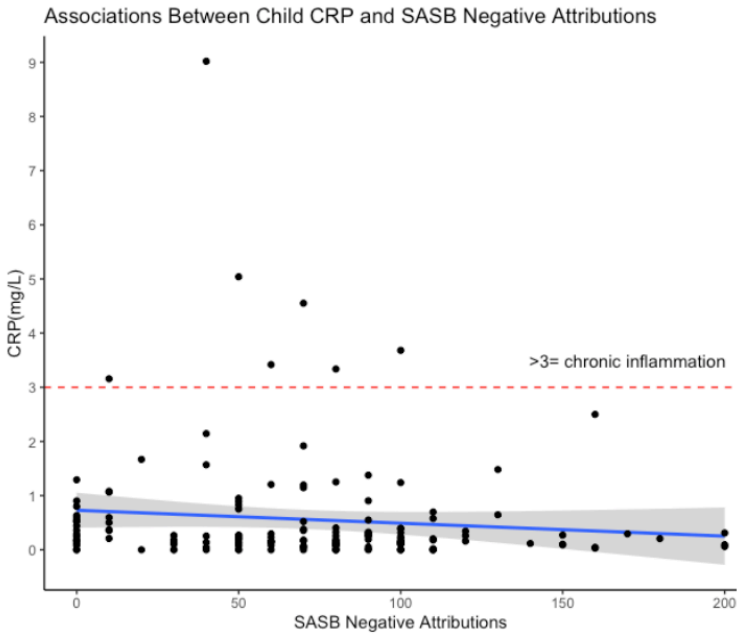


Figure 18. SASB= Structural Analysis of Social Behavior. SASB Negative Attributions reflect the degree of harsh controlling behavior parents perceive their child to demonstrate towards them; higher scores higher levels of hostile/controlling parental attributions.

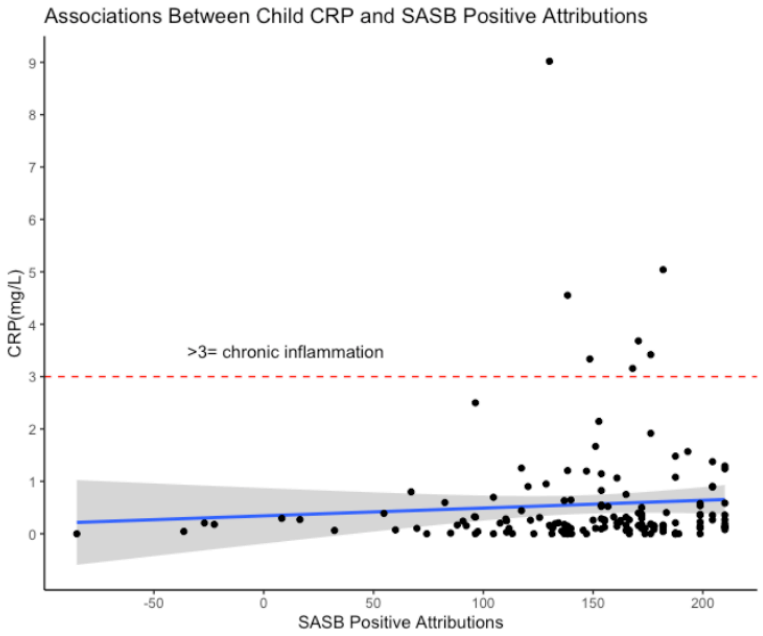


Figure 19. SASB Positive Attributions reflect the degree of warmth and affiliation parents perceive their child to behave toward them. Higher scores indicate higher levels of warm/affiliative parental attributions.

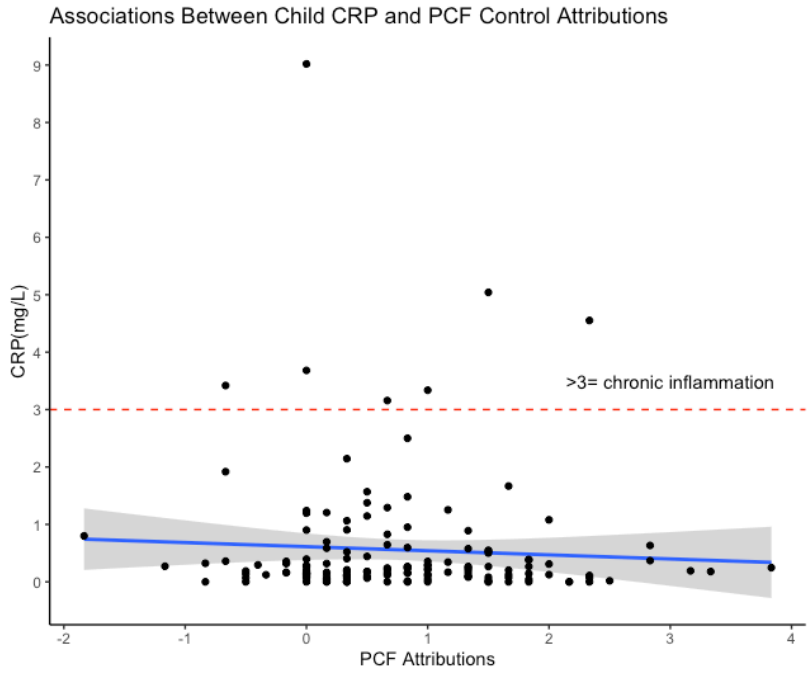


Figure 20. PCF = Perceived Control over Failure. Higher scores (High Perceived Control) indicate parents attribute themselves to maintain a higher degree of control than their child over negative outcomes of parent-child interactions. Lower scores (Low Perceived Control) indicate parents perceive their child to be responsible for negative outcomes of difficult parent-child interactions. CRP = C-Reactive Protein; levels > 3 mg/L indicate chronic inflammation.

Main Analyses

Testing Direct Associations between Parenting Behavior and Child CRP.

Although neither DPICS-IV Positive nor DPICS-IV Harsh Parenting scores exhibited statistically significant bivariate linear correlations with child CRP ($r(141) = .04$, $r = -.04$, $p > .05$, respectively) a linear regression analysis was used to test the hypothesis that that parenting behavior would relate to child CRP levels, such that higher DPICS-IV Harsh Parenting scores would predict higher child CRP while higher DPICS-IV Positive Parenting scores would predict lower child CRP. As seen in Table 4, the step 1 entry of covariates (child age, sex, and waist circumference), were not significantly related to child CRP levels, $F(3, 139) = 1.15$($p = .33$), $R^2 = .02$. Step 2 entry of DPICS-IV Harsh Parenting scores was also not significantly associated with child CRP levels, $F(1, 138) = .96$ ($p = .43$), $R^2 = .03$. Similarly, Table 5 presents null results of a linear regression analysis testing the hypothesis that higher DPICS-IV Positing Parenting scores would predict lower child CRP levels. Namely, there was no evidence that DPICS-IV Positive Parenting scores were associated with child CRP levels. Step 1 entry of child sex, age, and waist circumference was not significant, $F(3, 139) = 1.15$($p = .33$), $R^2 = .02$. Step 2 entry of DPICS-IV Positive Parenting scores was also not significantly associated with child CRP levels, $F(1, 138) = .93$ ($p = .44$), $R^2 = .03$.

