EXPLORING EFFECTS OF PARENT-CHILD INTERACTION THERAPY ON CHRONIC INFLAMMATION IN CHILD WELFARE-INVOLVED PARENTS

by

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A DISSERTATION

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DISSERTATION ABSTRACT

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Title: Exploring Effects of Parent-Child Interaction Therapy on Chronic Inflammation in Child Welfare-Involved Parents

Parent-Child Interaction Therapy (PCIT), an intensive parent-coaching program, has shown powerful behavioral effects on child welfare-involved families. PCIT is known to improve both parenting and child behavior as well as reduce child abuse recidivism among parents who have already abused their child. However, potential benefits of PCIT on improving parents' physical health have not been explored. Given the well-documented link between psychosocial stress and physical health, psychosocially disadvantaged families such as those involved with child welfare are at heightened risk for poor health outcomes. One key pathway whereby psychosocial stress leads to disease is through chronic peripheral inflammation, the immune system's response to repeated, stress-related activation. Several behavioral interventions have shown promise in reducing chronic inflammation in adults. However, no study to date has examined the effects of a parenting intervention on parental chronic inflammation.

In the current study, I aimed to examine whether PCIT may lower a biomarker of chronic inflammation, C-reactive protein (CRP), among a sample of child welfare-involved parents. I further aimed to explore parent risk factors that may moderate

treatment effects. Results provided no evidence that PCIT lowered parent CRP, and tested moderators were also not significant. I review these findings in the context of existing literature on behavioral interventions that may impact stress reactivity and chronic inflammation. Future directions are discussed, including recommendations for future studies to measure inflammation across a full panel of inflammatory markers, to use longitudinal designs to assess inflammation changes as they emerge over time, and to employ study designs that will allow for potential psychological and physiological treatment mechanisms to be assessed.

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CHAPTER I

INTRODUCTION

Parent-Child Interaction Therapy (PCIT), an intensive parent coaching program, has demonstrated powerful effects in vulnerable families, including strengthening positive parenting skills, reducing harsh, aversive parenting, lowering parents' stress, and improving child adjustment (Batzer, Berg, Godinet, & Stotzer, 2018; Chaffin et al., 2004; Chaffin, Funderburk, Bard, Valle, & Gurwitch, 2011; Cooley, Vedorale-Griffin, Petren, & Mullis, 2014; Thomas, Abell, Webb, Avdagic, & Zimmer-Gembeck, 2017; Timmer, Urqzuia, Zebell, & McGrath, 2005). However, to date, the potential physical health impacts of this intervention have not been examined. Given strong evidence for the link between psychosocial stress and physical health (e.g., Umberson, Crosnoe, & Reczek, 2010; Holt- Lunstad, Smith, & Layton, 2010), there is a growing recognition that psychosocially disadvantaged families, such as those involved with child welfare, are at heightened risk for stress-related diseases. Thus, researchers have begun to examine whether psychosocial interventions known to reduce stress and improve behavioral functioning may also improve physiological processes implicated in the development of stress-related illness. The presence of low-grade bodily inflammation due to chronic, stress-induced immune system activation has been identified as one key physiological mechanism linking psychosocial stress to poor health outcomes (Bierhaus et al., 2013; Nusslock & Miller, 2016; Slavich & Irwin, 2014). Moreover, evidence suggests that some behavioral interventions, such as cognitive behavior therapy (CBT) or mindfulness-based treatments, may be able to lower chronic inflammation in adults (Bower & Irwin, 2016; Lopresti, 2017). Thus, in the current study, I propose to investigate potential

impacts of PCIT on a biomarker of chronic inflammation in child welfare (CW)-involved parents.

PCIT Outcomes among Vulnerable Families

Parent Child Interaction Therapy (PCIT) was initially developed as a treatment for disruptive behavior problems among young children (ages 2-7), including issues such as temper tantrums, destroying objects, and defiance toward adults (e.g., Eyberg, 1995). Since its development, PCIT has been successfully applied to treat a range of child presenting problems, including internalizing problems (e.g., Carpenter et al., 2014) and attention-deficit/hyperactivity disorder (ADHD; Matos, Baumeister, & Bernal, 2009), and has been used to treat dyads in which caregiving impairments are present, with or without child behavioral problems (Herschel & McNeil, 2005). In PCIT, caregivers are taught a small set of focused parenting skills across two phases. In the first phase, Child-Directed Interaction (CDI), parents are taught to provide children with praise and positive attention for appropriate behavior and ignore minor misbehavior while following the child's lead in play (Eyberg, Boggs, & Algina, 1995). In the second phase of PCIT, Parent-Directed Interaction (PDI), parents learn a concrete sequence of skills to gain child compliance and manage challenging behavior (Eyberg et al., 1995). Of note, a majority of PCIT sessions are delivered using an intensive, live-coaching format in which therapists give parents instantaneous feedback during play interactions with their child, via a one-way mirror and specialized audio equipment (Borrego & Urquiza, 1998).

In addition to improving child behavioral outcomes (e.g., Thomas et al., 2017), PCIT has shown powerful effects on parenting behavior and parent functioning. For example, in one recent study of 139 Australian parents, self-reported improvements in

parents' own emotion regulation were observed following PCIT (Zimmer-Gembeck, Kerin, Webb, Gardner, Mastro Campbell, Swan, & Timmer, 2019). PCIT's effectiveness in reducing parenting stress has been also been well-documented. In their meta-analysis, Thomas et al. (2017) found effect sizes across 17 studies ranging from a mean difference (MD) of -6.98 to MD of -12.17 on the 36-item Parenting Stress Index (PSI; Abidin, 1995). Of note, PCIT has shown to be effective even among the most psychosocially challenged parent samples. In their 2012 randomized controlled trial (RCT), Thomas and Zimmer-Gembeck assessed PCIT outcomes among a sample of 151 mothers who were at high risk for or had a history of child maltreatment. Participants were screened into the study using an initial semi-structured interview to ensure that all referred parents were currently at high risk to maltreat their child, evidenced by their endorsing a) regular use of corporal punishment, b) high levels of parental distress, and c) high levels of intolerance for their child's behavior. Compared to waitlist controls, mothers who received PCIT reported reductions in their child's externalizing and internalizing behavior, reductions in parenting stress, and were observed to display more warm, sensitive behavior during interactions with their child.

Furthermore, substantial evidence shows that PCIT is effective in reducing future occurrences of maltreatment among parents who have already abused their child, a social problem for which intervention is notoriously difficult (e.g., Chaffin, Bonner, & Hill, 2001). Chaffin and colleagues conducted the first RCT examining PCIT among parents who had already perpetrated child maltreatment (Chaffin et al., 2004, 2011), using a sample of parents with severe, chronic child welfare services histories (i.e., majority of their children removed from their care). In this set of studies, 19% of parents receiving

PCIT had additional reports to child welfare at 18 months post-treatment, compared to 49% recidivism rates among those in the treatment as usual group (Chaffin et al., 2004, 2011). In concordance with this, a meta-analysis of PCIT outcomes among maltreating parents revealed the intervention was associated with lower rates of re-abuse across 11 controlled trials (Batzer, Berg, Godinet, & Stotzer, 2018). Indeed, PCIT may be the only parenting intervention shown to reduce future recidivism among parents who have already abused their child.

Socioeconomic Risk and Psychosocial Stress in Child Welfare-Involved Families

Socioeconomic disadvantage is a strong predictor of child welfare (CW) involvement (e.g., Cameron & Freymond, 2006; Courtney, Dworsky, Piliavin, & Zinn, 2005) and CW-involved families experience high levels of psychosocial stress related to low socioeconomic status (SES) conditions and associated impacts on family functioning. For example, low-income families frequently experience stressors such as transportation problems, financial problems, parent work overload, or family member medical problems (Brown, Seyler, Knorr, Garnett, & Laurenceau, 2016), which can in turn adversely impact parenting (Evans, Eckenrode, & Marcynyszyn, 2009) and lead to parent depression and anxiety symptoms over time (Santiago, Wadsworth, & Stump, 2011). Indeed, low income and CW-involved parents report elevated levels of parenting stress, compared to their higher SES counterparts (Raphael, Zhang, Liu, & Giardino, 2010; Rodriguez-Jenkins & Marcenko, 2014). Parenting stress, defined as the emotional and physical fatigue caregivers experience when demands of parenting are perceived to exceed their ability or resources to cope (Deater-Deckard, 2004), is a particular form of relational stress known to negatively influence parenting behavior and child outcomes

(Crnic, Gaze, & Hoffman, 2005; Gutermuth et al., 2005; Huth-Blocks & Hughes, 2008). Taken together, the existing evidence demonstrates that child-welfare involved families are likely to be exposed to elevated levels of psychosocial stress, including parenting stress, which may leave them at greater risk for poor health outcomes (Matthews & Gallo 2010; Sapolsky, 2004).