Table 4

Summary of Null Findings for Linear Regression Analysis for DPICS-IV Harsh Parenting Predicting Child CRP

	Model 1			Model 2		
Variable	*b*	95% CI	*β*	*b*	95% CI	*β*
Child age	-.01	[-.06, .03]	-.04	-.01	[-.05, .03]	-.04
Child sex	.05	[-.08, .17]	.06	.05	[-.08, .18]	-.04
Child waist circumference	.18	[-.03, .40]	.14	.19	[-.03, .40]	.14
DPICS-IV Harsh Parenting				.03	[-.06, .11]	.05
R^2		.024			.027	
F for change in R^2		1.15			.40	

Note. n=187

Table 5

Summary of Null Findings for Linear Regression Analysis for DPICS-IV Positive Parenting Predicting Child CRP

	Model 1			Model 2		
Variable	*b*	95% CI	*β*	*b*	95% CI	*β*
Child age	-.01	[-.06, .03]	-.04	-.01	[-.06, .03]	-.04
Child sex	.05	[-.08, .17]	.06	.04	[-.09, .17]	.05
Child waist circumference	.18	[-.03, .40]	.14	.19	[-.03, .40]	.14
DPICS-IV Positive Parenting				-.001	[-.003, .002]	-.05
R^2		.024			.026	
F for change in R^2		1.15			.28	

Note. *n*=187

Testing Parenting Behavior as a Mediator of Parental Attributions and Child CRP. Four regression-based mediation analyses implemented with the PROCESS macro version 3 (Hayes, 2017) were used to test the hypotheses that parenting behavior would mediate an association between parental attributions and child CRP. Child age, sex, and waist circumference were included as covariates. As Figure 21 illustrates, there was no evidence to support mediation in this model. The standardized regression coefficient between DPICS-IV Harsh Parenting scores and SASB Negative Attribution scores was not statistically significant, nor was the standardized regression coefficient between SASB Negative Attribution scores and child CRP. The standardized indirect effect was tested using the percentile bootstrap estimation approach with 10,000 samples (Shrout & Bolger, 2002). These results provided no evidence that the indirect coefficient was different from zero, B = -.002 , SE = .01 95% CI = -.03, .01.

Figure 22 presents the hypothesized mediation of SASB Positive Attribution scores and child CRP by DPICS-IV Positive Parenting scores. The model found no evidence of mediation. The standardized regression coefficient between DPICS-IV Positive Parenting scores and SASB Positive Attributions was not statistically significant, nor was the standardized regression coefficient between SASB Positive Attributions and child CRP. The standardized indirect effect was tested using the percentile bootstrap estimation approach with 10,000 samples (Shrout & Bolger, 2002) and emerged as not significant, B = .01 , SE= .01 95% CI = -.04, .02.

Figure 23 presents the hypothesized mediation model DPICS-IV Harsh Parenting scores mediated a relationship between PCF Control Attribution scores and child CRP. The model again found no evidence of mediation. Namely, the standardized regression

coefficient between DPICS-IV Harsh Parenting scores and PCF Control Attributions was not statistically significant, nor was the standardized regression coefficient between PCF Control Attribution scores and child CRP. The standardized indirect effect was tested using the percentile bootstrap estimation approach with 10,000 samples (Shrout & Bolger, 2002). These results indicated the indirect coefficient was not significant, *B* = -.003 , *SE* = .01 95% CI = -.03, .02.

Last, there was no evidence to support the hypothesis that DPICS-IV Positive Parenting scores mediated an association between PCF Control Attribution scores and child CRP. As illustrated in Figure 24, the standardized regression coefficient between DPICS-IV Positive Parenting scores and PCF Control Attribution scores was not statistically significant, nor was the standardized regression coefficient between PCF Control Attributions and child CRP. The standardized indirect effect emerged as not significant, *B* = .00, *SE* = .007, 95% CI = -.02, .01, and was tested using the percentile bootstrap estimation approach with 10,000 samples (Shrout & Bolger, 2002).

Post-Hoc Analyses. Given the null findings observed using raw frequency counts to calculate the DPICS-IV Positive and Harsh Parenting scores, each mediation model was re-run using proportion counts of DPICS-IV codes to calculate DPICS-IV parenting behavior scores (see pp 25 for details on variable construction with DPICS-IV proportion counts). Thus, rather than DPICS-IV Positive and Harsh Parenting scores representing the total number of positive or negative parental verbalization directed toward their child during the interaction tasks, DPICS-IV Positive and Harsh Parenting using proportion scores represented the degree of positive/negative behavior that occurred in the context of total parental verbalizations. Models rerun using proportions of observed parent behavior

that were positive (DPICS-IV Positive Proportional Parenting) or were negative (DPICS-IV Harsh Proportional Parenting), each mediation model similarly emerged as nonsignificant.

Testing Parental Attributions as a Moderator of Parenting Behavior and Child CRP. To test the hypothesis that parental attributions would moderate relations between parenting behavior and child CRP, four moderation analyses using the PROCESS macro for SPSS (version 3, Hayes 2017) were employed. Child age, sex, and waist circumference were again entered as covariates. Table 6 illustrates the first moderation analysis which examined child CRP as the outcome variable, DPICS-IV Harsh Parenting scores as the predictor variable, and PCF Control Attribution scores as a moderator. There was some evidence to suggest that the interaction between DPICS-IV Harsh Parenting and PCF Control Attribution scores accounted for a small proportion of the variance in child CRP concentrations, $\Delta R = .03$, $F(1, 132) = 3.74$, $p = .05$, 95% CI [-.001, .101]. Figure 25 depicts the interaction plot of PCF Control Attributions as a moderator between DPICS-IV Harsh Parenting scores and child CRP. The effect of DPICS-IV Harsh Parenting on children's CRP concentrations was strengthened by the presence of High PCF Control Attributions. DPICS-IV Harsh Parenting scores was related to higher levels child CRP only in the presence of High PCF Control Attributions.

A simple slopes analysis was used to probe the interaction at 1 SD below the mean PCF Control Attribution score, at the mean, and 1 SD above the mean. The unstandardized simple slope for parents 1 SD below the mean of PCF Control Attributions was $b = -.02$, $t(132) = -.60$, $p = .55$; the unstandardized simple slope for parents with a mean level of PCF Control Attributions was $b = .03$, $t(132) = 1.0$, $p = .32$;

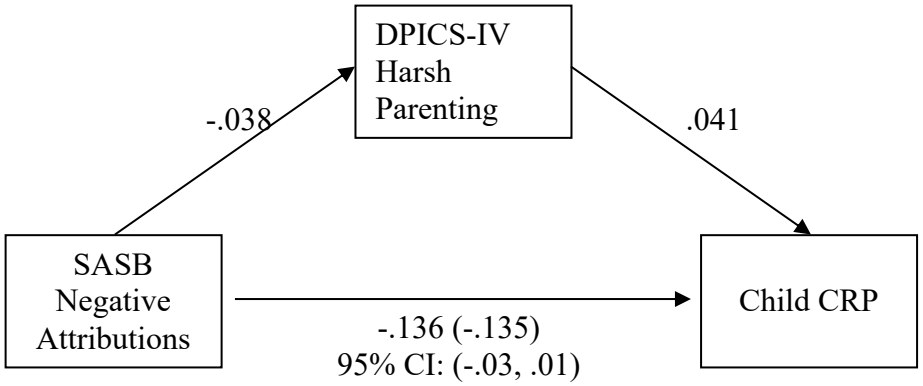
and the unstandardized simple slope for parents 1 SD above the mean of PCF Control Attributions was $b = .07$, $t(132) = 1.89$, $p = .06$. In other words, at low levels of PCF Control Attributions, there was no evidence that DPICS-IV Harsh Parenting had an effect on child CRP; however, at high levels of PCF Control Attributions, DPICS-IV Harsh Parenting had a significant trending effect on child CRP. Examination of the Johnson-Neyman conditional effects table showed that at a PCF score of 1.28 the interaction between DPICS-IV Harsh Parenting and child CRP became significantly related, $b = .09$, $t(132) = 1.98$, $p = .05$. As PCF Control Attribution scores increased, the relationship between DPICS-IV Harsh Parenting and CRP became significantly more positive, with the highest PCF Control Attribution scores $b = .18$, $t(132) = 2.05$, $p = .04$, 95% CI [.006, .350]. Figure 26 presents the interaction between DPICS-IV Harsh Parenting scores and PCF Control Attribution scores plotted within the region of significance. Both upper and lower confidence intervals excluded zero when PCF Control Attribution values equaled 1.28 or above. The correlation between DPICS-IV Harsh Parenting and PCF Control Attribution scores was positive within the region of significance, indicating that greater harsh parenting related to higher child CRP levels in the presence of high perceived control attributions.

Table 7 shows the second moderation model, with child CRP as the outcome variable, SASB Positive Parenting scores as the predictor variable, and PCF Control Attribution scores as a moderator. The interaction between SASB Positive Parenting scores PCF Control Attribution scores was not significant ($\Delta R = .004$, $F(1, 133) = .55$, $p = .46$). Similarly, the third moderation model, with child CRP as the outcome variable, DPICS-IV Harsh Parenting scores as the predictor variable, and SASB Negative

Attribution scores as the moderator emerged as nonsignificant (see table 8), as the interaction between DPICS-IV Harsh Parenting and SASB Negative Attribution scores did not reach statistical significance, $\Delta R = .0001$, $F(1, 134) = .009$, $p = .92$. Finally, the fourth moderation model examining SASB Positive Attributions as a moderator of DPICS-IV Positive Parenting scores and child CRP, emerged as non-significant (see table 9). The interaction between DPICS-IV Positive Parenting scores and SASB Positive Attribution scores did not account for a significant proportion of the variance in child CRP, $\Delta R = .001$, $F(1, 134) = .164$, $p = .67$.

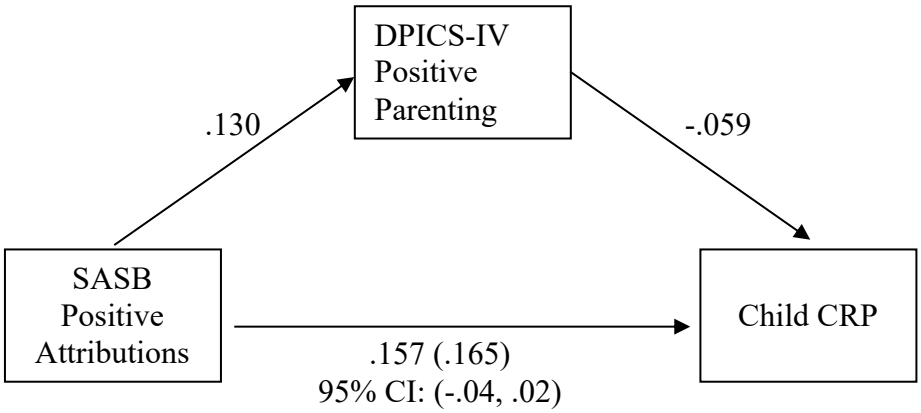
Post-Hoc Analyses. Consistent with the post-hoc analyses performed for the mediation models described previously, each moderation model was similarly re-run with positive and negative parenting behavior operationalized as proportions of observed parenting behaviors rather than raw frequency count of DPICS-IV skills (i.e., DPICS-IV Positive Proportional Parenting; DPICS-IV Harsh Proportional Parenting). Each moderation model similarly emerged as nonsignificant.

Figure 21. Mediation model representing the relationship between SASB Negative Attributions and child CRP levels, mediated by DPICS-IV Harsh Parenting behavior.



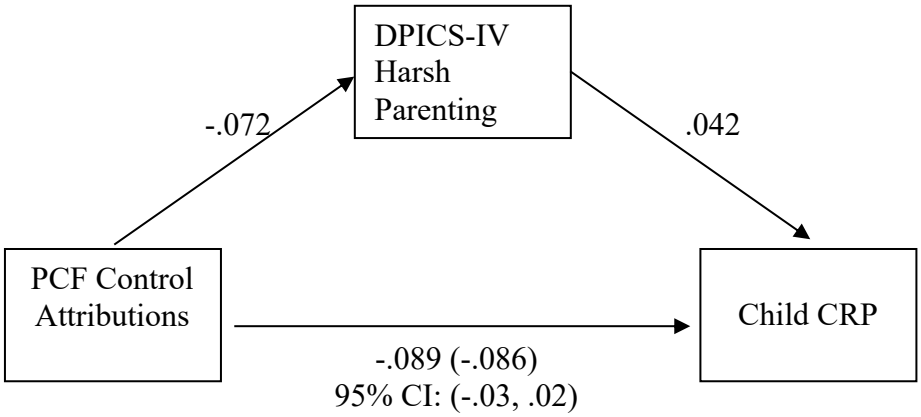
Note. *n*= 141. Standardized regression coefficients for the relationship between SASB Negative Attributions and child CRP as mediated by DPICS-IV Harsh Parenting. The standardized regression coefficient between SASB Negative Attributions and child CRP, controlling for DPICS-IV Harsh Parenting, is in parentheses. 95% Confidence Intervals for the completely standardized indirect effect are included above.

Figure 22. Mediation model representing the relationship between SASB Positive Attributions and child CRP levels, mediated by DPICS-IV Positive Parenting behavior.



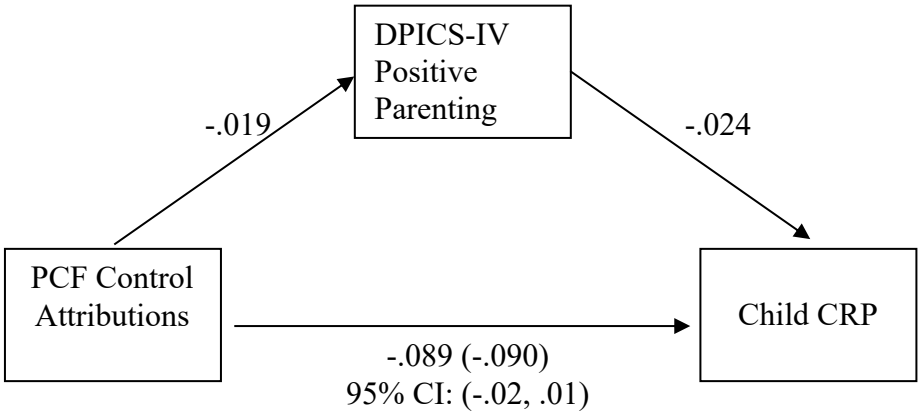
Note. *n*= 144.