Chronic Inflammation and Stress-Related Disease

In recent years, social and medical science have significantly advanced understanding of the neurobiological processes whereby chronic psychosocial stress, particularly in early childhood, disrupts multiple physiological systems, leading to altered stress responsivity and exaggerated immune system response (Miller, Chen, & Parker, 2011; Shonkoff et al., 2012). Within the body's complex, multisystemic response to stress, chronic, low-grade inflammation has been implicated as a key driver in the development of stress-related disease (Bierhaus et al., 2013; Nusslock & Miller, 2016; Slavich & Irwin, 2014). Time-limited periods of acute inflammation in response to infection or injury are an essential, life-saving function of the immune system. However, chronic inflammation, indexed by underlying, low levels of peripheral pro-inflammatory cytokines (e.g., interleukin-6, interleukin-1, tumor necrosis factor alpha) and acute phase proteins (e.g., C-reactive protein), has harmful effects (Slavich, 2015). Chronic, low-grade inflammation has been linked to a host of psychiatric disorders (Haroon, Raison, & Miller, 2012) and diseases, including leading causes of death in the U.S. such as heart disease (Araujo et al., 2009), stroke, diabetes (Hotamisligil, 2006; Ridker, 2007), and certain cancers (Berasain, Castillo, Perugorria, Latasa, Prieto, & Avila, 2009; Yao & Rahman, 2009).

One biomarker of chronic, low-grade inflammation is C-reactive protein (CRP). CRP is a proinflammatory acute phase protein generated in the liver, under the transcriptional control of the cytokine IL-6 (Pepys & Hirschfield, 2003). Circulating levels of CRP, collected through blood plasma samples, represent a non-invasive measure of systemic inflammation. Whereas expected levels of blood plasma CRP in healthy samples range from 0.8mg/L to 3.0 mg/L, CRP can rise to levels as high as 100-500 mg/L in response to acute infection. The development of highly sensitive assay technology has allowed researchers and medical practitioners to detect slight elevations in CRP (e.g., 3.0 mg/L – 10 mg/L) that indicate chronic, low-grade inflammation. This biomarker is recommended for early risk detection and treatment planning among medical doctors by organizations such as the American Heart Association and Center for Disease Control (Pearson et al., 2003) and has increasingly been utilized by social science researchers to deepen understanding of how behavioral and environmental factors may influence immune responses to stress.

Predictors of Elevated CRP

A wide range of physiological, behavioral, and environmental factors can influence circulating levels of CRP and other pro-inflammatory markers. Low-grade inflammation is associated with sleep problems (Irwin, Olmstead, & Carrol, 2016), abdominal adiposity (Brooks, Blaha, & Blumenthal, 2010; Lapice, Maione, Patti, Cipriano, Rivellese, Riccardi, & Vaccaro, 2009) and obesity (Aronson, Bartha, Zinder, Kerner, Markiewicz, Avizohar, Brook, & Levy, 2004). Conversely, physical exercise and nutrient-dense diet are both known to reduce inflammation (Fedewa, Hathaway, & Ward-Ritacco, 2017; Kuczmarski, Mason, Allegro, Zonderman, & Evans, 2013; de Maat &

Kluft, 2001). CRP levels increase with age (de Maat & Kluft, 2001) and are often higher among women (Khera et al., 2009) and non-White ethnic groups (Nazmi & Victora, 2007). Elevated CRP levels are associated with smoking (de Maat & Kluft, 2001; van der Vaart et al., 2005) and exposure to air pollutants (Pilz et al., 2018). There is also a well-documented, inverse association between chronic inflammation and SES (Gruenewald, Cohen, Matthews, Tracy, & Seeman, 2009; Panagiotakos et al., 2004). Some studies suggest the association between socioeconomic disadvantage and inflammation may be best explained by high levels of psychosocial stress experienced by those living in poverty (Hong, Nelesen, Krohn, Mills, & Dimsdale, 2006). Alternatively, other findings indicate the link between low SES and inflammation may be driven by environmental conditions of poverty that impact health more broadly (e.g., through illness or body mass index; Alley, Seeman, Kim, Karlamanga, Hu, & Crimmins, 2006), However, there is growing public interest in the link between chronic, low-grade inflammation and psychosocial stress, given that this factor may be more readily intervened on by health care providers.

Psychosocial Stress and CRP

Chronic psychosocial stress is associated with low-grade inflammation in adulthood (Gouin, Glaser, Malarkey, Beversdorf, & Kiecolt-Glaser, 2012). For example, elevated levels of circulating CRP and inflammatory cytokines have been observed in populations known to face significant, ongoing stressors, such as family caregivers of critically ill patients (Gouin et al., 2012; Sherwood et al., 2016) or immigrants to the United States (Ablow Measelle, McClure, Snodgrass, Martinez, Jimenez, & Isiordia, 2019). In their Social Transduction Theory of Depression, Slavich and Irwin (2014)

outline how experiences of psychosocial stress work to upregulate immune activity, leading to chronic inflammation. Inflammatory activity is regulated by neurological stress response processes, such as activity in the sympathetic nervous system (SNS) and hypothalamic pituitary adrenal (HPA) axis and responds to activity in these systems in order to prepare against physical injury. For example, in the SNS, the release of the neurotransmitter norepinephrine into peripheral tissues results in increased transcription of pro-inflammatory immune response genes (i.e., *IL1*, *TNF*, and *IL6*) and in the release of pro-inflammatory cytokines. While this anticipatory immune response is evolutionarily adaptive and prepared our ancestors for wound healing following a physical attack (Antoni et al., 2012) chronic immune activation in response to present-day social stressors results in long-term inflammation and health risk (Irwin & Cole, 2011; Slavich & Irwin, 2014).

How significant or prolonged must psychosocial stress be in order to lead to peripheral inflammation? There is evidence that stress related to the quality of daily interactions with partners, family members, and friends can influence inflammatory markers (Chiang, Eisenberger, Seeman, & Taylor, 2012; Fuligni, Telzer, Bower, Cole, Kiang, & Irwin, 2009; Marin, Chen, Munch, & Miller, 2009; Kiecolt-Glaser, Gouin, & Hantsoo, 2010). In one study, a sample of 69 adolescents was asked to complete a daily diary in which they reported on their experiences of negative interactions at home and at school, including conflict with family and friends, peer harassment, and punishment by parents or teachers. Blood samples collected 8 months later showed that greater daily negative interactions were associated with higher CRP, even after controlling for body mass index, socioeconomic status, substance abuse, life events, rejection sensitivity,

psychological distress, and frequency of daily interpersonal stressors 2 years prior (Fuligni, Telzer, Bower, Cole, Kiang, & Irwin, 2009). In keeping with Slavich & Irwin's theory (2014), this finding suggests that the experience of social threat and even more subtle elevations in interpersonal stress are, indeed, salient to the immune response, with critical implications for long-term health. Given this, interventions that reduce interpersonal stress and improve the quality of key relationships may also have the potential to confer important health benefits.

Behavioral Interventions Can Impact Physiological Stress Response

A growing body of evidence suggests that psychosocial interventions can effect change in neurobiological stress systems, as well as behavior. For example, Dozier and colleagues have found that their attachment-based intervention, Attachment and Biobehavioral Catch-Up (ABC), which targets caregivers' support of young children's regulatory capabilities, also normalizes cortisol production among maltreated/child welfare-involved children (Bernard, Dozier, Bick, & Gordon, 2015; Bernard, Hostinar, & Dozier, 2015; Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008). In a randomized clinical trial of 101 infants and toddlers judged by Child Protective Services to be at-risk for neglect, those children who received ABC showed a more typical post-treatment diurnal cortisol response, including higher waking cortisol levels and steeper diurnal rise, compared to children who received a control intervention (Bernard, Dozier, Bick, & Gordon, 2015). Further, ABC's effects on normalization diurnal cortisol regulation persisted for three years following treatment (Bernard, Hostinar, & Dozier, 2015). Similarly, Fisher & Stoolmiller (2008) reported findings from a randomized controlled trial of Multidimensional Treatment Foster Care for Preschoolers (MTFC-P) where the

intervention resulted in reduced caregiver stress and normalized diurnal cortisol response among preschoolers, relative to a comparison group.