Figure 23. Mediation model representing the relationship between PCF Control Attributions and child CRP levels, mediated by DPICS-IV Harsh Parenting behavior.



Note. n = 140.

Figure 24. Mediation model representing the relationship between PCF Control Attributions and child CRP levels, mediated by DPICS-IV Positive Parenting behavior.



Note. n = 140.

Table 6

Hierarchical Regression for Moderation of DPICS-IV Harsh Parenting and Child CRP by PCF Control Attributions

Variable	ΔR^2	*F*	*b*	*SE B*	95% CI	*t*
Model Summary	.04	1.08				
Child age			-.003	.02	[-.05, .04]	-.12
Child sex			.06	.07	[-.07, .02]	.98
Child WC			.11	.11	[-.11, .33]	.97
DPICS-IV Harsh Parenting			.03	.03	[-.03, .08]	.1.0
PCF Control Attributions			-.05	.04	[-.12, .02]	-1.31
DPICS-IV Harsh Parenting x PCF Control Attributions	.03	3.74†	.05	.03	[-.001, .10]	1.93†

Note. n=140; †p = .05.

Table 7

Hierarchical Regression for Moderation of DPICS-IV Positive Parenting and Child CRP by PCF Control Attributions

Variable	ΔR^2	*F*	*B*	*SE B*	95% CI	*t*
Model Summary	.04	.82				
Child age			-.01	.02	[-.06, .04]	-.45
Child sex			.04	.07	[-.09, .17]	.67
Child WC			.17	.11	[-.05, .40]	1.50
DPICS-IV Positive Parenting			-.003	.001	[-.003, .002]	-.24
PCF Control Attributions			-.03	.04	[-.10, .04]	-.96
DPICS-IV Positive Parenting x PCF Control Attributions	.004	.56	.001	.001	[-.001, .003]	.75

Note. *n*=140.

Table 8

Hierarchical Regression for Moderation of DPICS-IV Harsh Parenting and Child CRP by SASB Negative Attributions

Variable	ΔR^2	*F*	*B*	*SE B*	95% CI	*t*
Model Summary	.04	1.01				
Child age			-.01	.02	[-.05, .04]	-.40
Child sex			.06	.06	[-.07, .19]	.95
Child WC			.18	.11	[-.04,.40]	1.58
DPICS-IV Harsh Parenting			.02	.05	[-.07, .11]	.47
SASB Negative Attributions			-.001	.001	[-.003, .000]	.45
DPICS-IV Harsh Parenting x SASB Negative Attributions	.0001	.009	-.0001	.001	[-.002, .002]	-.10

Note. *n*=141.

Table 9

Hierarchical Regression for Moderation of DPICS-IV Positive Parenting and Child CRP by SASB Positive Attributions

Variable	ΔR^2	*F*	*B*	*SE B*	95% CI	*t*
Model Summary	.05	1.23				
Child age			-.01	.02	[-.06, .03]	-.55
Child sex			.04	.06	[-.09, .17]	.65
Child WC			.20	.11	[-.02, .42]	1.79
DPICS-IV Positive Parenting			-.001	.001	[-.003, .001]	-.75
SASB Positive Attributions			.001	.001	[-.00, .002]	1.79
DPICS-IV Positive Parenting x SASB Positive Attributions	.001	.16	.00	.00	[.00, .00]	-.41

Note. *n*=140

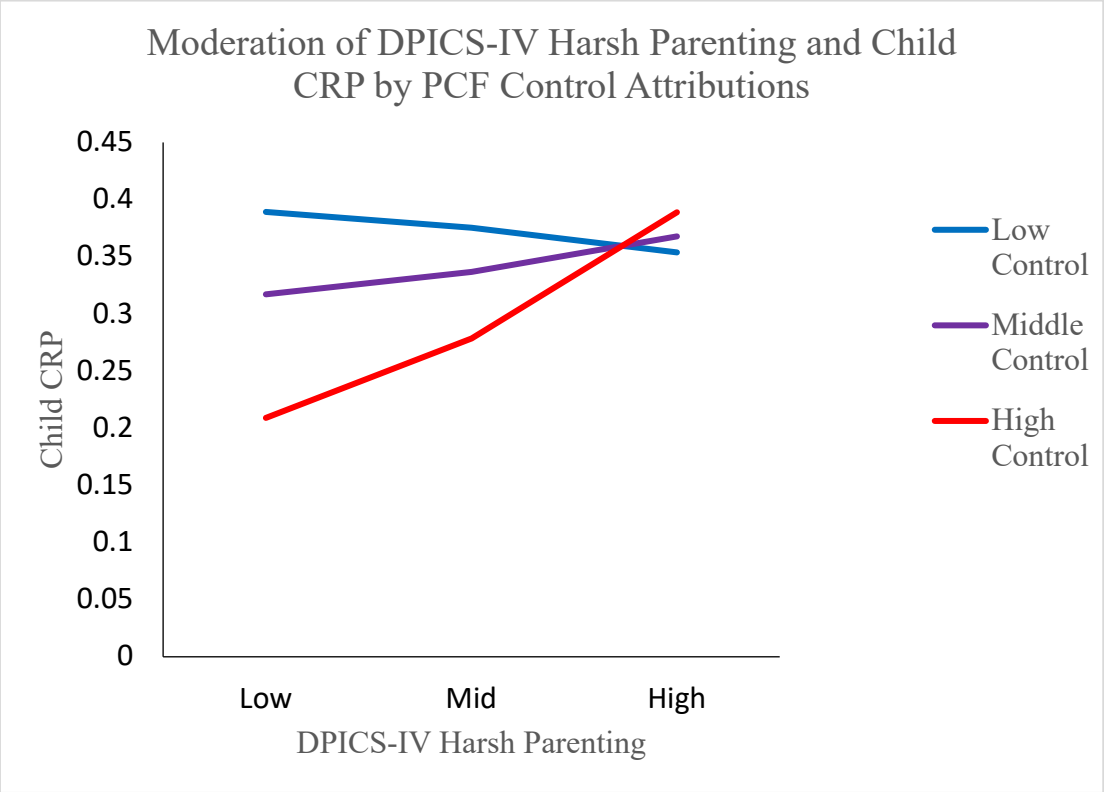


Figure 25. Moderation model representing the relationship between DPICS-IV Harsh Parenting and child CRP levels, moderated by PCF Control Attributions. The effect of parents' harsh behavior (DPICS-IV Harsh Parenting) on their children's inflammation (Child CRP) was strengthened in the presence of High PCF Control Attributions (parents who attributed causes of a negative parent-child interactions to be their responsibility rather than their child's). Where parents perceived themselves more in control, lower levels of harsh parenting predicted lower child CRP values. No significant interaction between DPICS-IV Harsh Parenting and Child CRP concentrations was present under conditions of Low PCF Control Attributions ("Low Perceived Control"; parents who attributed causes of negative parent-child interactions to be the responsibility of their child, rather than their own).

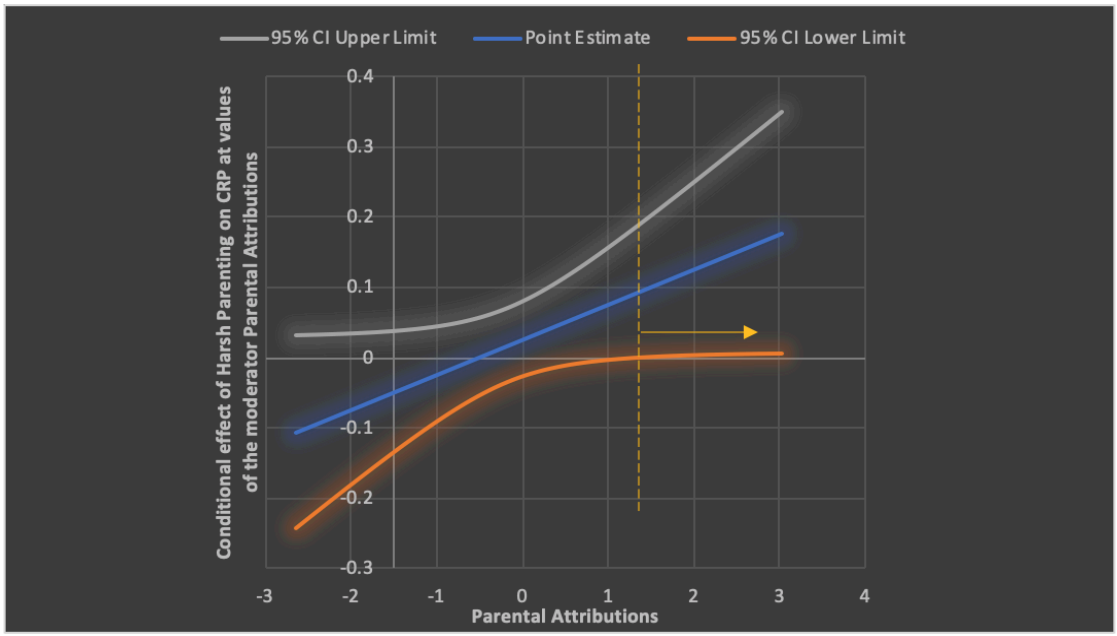


Figure 26. Conditional Effect of DPICS-IV Harsh Parenting as a Function of PCF Control Attributions. The interaction of DPICS-IV Harsh Parenting and PCF Control Attributions is plotted within the region of significance. The correlation between DPICS-IV Harsh Parenting and PCF Control Attribution scores was positive within the region of significance, indicating that greater harsh parenting related to higher child CRP levels in the presence of high perceived control attributions.

CHAPTER IV

DISCUSSION

The purpose of this study was to explore whether and how parents' attributions and behavior toward their child relates to their child's biology (i.e., chronic inflammation) in a sample of high-risk, low-income parents and their 3-7-year-old children. An additional exploratory aim was to understand how experiences of early adversity may relate to concurrent chronic inflammation (i.e., CRP) in early childhood. There is a relative lack of research examining links among the quality of parent-child relationships, family environmental risk, and CRP levels in preschool-aged children; thus, this study sought to clarify questions that remain regarding how early experiences may manifest in children's biological systems during the early childhood years. Understanding whether and how the dimensions of early caregiving (i.e., parents' patterns of behaving and/or perceptions of their child), and experiences of environmental risk, show linkages with children's physiological systems is an important first step in preventing the development of chronic disease states in adulthood. Identification of those most at risk for developing chronic health conditions at the earliest point of detection is critical in preventing lifelong health disparities.

Prior research has evidenced some associations between parenting quality and child chronic inflammation, with elevations in CRP occurring most often in adolescence (Kuhlman, Horn, Chiang, & Bower, 2019). For this study, I was interested in exploring social cognitive factors (i.e., parental attributions) that influence parenting behavior and their associations with chronic inflammation in 3 to 7-year-old children. I reasoned that the ways parents think about their children, including the cognitive schemas parents hold

toward parenting responsibilities in the caregiving relationship, could exert an effect on how they behave, and that attributions and parenting behavior together may uniquely "embed" (e.g., Miller et al., 2011) in the biological systems of their children. I posed two competing hypotheses regarding how parenting behavior and attributions may relate to child inflammation. First, I predicted that parenting behavior may serve a mediating role between parental attributions and child CRP, based on prior research indicating that social cognitive processes may drive parent's behaviors toward their children, thus influencing child outcomes (Crouch et al., 2017; Slep & O'Leary, 1998). Alternatively, I hypothesized that attributions may instead moderate relations between parenting behaviors and child inflammation, as some studies suggest that parents' pre-existing attributional "styles" strengthen or weaken the effect of parenting behavior on child outcomes (Bugental 1987; Bugental 2009; Katsurada & Sugawara, 2000).

No Evidence that Children's CRP is Associated with Demographic or Risk Variables

Preliminary analyses demonstrated that variability in children's chronic inflammation levels were limited in this sample. Of the 152 child participants who provided dried blood spot samples, approximately 25% of children had CRP levels less than .10 mg/L, and 12.5% children had CRP values equal to .00 mg/L. A majority of children (n = 129, 85%) showed CRP values less than 1.0. Only seven children (4.6%) in the study sample had CRP levels between 3mg/L and 10mg/L, which research has established indicates low-grade, chronic inflammation and corresponding increased risk for chronic diseases (Lippy, 2001; Mantovani et al., 2008; Miller et al., 2002). Two children showed CRP values greater than 10 mg/L indicating acute infection (Snodgrass

et al., 2007) and thus were removed from the sample. The distribution of CRP values in this sample are in line with those reported in other published studies of children in a similar age range. Limited variability in blood-level CRP concentrations detected in young children appears to be relatively common (Hostinar, Nusslock, & Miller, 2018; G. Miller, personal communication, March 13, 2020) as most physically healthy children, as well as those who have experienced early adversity, show average CRP levels at approximately 1.0mg/L (Cook et al., 2000; Dowd et al., 2010; Fluori et al., 2020). Further, some studies contain samples of participants who evidence a sizeable percentage of CRP values falling below the lower detection limit of .10 mg/L (37.5%; Broyles et al., 2012; 35%; Dowd et al., 2010). Thus, the small range in CRP concentrations observed here are aligned with other studies showing that CRP is expressed in very low concentrations in both healthy and high-risk children ages 2-9 years old (Broyles et al., 2012, Dowd et al., 2010; Flouir et al., 2020). Hostinar and colleagues (2018) confirm this assertion and propose that assays may in fact lack the sensitivity to detect fine-grained differences in such low levels of CRP (Hostinar et al., 2018). Taken together with the inconsistent findings of the few studies that have examined CRP in high-risk children within the early childhood years, there are several plausible explanations for the limited variability observed in early childhood. It may be that 1) proinflammatory phenotypes provoked by early adversity exhibit latent effects that do not manifest until early adolescence; or 2) inflammatory cytokine production resulting from contextual stress is masked by other markers of stress-response physiology (Hostinar et al., 201; Kuhlman et al., 2019).