Although this literature is more nascent, there is some evidence that psychosocial interventions may also be able to reduce chronic inflammation. Current evidence for this is perhaps strongest in the depression treatment literature. Cognitive behavior therapy (CBT), long known to be effective in reducing depression symptoms, has also shown potential to lower inflammatory markers in adults (for a review see Lopresti, 2017). Randomized controlled trials comparing CBT to control groups (e.g., Zabihiyeganeh et al., 2019), or to alternate depression treatments (e.g., Moreira et al., 2015) have shown significant post-treatment reductions in serum levels of pro-inflammatory cytokines, such as IL-6 and TNF-α and acute phase proteins (i.e., CRP), although some studies in this area have found no effect on inflammatory markers (Claesson et al., 2006) or even post-CBT increases in participant inflammation (Berk et al., 2015; Keri, Szabo, & Kelemen, 2014). Mind-body therapies (MBTs), such as Tai Chi, Qigong, yoga, and meditation, have also shown promise in lowering inflammation (Bower & Irwin, 2016). In their 2016 qualitative review, Bower and Irwin discuss positive treatment effects in adults seeking treatment for sleep disturbance or stress related to medical conditions, demonstrated by post-treatment reductions in circulating levels of CRP, cellular markers of inflammation, and inflammation-related gene expression, although null results have also been documented (e.g., Bower et al., 2014; Irwin & Olmstead, 2012). Some trials investigating inflammation among older adults with sleep disturbance have compared mind-body therapies to a form of CBT adapted to treat insomnia, with similar reductions on inflammatory markers observed across these treatments (Irwin et al., 2014, 2015). Given

that CBT and MBTs can achieve comparable effects on inflammation, despite differences in treatment modality, potential mechanisms of these interventions may provide insight about how behavioral therapies can work to lower inflammation.

Mechanisms for the effect of CBT on inflammation have not yet been empirically examined. However, researchers have suggested (Lopresti, 2017) that CBT may work to reduce inflammation through increasing patients' use of a variety of positive coping strategies that reduce stress as well through lifestyle improvements linked to lower inflammation, including the use of relaxation strategies (Kang et al., 2011), engagement in daily pleasurable activities (Sin, Graham-Engeland, & Almeida, 2015), or improved sleep (Irwin, Olmstead, & Carroll, 2016). MBTs have been better studied in this regard and are thought to lower inflammation by reducing stress reactivity. Active ingredients of MBTs include focused attention, controlled breathing, and physical movement (Bower & Irwin, 2016). Thus, CBT and MBTs likely influence immune activity through stress reduction, although they may also lower inflammation through positive lifestyle changes such as increased physical activity or improved sleep. It therefore follows that an intervention targeting relational quality and reducing relational stress, such as PCIT, may also confer benefits on lowering inflammation. Moreover, whereas CBT and MBTs have been assessed primarily in samples of adults with depression or medical conditions (Bower & Irwin, 2016; Lopresti, 2017) it is unknown whether behavioral treatments can reduce inflammation among psychosocially disadvantaged parents, making this an important gap in existing literature.

I am aware of only one study to date that has examined effects of a parent-coaching program for vulnerable families on inflammation, albeit among youth, rather than parents. In 2014, Miller and colleagues studied the biobehavioral effects of a family intervention delivered to low-SES, African American mothers and their 11-year-old child in the rural South (Miller, Brody, Yu, & Chen, 2014). The sample included 272 families who were randomly assigned to receive the psychosocial intervention Strong African American Families (SAAF) or to the control condition. SAAF targets increasing parenting skills, strengthening family relationships, and building children's competencies (Brody et al., 2004) and was delivered across seven weekly sessions. At an 8-year follow-up assessment, when participating youth were 19-years-old, chronic inflammation was measured via circulating levels of six major pro-inflammatory cytokines (i.e., IL 1β, -6, -8, and -10, tumor necrosis factor α, IFN-γ) in peripheral blood samples. Those youth whose families received the SAAF intervention showed significantly less chronic, low-grade inflammation than controls, demonstrated by lower levels of cytokine production. Further, the effect of the intervention on lowering youths' chronic inflammation was shown to be partially mediated by mothers' improved parenting skills (Miller, Brody, Yu, & Chen, 2014). These results demonstrate that even a relatively brief family intervention may yield lasting benefits in reduced peripheral inflammation with profound health implications, and that improving parenting skills is one mechanism for this powerful effect. However, no study to date has examined whether such behavioral interventions may work to reduce chronic inflammation in parents, as well as their children.

Current Study

Given that behavioral interventions can lower inflammation in adults and parenting interventions can lower chronic inflammation in adolescent offspring, I reasoned that a parenting intervention may also exert a positive impact on lowering inflammation in participating parents. The current project addressed this gap in the literature by testing the following aims:

Aim 1): Investigate the effects of PCIT on a biomarker of chronic inflammation (CRP) among CW-involved parents. **H1.** Parents randomly assigned to receive PCIT will display lower levels of CRP at post-treatment, relative to family services-as-usual (SAU) controls. **Aim 2):** Test whether PCIT lowers parents' inflammatory markers through reductions in parenting stress. **H2.** PCIT-driven reductions in parenting stress will mediate the effect of PCIT on post-treatment reductions in parental CRP. **Aim 3):** Explore parent characteristics and family risk factors that may moderate the effect of PCIT on parent CRP, including parent age, parent sex, and exposure to early adversity. **H3.** Although this final aim is exploratory, evidence suggests that behavioral interventions may confer greater inflammatory reductions among those with higher inflammation (Morgan et al., 2014), at study entry. Therefore, I explored whether the impact of PCIT on CRP will be amplified among mothers versus fathers (Khera, Vega, Das, Ayers, McGuire, Grundy, & Lemos, 2009), older parents (Lowe, 2005), and among parents with greater exposure to childhood adversity (Miller et al., 2011).

CHAPTER II

METHOD

Participants

Data were drawn from a larger NIDA-funded, randomized clinical trial of PCIT versus family services as usual (SAU) in *n* = 204 child welfare-involved parents and their 3 to 8-year old children (R01DA036533). Participants in the current study are *n* = 199 parents from whom CRP was collected at baseline and post-treatment. Participants were recruited via direct correspondence with their child welfare or self-sufficiency case worker within a local Department of Human Services (DHS) office. In order to be eligible for the study, participating parents were primary caregivers to their participating child, living together at least 50% of the time and had current DHS involvement via child welfare or self-sufficiency services. A majority of parents (97%) were the child's biological parent; participating caregivers were also the child's grandparent (1%), adoptive parent (.5%) or other relative (1%). Parents were 12% male and 88% female and ranged in age from 18 to 50 years (*M* = 32.30, *SD* 6.43). Parents were 70.8% European American/White, 2.6% Hispanic/Latino/a, 2.1% African American/Black, 1.0% Pacific Islander, 1.5% Native American, 20.4% multi-ethnic, and 1.5% unknown. Regarding socioeconomic indicators, 49% of parents reported having completed at least high school/GED certificate, 44% reported receiving temporary assistance for needy families (TANF) aid, and 54.5% of parents reported being unemployed. A majority (81%) of parents reported being single. Participating children were 54.6% male and 45.4% female (*M*age = 4.77 years, *SD* = 1.39).

Procedure

As part of the larger parent study, biobehavioral assessments of parents and children were conducted at the University of Oregon in three waves: pre-treatment/baseline (Wave 1), mid-treatment (Wave 2) for PCIT condition families only, and post-treatment (Wave 3) for PCIT intervention and SAU control condition families. The post-treatment assessment occurred approximately 6 months following the baseline assessment for SAU families (M = 7.21 months, *SD* = 1.86, range = 5 to 18 months) and immediately following treatment completion for PCIT families (time from baseline assessment: *M* = 7.79 months, *SD* = 2.65, range = 3 to 16 months). At each assessment wave, families completed two laboratory visits lasting approximately 2-3 hours, during which a range of biobehavioral measures were collected, including behavioral observations of parent–child interactions, autonomic physiology (respiratory sinus arrhythmia and pre-ejection period), high density EEG, behavioral and survey measures of self-regulation, socio-demographics, and biomarkers of health such as blood pressure, height, weight, spirometry, and blood spots.