In the current study, I aimed to uncover patterns of elevated chronic inflammation that may exist in a sample of high-risk, young children, to better understand if and how adversity influences inflammatory responding during the early childhood years. No significant relationships emerged between sociodemographic or parenting variables of interest within the sample of 3 to 7-year-old high-risk, low income children included in this study. Prior research has observed associations between environmental risk factors and elevated CRP in childhood, including low family income or poverty (Broyles et al., 2012; Schmeer & Yoon, 2015), adverse childhood experiences (Slopen et al., 2013) including child maltreatment (Baldwin et al., 2018), and parenting factors (i.e., problematic parenting behavior; Brody et al., 2014; Byrne et al., 2017). Demographic characteristics including sex (Wener et al., 2000), ethnicity (Ford et al., 2003; Wener et al., 2000), age (Ford et al., 2003), and waist circumference (Cook et al., 2000; Messiah et al., 2012) have also been found to associate with higher chronic inflammation concentrations throughout childhood and adolescence. However, studies confirming associations between sociodemographic variables and inflammation have not focused solely on at-risk children within the age range of 3 to 7 years (Baldwin et al., 2018; Byrne et al., 2017; Schmeer & Yoon, 2015; Slopen et al., 2013), and often include heathy participant samples of younger children, adolescents, and teens (e.g., Cook et al., 2000; Ford et al., 2003, Messiah et al., 2012;Wener et al., 2000). I found that child age, sex, waist circumference, sociodemographic risk variables (i.e., ACEs and family income), and parenting factors (i.e., attributions and behavior) were not statistically related to chronic inflammation in the high-risk young children involved in the current study.

There are several important issues to consider when interpreting these null findings. Most notably, the limited variability observed in child CRP concentrations significantly decreased the ability for meaningful associations to be detected. Moreover, the inconsistencies that exist in the current, limited research on adversity and markers of inflammation in 3 to 7 year old children present many unanswered questions regarding 1) the age at which early adversity begins to influence biomarkers of inflammation and 2) the best collection methods and biomarkers for early identification of later health problems. Kuhlman and colleagues (2019) conducted a comprehensive systematic review of existing research on the relationship between early life adversity and biomarkers of inflammation in infancy through late adolescence and observed minimal evidence of significant findings in studies conducted in early childhood. Associations between early life adversity and CRP in studies included in the review were qualitatively stronger in infant and adolescent participant samples (i.e., Baldwin et al., 2018; Byrne et al., 2017; David et al., 2017; Measelle & Ablow, 2018; Nelson et al., 2017), while those conducted in early childhood revealed mostly null findings and small effect sizes (i.e., Bernard et al., 2018; Hadley & Decaro, 2014; Tyrka et al., 2015). These investigations across infant, early childhood, and adolescent participant samples utilized inconsistent methods for CRP collection, with most studies using salivary measures to assess inflammation (Cicchetti et al., 2015; David et al., 2017; Measelle and Ablow, 2018; Tyrka et al., 2015) rather than blood spot collection. Collection of inflammatory biomarkers via salivary measures has not been validated as widely as blood-based methods have been, and findings across studies may not be comparable (Kuhlman et al., 2019). However, even among studies that utilized a common measure of CRP (i.e., salivary CRP), some found

significant relationships among adversity and CRP (David et al., 2017; Measelle & Ablow 2018) and others did not (Cicchetti et al., 2015; Tyrka et al., 2015). Further, these studies utilizing salivary CRP collection included participant samples of high-risk infants or older children/adolescents and were not specific to the 3 to 7-year-old age range. Investigations have also varied in terms of whether CRP is examined in isolation (David et al., 2017; Hadley & Decaro 2014) or alongside other biomarkers of inflammation, such as the inflammatory cytokines IL-6 and TNF-α (Measelle & Ablow, 2018; Chen et al., 2013). IL-6 and TNF-α may be particularly important inflammatory biomarkers to examine alongside CRP as they are known to stimulate CRP production (Guidice &Gangestad, 2018). However, concentrations of IL-6 and TNF-α measured in circulation are also small in young children (Hostinar et al., 2018), and studies that have examined these biomarkers show inconclusive results as well (Flouir et al., 2020; Heard-Garris et al., 2020). In sum, research that exists on inflammatory markers in high-risk children is limited in the number of studies spread across child age ranges, the inconsistent CRP collection methods, and the measure of inflammation via multiple inflammatory cytokines.

Taken together with the limited variability that exists in young children's CRP concentrations, several conclusions may be drawn to inform future studies in this area. Investigations of young children's chronic inflammation in relation to early experience should a) implement consistent CRP collection methods and a uniform panel of inflammatory biomarkers; b) include participant samples that are adequate in size and similar in age and risk status across multiple studies; and c) consider how alterations in immune development differentially present over time from infancy into childhood,

adolescence, and adulthood. Performing more rigorous, uniform research in this area will deepen our understanding of inflammatory processes associated with risk during early childhood. If limited variability in biomarkers and null findings continue to emerge across well-powered investigations, it will be an important contribution to understanding whether and how proinflammatory phenotypes may emerge in the early childhood developmental period.

Indeed, there is evidence to suggest that a dysregulated inflammatory response to contextual stressors may remain undetected in early childhood due the synchronized activity of other stress-responding systems, such as the hypothalamic-pituitary-adrenal [HPA]-axis and sympathetic nervous system (Bierhaus et al., 2003; Kuhlman et al., 2016, 2019; Steptoe et al., 2007) and their end-products (i.e., cortisol; Kuhlman et al., 2016, 2019). It is well established that psychological stress, in the form early life adversity and problematic family environments, disrupts the functioning of the HPA-axis, thus provoking a downstream inflammatory response (Danese & Lewis, 2017; Kuhlman et al., 2017; Miller et al., 2010). However, cortisol secretion occurs immediately in response to acute stressors (Dedovic et al., 2009) and binds to glucocorticoid receptors in healthy immune cells, which effectively inhibits the production of inflammatory cytokines (Waage et al., 1990; Kuhlman et al., 2019). Thus, children experiencing chronic environmental stressors may exhibit lower inflammatory biomarkers due to the inhibitory effect that glucocorticoids exert on the immune system (Kuhlman et al., 2019). Immune cells may become less sensitive to this inhibitory effect over time (Miller & Chen, 2010), and as they accumulate, inflammatory cytokines are more likely to be observed (such as later in adolescence; Kuhlman et al., 2019). However, few studies have included an index

of HPA-axis functioning along with inflammatory cytokines when examining the effect of early adversity on inflammatory processes in young children. Including measures of cortisol when assessing inflammatory biomarkers would help clarify whether null findings between early adversity and children's current levels of inflammation are explained better by the presence of elevated cortisol (Kuhlman et al., 2019).