Measures for the current study were collected at Waves 1 (baseline) and 3 (post-treatment). During visit 1, parents were read and given a copy of the informed consent document and provided consent for themselves and their children to participate. The family's DHS caseworker provided study consent for children when they were not in parents' legal custody, though living together with parent. Parents' height (in cm) and weight (in kg) was collected using a digital scale and stadiometer to calculate body mass index (BMI). Parents' waist circumference was measured in cm while in a relaxed, standing posture by a research assistant using a plastic measuring tape over bare skin,

using the top of the iliac crest as a bony landmark. Next, parents completed a brief demographic interview in which they reported on their age, racial/ethnic identity, household income, and subjective ratings of SES relative to their own community and to the United States. They were also asked whether they have a list of common health problems (e.g., high blood pressure, diabetes, asthma) and if they had taken medications during the last 24 hours.

Families returned for a second laboratory visit approximately one week later. At this assessment visit, parents completed standardized questionnaires reporting on their parenting stress, current mental health symptoms, and exposure to childhood adversity. To accommodate variations in parent literacy, a trained research assistant read questionnaire items aloud and provided a card displaying item response options. The research assistant recorded participant responses on a laptop computer. Next, blood spots were obtained from parents. Trained research assistants wiped the fingertip with an alcohol pad and made a single finger prick using a sterile, disposable micro-lancet which is triggered, then discarded in a biohazard container. For each consenting parent, five full drops of whole blood were collected on sterile Whatman 903 ™ filter paper, after which a bandage was applied. The filter paper was dried at room temperature for 24 hours, then placed in individual plastic bags with desiccant and temporarily stored in a small, in-lab freezer. Samples were periodically transferred for long-term storage in a padlocked, -80^0C freezer located in a secure area of the University of Oregon Human Biology Research Laboratory.

At the conclusion of the second research visit in Wave 1, parents were given a sealed envelope with a letter explaining whether they had been randomly assigned to the SAU control condition or to receive PCIT. Families could take a brief break midway through each assessment visit and were provided with a snack. At the end of each laboratory visit, parents were paid ($100 for visit 1, $75 for visit 2), compensated for transportation, and their children received a prize. The study was approved by the University of Oregon Institutional Review Board and the State of Oregon Department of Human Services IRB.

Intervention. PCIT was delivered to randomized families in weekly sessions at the University of Oregon clinic. Prior to the standard PCIT package, we employed a motivational enhancement treatment (MET) component used with child welfare-involved families by Chaffin and colleagues (Chaffin et al., 2009), adapted for use in individual sessions. Specifically, in consultation with PCIT master trainer Beverly Funderburk, we developed a two-session, individual MET component derived from the original, six-session group format MET employed in Chaffin et al (2004)'s clinical trial. The intervention was delivered by 8 therapists, including 6 doctoral graduate students who had obtained a masters' degree, a licensed social worker, and licensed psychologist. All therapists were trained by master PCIT trainers and received weekly remote consultation and live in-session supervision with a master PCIT trainer at the University of Oklahoma. Therapists completed a fidelity checklist at the conclusion of each session and independent raters monitored fidelity to the treatment model by coding session videotapes. Therapists were required to maintain an 80% or greater level of fidelity.

Families were over-allocated to the intervention group at a rate of 1.5:1 to ensure a sufficient number of families accessed the intervention. Thus, 122 parents were randomly assigned to receive PCIT, whereas 84 were assigned to the control group (i.e., family services as usual in the community). Treatment group allocation was made using a computerized random number table and allocation was concealed to participants and experimenters using a sealed envelope. Of the parents assigned to the PCIT condition, 88 (72%) attended at least one treatment session; 65 (53%) completed the first phase of treatment, CDI, and 49 parents (40.9%) received a full course of CDI and PDI sessions, defined as completing at least three PDI sessions. Of those who engaged in treatment (i.e., attended at least one session), the average number of sessions received was 12 (range = 1 to 31).

Control condition (Family Services-as-Usual). Families randomized to the control condition were free to access the range of social services available in the community. This is an ethical, ecologically valid alternative to a strict control condition for vulnerable families. At the post-treatment assessment, parents were asked to report on their families' participation in social and behavioral support services (e.g., counseling, in-home therapy services, skills coaching, case management, etc.) during the prior 6 months.

Measures

C-Reactive Protein

Parents' dried blood spots (DBS) were assayed to ascertain levels of CRP using high-sensitivity enzyme-linked immunosorbent assays (ELISA). Blood spot measurement of CRP is used in epidemiological studies (e.g., McDade, Williams, & Snodgrass, 2007;

McDade et al., 2006; Williams & McDade, 2009) and is shown to provide accurate assessment of CRP, commensurate with levels obtained through venous blood serum collection. Collection time of DBS was variable; however, CRP does not show diurnal variation (Meier- Ewert et al., 2001). CRP remains stable in DBS for at least 5 days at room temperature or 14 days at 4 degrees C and are stable for years when stored at -80 degrees C. Following ELISA, serum equivalents were calculated using the following algorithm: serum (high-sensitivity CRP) = 1.38 * (blood spot CRP value) −.97 (McDade et al. 2006). Higher values indicate greater levels of systemic inflammation. Expected levels of blood plasma CRP in healthy samples range from 0.8mg/L to 3.0 mg/L, whereas levels greater than 10.0 mg/L indicate frank infection. Figures 1 shows distributions for raw parent CRP DBS concentrations at pre- and post-treatment. After excluding extremely high values, parents showed mean CRP levels of 2.39 mg/L at baseline (*SD* = 3.17) and 2.02 mg/L at post-treatment (*SD* = 2.39). Parent CRP levels were in a range typically associated with chronic, low-grade inflammation (i.e., between 3.0 mg/L and 10.0 mg/L) for 30% of parents at baseline and 18% of parents at post-treatment.

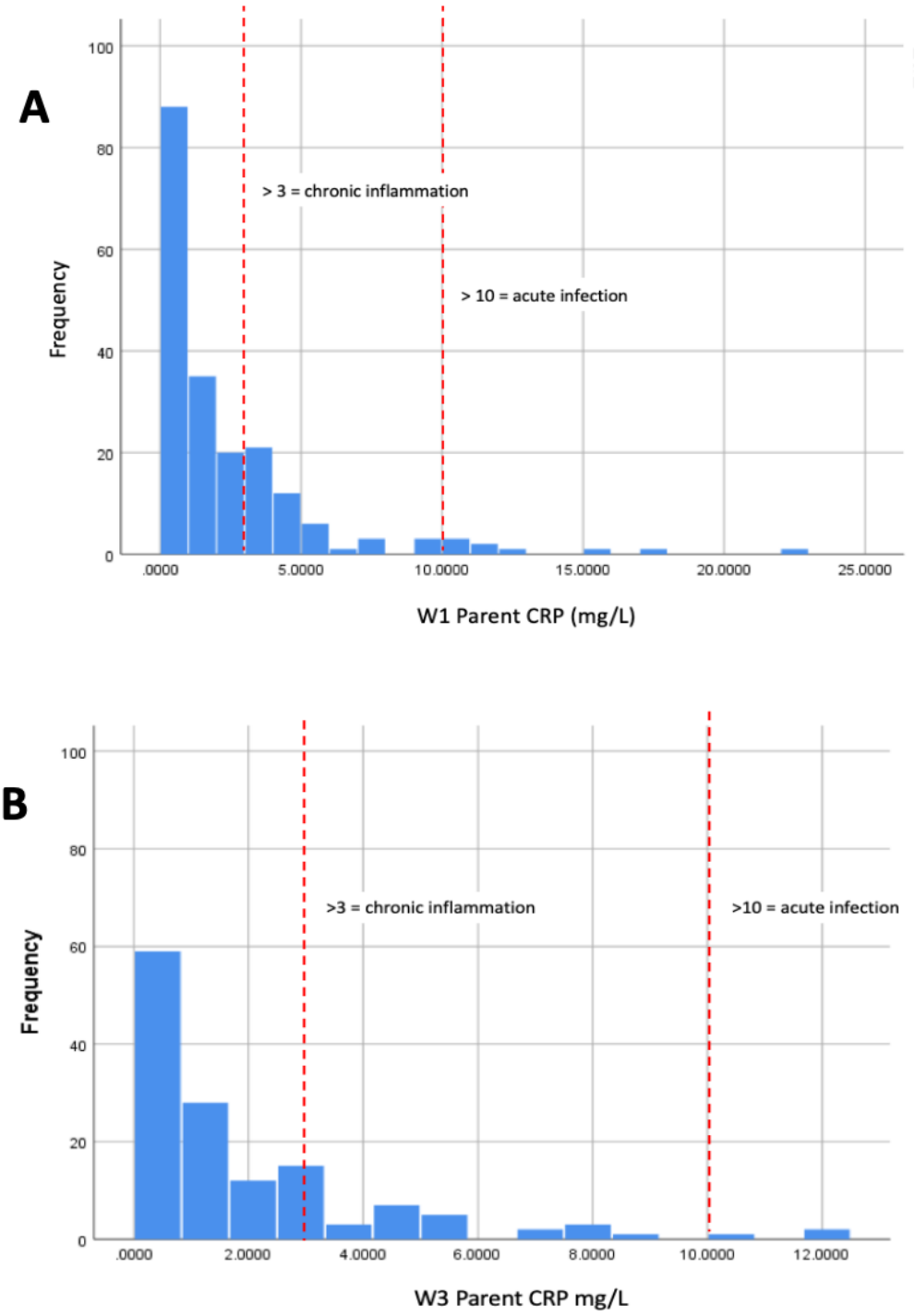


Figure 1. Distribution of raw CRP concentrations for parents at baseline (A) and post-treatment (B) among full sample.

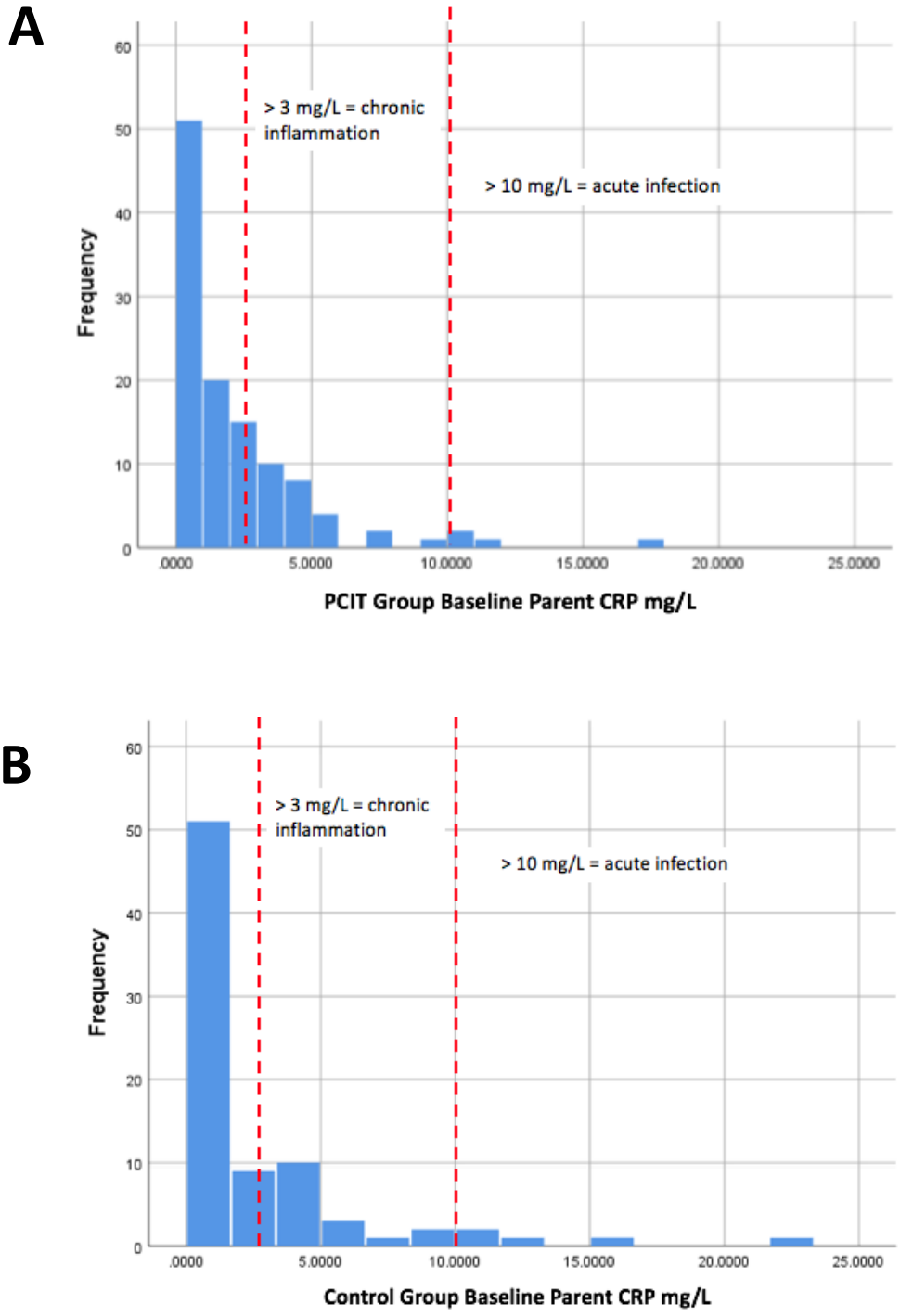


Figure 2. Distribution of baseline parent CRP among parents in the PCIT group (A) and SAU control group (B).

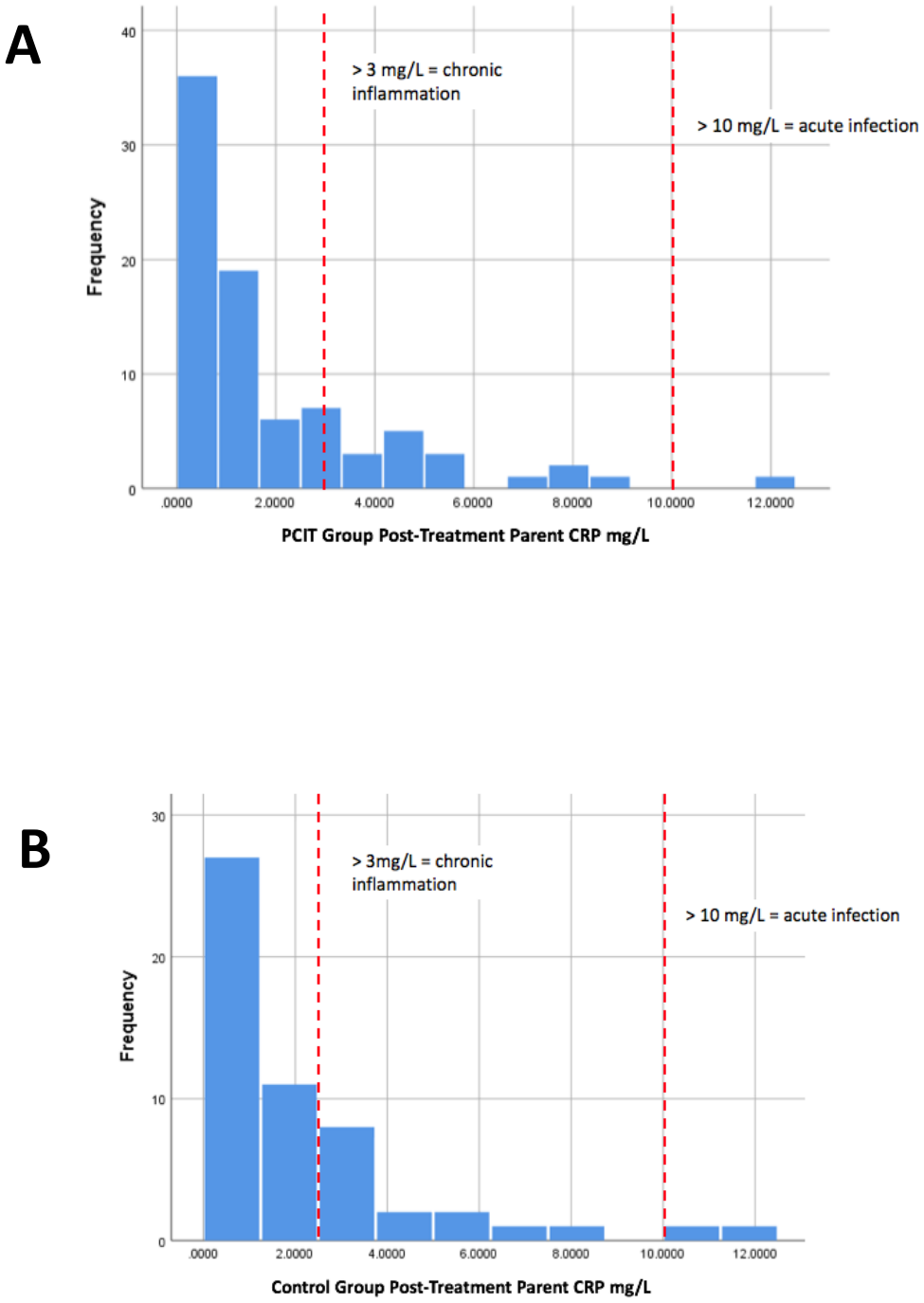


Figure 3. Distribution of post-treatment parent CRP among parents in the PCIT group (A) and SAU control group (B).