In sum, research largely confirms that elevations in CRP in relation to experiences of adversity are observed most strongly in adolescence, and although several studies evidenced CRP elevations emerging in infancy, these studies were drawn from a single participant sample (David et al., 2017; Measelle & Ablow, 2018; Nelson et al., 2019). Adolescence is a critical period where physiological systems experience significant change due to pubertal developmental processes. For example, evidence suggests that the HPA-axis is substantially altered during this developmental stage in part due to pubertal changes (Gunnar & Quevedo, 2007; Tarullo & Gunnar, 2006) and behavioral alterations that occur as a function of adolescence, such as risky behaviors (i.e., smoking, unhealthy eating and exercise habits; Raposa et al., 2014) and impulsivity that increases exposure to psychological stress (Hanson et al., 2015; Lovallo, 2013), decreased parental monitoring and/or shifts in parent-child relationship quality. Thus, the multiple biological and behavioral determinants that occur during adolescence may lead to more meaningful detection of elevated inflammatory biomarkers in risk-exposed youth.

Some Evidence that Attributions Moderate Associations Between Parenting Behavior and Child Inflammation

I examined the hypothesis that attributions may serve as a moderator of parenting behavior and child inflammation. I reasoned that the presence of negative, threat-

sensitive attributions would strengthen associations between harsh parenting and chronic inflammation in children. I also predicted that under conditions of positive attributions, links between parenting behavior and children's chronic inflammation would be weaker. Positive, warm parental attributions and perceptions of parent vs. child control did not emerge as a moderator of positive parenting behavior and child inflammation. Similarly, negative, threat-sensitive attributions did not emerge as a moderator of harsh parenting behavior and child inflammation. However, I found that parents' perceptions of parent vs. child control moderated an association between harsh parenting behavior and child inflammation levels at near significance ($p = .05$). In other words, the effect of parents' harsh behavior on their child's inflammation was strengthened by the presence of high perceived parental control attributions (i.e., parents attributed negative outcomes of difficult parent-child interactions to be their own responsibility, rather than their child's). Harsh parenting behavior was related to higher levels of children's inflammation only in the presence of high perceived parental control attributions. This moderating effect should be interpreted with caution given the limited variability in sample CRP levels and the trend-level significance ($p = .05$). However, interpretation of this finding is meaningful given its novelty within the published literature on parent-child relational quality and children's biomarkers of inflammation.

These findings are consistent with the body of research that identifies parental attributional style as a moderator of parent-child interactions (Bugental, 1987, 1990). The trend-level moderating effect found here supports the notion that parents maintain a set of beliefs about the causes of caregiving successes and failures that in turn influence dynamics with their child (i.e., behavior; Bugental 1987; Bugental et al., 1989; negative

affect; Katsurada & Sugawara, 2000). Research has generally found that the presence of low perceived parental control attributions is most problematic for parents, with studies showing that parents who endorse lower perceived control are more likely to use more harsh, abusive parenting practices relative to those who endorse high perceived parental control (Bugental et al., 1989; 1987; Katsurada & Sugawara, 2000). The results of this study instead suggest that the presence of low perceived control attributions had no significant effect on the relationship between harsh parenting behavior and their child inflammation concentrations. Instead, harsh parenting was associated with greater chronic inflammation in children only when high perceived parental control attributions were present (i.e., parents believing the negative outcomes of difficult parent-child interactions are their responsibility, that is, within their control). It appears as though the unique combination of high harsh parenting behavior together with high perceived control was worse for children's inflammation.

Perhaps observed harsh parenting is more potent for children when parents held high perceived control attributions because this combination captures evidence of a more authoritarian versus authoritative parenting style. Authoritarian parenting is marked by power assertion without warmth, frequent demandingness with high expectations for compliance, and is often associated with the use of physical punishment (Coplan et al., 2002; Valentino et al., 2012). Indeed, children of authoritarian parents tend to show worse behavioral, emotional, cognitive, and academic outcomes (i.e., Crouch et al., 2017; Shears et al., 2008), and are more likely to be recipients of child maltreatment (Crouch et al., 2017; Valentino et al., 2012). It is possible that the observed harsh parenting effects conditioned on the high control attributional style indicate some evidence of

authoritarian-type parenting relating to children's chronic inflammation. It could be that parenting marked by high behavioral and social-cognitive control was especially harmful for children because it reflected a parent-child dynamic where negatively charged parental authority and control over their child was emphasized. The relationship observed with children's higher levels of inflammation could suggest that stressful, harsh parent-child relationships are one contextual stressor that stimulates a proinflammatory phenotype as a result of dysregulated stress-response physiology (e.g., Miller et al., 2011). However, authoritarian beliefs and practices were not specifically assessed in this study, and child maltreatment status was not significantly associated with children's CRP levels, so these interpretations warrant further investigation to determine their veracity. Future studies should assess parents' authoritarian-specific attributions to uncover if parental beliefs about power and control over their children replicate the moderation effect found in this study.

No Evidence of Parenting Behavior Mediating Associations Between Attributions and Child Inflammation

Last, I theorized that the ways parents think about their child would shape their behavior, thus impacting an index of their child's chronic inflammation. I predicted that negative, threat-sensitive attributions towards one's child would drive harsh, controlling parenting behavior, which would be linked to increased inflammation levels in children. Conversely, I predicted that holding warm, positive attributions about one's child would lead a parent to engage in warm, responsive parenting behavior, and predict lower inflammation in their children. Contrary to my hypotheses, parenting behavior did not emerge as a mediating variable between attributions and child inflammation.

These null findings do not deviate substantially from the current literature on relationships among parental cognitions and parenting behaviors. Although no study to date has examined if an attribution-parenting behavior link affects child outcomes at a biological level, there is a body of work that has explored if cognitions precede, or potentiate, parenting behaviors in general. Research in this area is undecidedly mixed and establishing a linear association between parenting cognitions and practices has proven to be inconsistent (Bornstein, 2019; Coleman & Karraker, 2003; Cote & Bornstein, 200; Goodnow & Collins, 1990, Holden, 2002), suggesting that the relationship between thoughts and behavior is more complex than one simply driving the other. For example, perhaps parents in the current study were able to hold contradictory thoughts about their child (i.e., think of their child negatively) and still act in a developmentally appropriate manner, thus inhibiting cognitions from prompting their behavior. Indeed, some research has suggested that parental self-regulation skills are critical when considering parents' behavioral strategies (Anderson et al., 2007; Johnston, Park, & Miller, 2018; Sturge-Apple, Suor, & Skibo, 2014; Uleman et al., 2008). For instance, a stressful parent-child interaction (i.e., parent command— child noncompliance event) might trigger negative parental attributions (e.g., *"my child is misbehaving to spite me"*); however, a parent skilled in self-regulation of their affect and behavior may override the automatic, negative perceptions and respond with appropriate positive, responsive parenting (Johnston et al., 2018). Parents who are able to regulate emotional and affective responses in stressful parent-child interactions, and successfully inhibit negative cognitive appraisals from evoking problematic parenting practices, are likely to have children that benefit behaviorally, emotionally, and physiologically in response to their

parent's self-management. Studies assessing the effects of self-regulation skills, such as inhibitory control, in the context of parental attributions and behavior are relatively scarce, however, and more investigations are needed to understand the depth of these links and their effects on children's outcomes.