Sociodemographic Characteristics

Parents completed a semi-structured interview in which they reported on their age, race/ethnicity, socioeconomic indicators, and tobacco use. Number of cigarettes smoked per day was measured using a single self-report item: "In general, how many cigarettes do you smoke per day?" A variety of socioeconomic indicators were collected including: household income, parent employment status and hours worked, and parent educational attainment. The MacArthur Scale of Subjective Social Status (Adler, Epel, Castellazzo, & Ickovics, 2000) was used to collect parents' subjective SES ratings. Parents were presented with an image of a ten-rung ladder representing "where people stand," with higher rungs representing higher standing (i.e., "more education, more money, better jobs"), and asked to indicate a rung reflecting how "well-off" they considered themselves in relation to others, first within their community and then in relation to others across the entire United States. Possible scores on this measure range from 1 to 10, with higher scores reflecting higher perceived SES.

Parents reported a mean subjective SES ranking of 5.01 (*SD* = 1.84) relative to their community and mean subjective SES ranking of 3.97 (*SD* = 1.98) relative to the entire U.S. Parents reported smoking between 0 and 30 cigarettes per day (*M*=4.53, *SD*=6.31), with 52% indicating daily cigarette use. Body mass index (BMI) was calculated for parents at each wave of data collection using kg/m^2. Baseline BMI score fell in the obese range (i.e., ≥ 30) for 52.6% of parents (*M* = 29.99, *SD* = 7.41; see Figure 4).

Adverse Childhood Experiences

Parent exposure to adverse childhood experiences (ACEs) was assessed using the ACEs Questionnaire (Dube et al., 2013; Felitti et al., 1998). The ACEs Questionnaire has been adapted to retrospectively assess the various forms of adverse experiences identified as long-term health risks in the original Kaiser Permanente epidemiological study (Felitti et al., 1998). Parents were asked whether they experienced a standard list of 10 adverse experiences before the age of 18 and could respond yes or no. Items assessed experiences related to childhood abuse (i.e., psychosocial, physical, or sexual), neglect (i.e., emotional or physical), and exposure to household dysfunction (i.e., substance abuse, parental separation/divorce, mental illness, battered mother, criminal behavior). For example, one item reads: "While you were growing up, during your first 18 years of life, did you often feel that you didn't have enough to eat, had to wear dirty clothes, and had no one to protect you?" Scores are summed across items, with higher scores indicating greater exposure to adversity. Parents reported an average of $M = 5.18$ adverse childhood experiences (SD=2.71). The distribution of parent ACE scores is shown in Figure 4; 73.2% of parents reported experiencing 4 or more adverse childhood experiences, a cut-off found by Felitti et al. (1998) to be associated with increased disease risk.

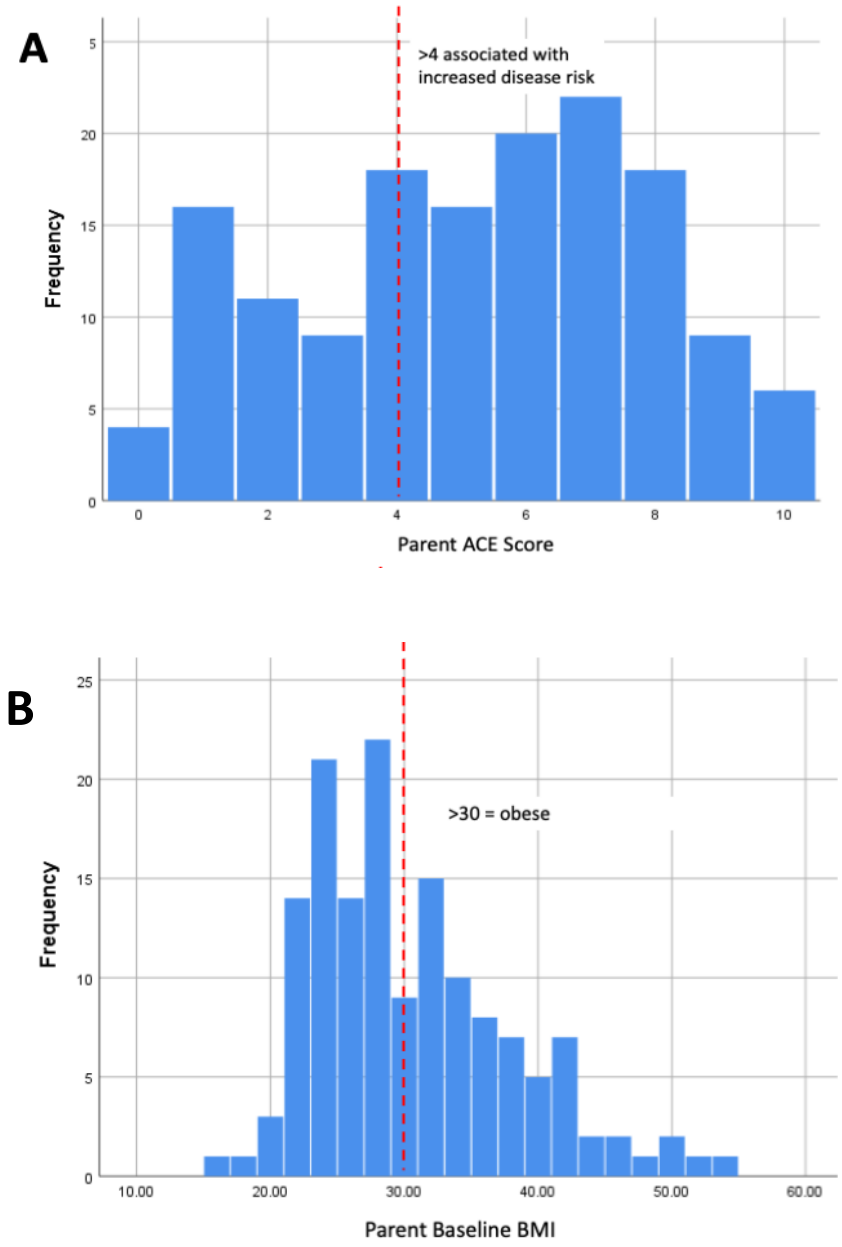


Figure 4. Distribution of parent adverse childhood experience (ACE) scores (A) and baseline body mass index (BMI; B)

Parenting Stress

Parents reported on parenting stress levels using the Parenting Stress Index, Fourth Edition Short Form (PSI-4SF; Abidin, 1995). The current study included two of the three original subscales, the Parent-Child Dysfunctional Interaction and Difficult

Child subscale, comprising 24 items. Parents responded to a series of statements describing their child using a 5-point Likert scale (1 – *strongly disagree* to 5- *strongly agree*). Items for the Parent-Child Dysfunctional Interaction subscale include statements such as "Sometimes I feel my child does not like me." Items for the Difficult Child subscale include items such as, "My child is very moody and easily upset." Responses are summed across items for each subscale, with higher scores indicating greater levels of parenting stress. The PSI has demonstrated strong psychometric properties in community samples (Haskett, Ahern, Ward, & Allaire, 2006; Whiteside-Mansell, Ayoub, McKelvey, Faldowksi, Hart, & Shears, 2007) as well as in low- income, samples of parents with preschool children (i.e., Head Start; Reitman, Currier, & Stickle, 2002). Internal consistency in the current sample was high for both the Parent-Child Dysfunctional Interactions subscale (Cronbach's $\alpha = .83$) and the Difficult Child subscale (Cronbach's $\alpha = .81$). At baseline, 37% of parents' Dysfunctional Parent-Child Interactions scores fell within the elevated range (i.e., above 80th percentile; $M = 22.85$, $SD = 6.64$) and 46.4% of Difficult Child scores fell within the elevated range ($M = 30.02$, $SD = 7.95$).

Depression Symptoms.

Parent depression symptoms were measured using the Depression subscale of the Brief Symptom Inventory (BSI; Derogatis & Spencer, 1982), an abbreviated psychological symptom checklist. The BSI has demonstrated strong psychometric properties (Boulet & Boss, 1991), including when administered to economically disadvantaged community samples (Prelow, Weaver, & Swenson, 2005). The depression subscale comprises 6-items of the 53-item inventory. Parents were asked to report how much they have been "bothered by" depressive symptoms such as "Feeling no interest in

things" during the past week on a 5-point Likert scale ranging from 0 (not at all) to 4 (very much). Internal consistency for the depression subscale in the current sample was high (Cronbach's $\alpha = .85$). For ease of interpretation, raw scores were converted to standardized T-scores using non-clinical population norms. Clinically elevated scores (i.e., T score > 63 were observed among 24% of parents at baseline ($M = 55.86$, $SD = 9.36$) and 20% of parents ($M = 54.02$, $SD = 10.03$) at post-treatment.

CHAPTER III

RESULTS

Data Preparation

Data Screening

I screened the raw data for outliers and examined the distributions for key study variables by visually inspecting histograms and conducting skewness tests. Figures 1 and shows histograms for raw parent CRP levels at baseline and post-treatment within the full sample. Figures 2 and 3 display parent CRP at baseline and post-treatment, partitioned by treatment condition. Consistent with previous studies (e.g., Nelson et al., 2017; Taylor et al., 2006), parent CRP data were highly positively skewed and so I performed a logarithmic transformation for both baseline and post-treatment CRP data, after following procedures for assessing and imputing missing data described below. Distributions for all other key study variables were found to be within acceptable limits (i.e., skewness statistic not greater than 1.0 or less than -1.0).

Exclusions

In keeping with previous studies (McDade et al., 2006; Snodgrass et al., 2007; Taylor et al., 2006), I excluded parents with extremely high CRP values indicative of acute infection (i.e., >10mg/L) as well as parents who reported having taken the following major anti-inflammatory medications in the 24 hours prior to each wave of DBS collection: antihistamines, NSAIDs, corticosteroids, cold and flu medications (Assanasen & Naclerio, 2002; El-Sharraway, El-Hakim, & Sameeh, 2006; Mainous & Pearson, 2003). Reported medication usage for the sample was high: 48.5% of parents at baseline and 69.6% of parents at post-treatment reported any medication use prior to DBS

collection. Further, parents did report using other medications with potential anti-inflammatory effects which I determined not to exclude (i.e., statins, anti-depressants, opioids, cannabis). Use of any medication with prior evidence of anti-inflammatory effects was reported by 35.8% of parents at baseline collection and 49.1 % of parents at post-treatment. Sensitivity analyses revealed that results across analyses were unchanged when I excluded parents who used any potential anti-inflammatory medication versus only those who used the major anti-inflammatory medications listed above.

For baseline CRP, 9 parents were excluded due to CRP levels >10 mg/L and 41 were excluded due to major anti-inflammatory medication usage. For post-treatment CRP, 3 parents were excluded due to levels > 10 mg/L and 29 parents were excluded based on recent, major anti-inflammatory medication usage. Given that different parents were excluded at each wave, this resulted in a complete analytic sample of $n = 85$.

Missing Data

Baseline missing data analysis. Prior to conducting analyses, I examined rates of missing data among study variables. At baseline, rates of missing data were minimal for key study variables: less than 2% missing for parent CRP, less than 1% missing for parenting stress (PSI scores), and complete data were obtained for parent depression symptoms (BSI scores) and parent ACEs scores. Missing data were observed in some baseline demographic variables: 21.48% of household income cases were missing due to an anomaly in data processing that led parents' reports of no income to be designated as missing, and 6.71% of parents' subjective SES ratings were missing. Although true missing baseline CRP data were nominal, I determined to consider excluded cases at baseline as missing CRP for purposes of missing data exploration and imputation,

because this approach allowed me to assess both waves of CRP data simultaneously, with optimal statistical power. Including the removed cases as discussed above, baseline CRP was missing for 26.12% of the sample. I explored the mechanism for missing data at baseline using Little's Missing Completely at Random (MCAR) test (Little, 1988) in RStudio v. 3.6. The chi-square result was not statistically significant, χ^2 (64) = 82.45, p = .06, thus failing to reject the null hypothesis that data were missing completely at random.

Post-treatment missing data analysis. At post-treatment, parent CRP data were missing for 58 cases (36% of the post-treatment dataset, after exclusions). Missing post-treatment CRP data were due to: parent drop-out from the study (38 cases), blood samples available but not yet assayed (16 cases), parent attended post-treatment assessment but DBS were not acquired (i.e., unsuccessful finger stick, assessors ran out of time; 3 cases), or because parent refused (1 case). Post-treatment observations were also missing for body mass index (27.10%), parent depression scores (25.90%) and parenting stress (30.72%) due to non-return to one (4 cases) or both post-treatment assessment visits (39 cases) or because assessors ran out of time to administer all measures (2 cases). For post-treatment data, Little's MCAR test (Little, 1998) results also failed to reject the hypothesis that data were missing completely at random, χ^2 (13) = 48.04 p = .65.

Based on these results, I next conducted multiple imputation for the above pre-treatment and post-treatment variables with missing data using the fully conditional specification method in SPSS. CRP observations excluded due to extremely high concentration levels or to medication use were considered missing for baseline, but

removed for post-treatment, in order to produce a single, imputed dataset that allowed for pre- and post-treatment CRP to be analyzed together. To address potential bias that may have been introduced through multiple imputation, I compare main analyses below using both the imputed data and complete case data.

I also conducted attrition analyses to explore whether patterns of missing data at post-treatment were related to baseline variables by creating dummy codes (i.e., 1=missing, 0=not missing) for key study variables and performing a series of chi-square tests (for categorical variables) and independent samples *t* tests (for continuous variables) in SPSS v. 26 to assess whether rates of attrition were related to any other study variables. Patterns of missing data across key study variables were not found to be related to parent and family characteristics (i.e., parent age, parent sex, parent race/ethnicity, parent educational attainment, child age, household income) nor to baseline levels of study variables.

Assessing Equivalence of Treatment and Control Groups Due to Randomization

To assess whether the PCIT and SAU control groups created through randomization were equivalent at study entry, I performed a series of independent samples t-tests on baseline key study variables and select demographic characteristics, using complete cases. Results indicated baseline, raw CRP levels were not statistically significantly different between parents randomized to the treatment (M = 1.76, SD=1.94) and control groups [M = 1.69, SD=1.91; t (163) = -.27, p = .79]. Baseline parenting stress scores were also not significantly different between groups, for PSI Dysfunctional Parent-Child Interactions scores [PCIT M = 22.78, SD = 6.55; Control M = 22.82, SD = 6.30; t

(163) = .03, p = .98] or PSI Difficult Child scores [PCIT M = 30.03, SD = 8.17; Control Group M = 29.02, SD = 7.60; t (163) = -.77 p = .45]. Further, there were no statistically significant differences between treatment groups in regards to parent age [PCIT *Mage* = 32.10 years, SD = 5.96; Control *Mage* = 31.56 years, SD = 6.00; t (163) = .54, p = .59], ACE score [PCIT M = 5.18 , SD = 2.81, Control M = 5.25, SD = 2.52; t (163) = .14, p = .29], BSI depression score [PCIT M = 56.00 , SD = 9.42, Control M = 55.64 , SD = 9.33; t (163) = -.27, p = .79], or subjective SES ratings within a parent's community [PCIT M = 5.04, SD = 1.66; Control M = 4.95, SD = 2.02; t (151) = -.26, p = .80] or the United States [PCIT M = 3.95 , SD = 2.02; Control M = 3.90 , SD = 2.00; t (151) = .13, p = .90]. Using a cross-tabulation analysis, I also confirmed that rates of male and female parents did not significantly differ across the PCIT (87.5% female, 12.5% male) and control (83.6% female, 16.4% male) groups, χ^2 (1) = .45 p = .50.