Limitations and Future Directions

The primary contribution of this study stems from the limited variability observed in children's CRP concentrations and the resulting lack of zero-order associations found between indices of adversity, sociodemographic variables, parenting factors, and children's inflammation. These null findings deepen the understand of how chronic inflammation emerges in high-risk young children by suggesting that dysregulation of immune development in early childhood may not be readily apparent when measured by one single marker of inflammation (i.e., CRP) or without considering the cross-talk of other developing physiological systems (i.e., HPA-axis). Further, the null findings observed here support recently published research that similarly observed null, or weak, associations between early adversity and markers of inflammation in high-risk young children (i.e., Fluori et al., 2020; Kuhlman et al., 2019; Tyrka et al., 2015). However, the research that exists to date mostly includes a wider age range of children (i.e., 5-18; Broyles et al., 2012; 3-9 years; Cicchetti et al, 2015; 9-11; Flouri et al., 2020) who have experienced differing contextual stressors (i.e., child maltreatment, poverty, ACEs, or a combination), with inconsistent methods for CRP collection (i.e., salivary versus blood spot collection), or an inconsistent panel of inflammatory cytokines (i.e., CRP, IL-6, TNF-α), and these studies have produced mixed results. Thus, it is unknown whether an adversity-related proinflammatory phenotype emerges during early childhood or persists

across development. In order to answer this question, there is a critical need for researchers to examine inflammation in the early childhood years, specifically in the 3 to 7-year old age range, with longitudinal designs to asses when in development chronic inflammation emerges in relation to adversity. Studies would also do well to include comprehensive measures of immune development, such as a panel of inflammatory cytokines as well as other measures of stress-response physiology (i.e., cortisol), as healthy or dysregulated immune development is evidenced by the interrelated responding of multiple physiological systems (Hostinar et al., 2018; Kuhlman et al., 2019). There is also evidence that elevated inflammation provoked by environmental stress may be more evident at a cellular level (Naviaux, 2014; 2019). Research suggests that mitochondria regulate cellular responding to environmental threat or injury by trigger a healing process (including inflammatory and immune system responses; Naviaux, 2014), and overactivation of this cellular response leads to chronic illnesses (Naviaux, 2019). Thus, measuring cellular responding may be a promising avenue for further investigation to clarify links between early adversity and dysregulated immune development in young children.

To date, no study has examined parental attributions as a component that shapes parent-child relational quality in a way that would exert a measurable impact on their child's inflammation levels. The limited variability in children's CRP largely prevented meaningful exploration of this question; however, this study provides some insight into the relationship between parental attributions and behavior. The results observed here suggest that how parents think about caregiving responsibilities in the parent-child relationship may be an important factor in strengthening or weakening the effect of their

behavioral strategies on their child's outcomes. Moreover, this study points to the importance of considering parental attributions when conceptualizing the parent-child relationship.

This study is not without several limitations. First, parental attributions were collected via self-report questionnaires which are inherently susceptible to response distortion (Couch et al., 2017). Parents in the current study may have been reluctant to report undesirable responses to the SASB attribution questionnaires, resulting in underreporting of negative attributions toward their child. However, the Parental Attribution Test (PAT; Bugental et al., 1989) requires parents to consider hypothetical caregiving scenarios, rather than personal scenarios, which uniquely captures parents' cognitive schemas about caregiving responsibilities in all parent-child relationships. The use of the PAT is therefore a strength of the study, as this measure may not be as subject to response bias because parents are not asked to report their thoughts about their own child (Bugental et al., 1989; 2011). However, further research is needed to understand if parents' responses to hypothetical caregiving situations accurately reflects parental attributions toward their own child, and studies would do well to include multiple measures of attributions to support their findings.

Additionally, some research suggests that parents' thoughts about their children operate outside of their awareness (Johnston et al., 2018; Sturge-Apple et al., 2014). A parent may report an explicit understanding that children's misbehaviors are often unintended; however, when a parent is confronted by child misbehavior in stressful contexts (e.g., in public, in a crowded household, in a dangerous neighborhood), a parent's automatic evaluation of their child might be quite different, and thus exert

different effects on their subsequent behavior. Several studies have focused on examining implicit parental attributions and have found them to be less subject to impression management and more predictive of harsh parenting behaviors (Camilo, Garrido, & Calheros, 2016; Sturge-Apple et al., 2015). As this study relied on parents' self-report, the presence of negative attributions may have been underreported which left true associations obscured. Future studies should consider utilizing an implicit measure of attributions (e.g., Implicit Association Test; Johnson et al., 2017) to accurately capture parents' attributional style.

A strength of the current study was the use of observed parenting behavior rather than relying on parent's self-report. Utilizing in-vivo observations of parent's interactions with their children provides a more naturalistic, unbiased representation of parent behaviors (Haws & Dadds, 2006). However, the DPICS-IV coding system reflects parent verbalizations only and has limited capture of emotional valence in parent-child behavioral interactions. For instance, a parent may verbalize "sit down, now," which would be coded via the DPICS-IV as a "Command," but the emotional valence of this command is not fully captured by the DPICS-IV coding scheme (e.g., if the command was stated in a harsh versus a warm manner). These subtle nuances in parent verbalizations were not incorporated into the parenting behavior constructs utilized in the current study; thus, there is much to be hypothesized when considering the effect of parenting behavior on children in the participant sample. Research would benefit from utilizing more rich, descriptive measures of observed parenting behavior, such as the Structural Analysis of Social Behavior (SASB) behavioral coding system (Benjamin,

1996, 2011), which captures nuances in parenting (i.e., harsh vs. benign forms of parental control) that would better characterize emotion valence in observed parenting behaviors.

Concluding Remarks

The results of the study presented here provide insight into 1) the nature of chronic inflammation in 3 to 7-year old child in relation to contextual risk; and 2) associations among parental attributions, behavior, and children's chronic inflammation. I sought to clarify discrepancies in the current understanding of how elevated CRP emerges in early childhood, given that research in this area is highly inconsistent (Kuhlman et al., 2019). I also aimed to extend the existing knowledge regarding how parenting quality relates to child immune functioning (Miller et al., 2011) by examining the contributions of parental social cognitive processes. Ultimately, these findings point to the need for robust studies with consistent CRP collection methods in at-risk children to determine if chronic inflammation is a reliable marker of dysregulated immune development that follows exposures to adverse experiences. Further, the trending moderation effect of parental attributions found here implies that parental cognitions may be important in contributing to a parent-child dynamic that influences their child's physiological development. Parental attributions and behavior are easily targetable (Bornstein et al., 2018), and thus promising avenues for intervention. In fact, addressing attributions in intervention programs has been found to influence both treatment engagement (Morrissey-Kane & Prinz, 1999), parental behavior change (Sawrikar & Dadds, 2018), and child outcomes (i.e., behavior; Bornsetin et al., 2018). The results of the current study suggest that targeting attributions may offer a useful protective factor for children, however more research is needed to establish whether these social cognitive

factors exert causal influences on children's immune development and downstream physical health well into adulthood.

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