Preliminary Results

Descriptive statistics using complete cases (without imputation) for all study variables across the full sample, treatment, and control group are shown in Table 1. Zero-order correlations among all study variables at baseline are shown in Table 2.1 using multiple imputation data and in Table 2.2 using complete cases. The same pattern of associations was observed using complete and original data. Specific statistical results reported below are drawn from the imputed correlation matrix. Parents' baseline BSI Depression scale scores were positively related to parenting stress measured via the PSI Dysfunctional Parent-Child Interactions subscale [r (168) = .31, p <.001] and the PSI Difficult Child subscale [r (168) = .26, p < .001], such that elevated depression symptoms were associated with greater parenting stress scores. Lower subjective SES relative to

one's own community was significantly associated with higher parenting stress per PSI Dysfunctional Parent-Child Interaction scores [r (168) = -.18 p = .03] and PSI Difficult Child scores [r (168) = -.21, p = .003]. Parent ACE score was positively associated with depression symptoms [r (168) = .19, p = .01] and cigarette use [r (168) = .24, p = .003], indicating parents exposed to greater childhood adversity were more likely experience depression symptoms and smoked more cigarettes per day. Parent age was positively associated with waist circumference [r (168) = .25, p = .001] and BMI [r (168) = .18, p = .02], as well as educational attainment [r (168) = .33, p <.001]. Educational attainment was also associated with lower cigarette use [r (168) = -.20, p = .04].

Table 1

Baseline Descriptive Statistics for All Study Variables within Full Sample and by Treatment

	Full Sample		**Treatment Group**		**Control Group**	
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*
Parent CRP mg/L (raw)	2.29	3.20	2.14	2.83	1.69	1.91
PSI: Dysfunctional	22.93	6.42	23.04	6.42	22.82	6.30
PSI: Difficult Child	30.02	7.41	30.34	7.84	29.02	7.60
Depression T-Score	55.86	9.34	56.00	9.42	55.64	9.33
Waist (cm)	102.37	22.56	100.14	18.0	101.62	19.52
BMI	30.40	7.38	30.56	7.64	31.25	10.28
Cigarettes/day	4.53	6.31	3.90	5.93	5.37	7.00
Subjective SES:	5.01	1.87	5.93	2.04	6.05	1.66
Subjective SES: U.S.	3.97	1.95	7.03	1.91	7.05	2.02

Table 2.1

Zero-Order Correlations Among All Baseline Study Variables in Full Sample – Imputed Data

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Log CRP	___											
2. PSI: Dysfunctional Interactions	-.06	____										
3. PSI: Difficult Child	-.02	**.67****	____									
4. Parent Depression score	-.03	**.31****	**.26****	___								
5. Parent ACE score	-.11	.02	.04	**.19***	___							
6. Parent BMI	**.53****	.06	.08	.06	-.04	___						
7. Parent Waist Circumference	**.47****	.05	.12	.06	-.06	**.83****	___					
8. Cigarettes/day	-.05	.06	.15	**.20***	**.24****	.11	.09	___				
9. Parent Age	.04	-.08	-.08	.05	.10	**.18***	**.25****	.06	___			
10. Parent Education	.05	-.10	.00	-.04	-.05	.13	.12	**-.20***	**.33****	___		
11. Subjective SES: Community	-.05	**-.18***	**-.21****	-.15	-.09	-.03	-.08	-.03	.05	.05	___	
12. Subjective SES: U.S.	-.05	-.08	-.14	-.14	-.03	-.02	-.09	-.05	.03	**.30****	**.41****	___

Note. * = $p < .05$, ** = $p < .01$.

Table 2.2

Zero-Order Correlations Among All Baseline Study Variables in In Full Sample – Complete Cases

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Log CRP	___											
2. PSI: Dysfunctional	-.01	____										
3. PSI: Difficult Child	.02	**.68****	____									
4. Parent Depression score	-.00	**.31****	**.26****	____								
5. Parent ACE score	-.19	.07	.05	**.24***	___							
6. Parent BMI	**.53****	.09	.05	.09	-.02	___						
7. Parent Waist Circumference	**.54****	.09	.06	.05	-.09	**.88****	___					
8. Cigarettes/day	-.15	.02	.10	**.20***	**.24****	.05	.07	___				
9. Parent Age	-.01	-.04	-.07	.14	.11	.16	**.18***	.16	___			
10. Parent Education	.05	-.08	.00	-.06	-.05	.12	.10	**-.21***	**.25***	___		
11. Subjective SES: Community	-.12	**-.23***	**-.26****	**-.23****	-.09	-.12	-.15	-.04	.06	.07	___	
12. Subjective SES: U.S.	-.02	-.15	**-.23***	**-.20****	-.08	-.14	-.18	-.12	.04	**.30****	**.44****	___

Note. * = $p < .05$, ** = $p < .01$.

Table 2.3

Zero-Order Correlations Among All Baseline Study Variables for Treatment Group – Imputed Data

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Log CRP	___											
2. PSI: Dysfunctional	-.20	____										
3. PSI: Difficult Child	-.14	**.68****	____									
4. Parent Depression score	-.20	**.29****	**.22***	____								
5. Parent ACE score	-.17	-.02	-.07	.14	___							
6. Parent BMI	**.49****	-.05	-.05	.02	-.12	___						
7. Parent Waist Circumference	**.42****	-.10	-.05	.01	-.16	**.77****	___					
8. Cigarettes/day	-.06	-.05	.03	.07	**.29****	.13	.05	___				
9. Parent Age	.20	-.08	-.03	.06	.05	**.29****	**.41****	.03	___			
10. Parent Education	-.06	-.22	-.19	-.12	.02	.03	.06	-.15	.17	___		
11. Subjective SES: Community	-.03	-.11	**-.24***	**-.25***	-.13	-.04	-.12	-.03	-.04	.24	___	
12. Subjective SES: U.S.	.08	-.09	.12	-.19	-.08	-.07	-.16	-.05	.03	**.32***	**.35****	___

Note. * = $p < .05$, ** = $p < .01$.

Table 2.4

Zero-Order Correlations Among All Baseline Study Variables for Control Group – Imputed Data

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Log CRP	___											
2. PSI: Dysfunctional	-.06	____										
3. PSI: Difficult Child	-.02	**.67****	____									
4. Parent Depression score	-.03	**.31****	**.27****	____								
5. Parent ACE score	-.11	.02	.04	**.19***	___							
6. Parent BMI	**.53****	.06	.08	.06	-.04	___						
7. Parent Waist Circumference	**.47****	.05	.12	.06	-.06	**.83****	___					
8. Cigarettes/day	-.05	.06	.15	**.20***	**.24***	.11	.09	___				
9. Parent Age	.04	-.08	-.08	.05	.10	**.18***	**.25***	.06	___			
10. Parent Education	.14	-.04	.10	.01	-.09	**.22***	.16	**-.27***	**.43***	___		
11. Subjective SES: Community	-.05	**-.19***	**-.21****	-.15	-.09	.03	-.08	-.15	-.05	.03	___	
12. Subjective SES: U.S.	-.05	-.08	-.14	-.14	-.03	.03	-.09	-.05	.03	**.29****	**.41****	___

Note. * = $p < .05$, ** = $p < .01$.

I further examined zero-order correlations among pre-treatment study variables separately by treatment group, using imputed data. Associations among variables were observed in the treatment (Table 2.3) and SAU control conditions (Table 2.4) were aligned with those found in the full sample, with a few exceptions. There was a unique, negative association between BSI depression symptoms and subjective community SES, [r (101) = -.25, p = .02] in the treatment group, such that lower perceived community standing related to greater depression symptoms. Within the treatment condition, the correlation between ACE score and depression symptoms found in the full sample was not observed, nor were associations between educational attainment, age, and cigarette use.

Table 3.1 shows zero-order correlations among post-treatment variables (i.e., CRP, parenting stress, BMI, and depression scores) and baseline parent characteristics, using imputed data. Similar to baseline, post-treatment BSI depression scores were positively correlated with parenting stress indices [Dysfunctional Parent-Child Interactions: r (168) = .26, p = .02; Difficult Child: r (168) = .44, p <.001]. Scores from the post-treatment PSI Difficult Child subscale were also negatively associated with subjective SES ratings relative to the entire U.S. [r (168) = -.17, p = .04]. Post-treatment BSI depression scores were positively related to ACE scores [r (168) = .19, p = .03]. Findings varied only slightly when using complete cases (see Table 3.2). Specifically, the associations between parent age and post-treatment BMI and between parent ACE score and depression symptoms were not replicated in the observed data. I again further explored zero-order correlations using imputed data at post-treatment separately for the PCIT treatment (Table 3.3) and the SAU control group (Table 3.4). Several of the zero-order correlations reported for the full sample were not present when I partitioned parents by treatment condition. PSI Difficult Child scores were not associated with subjective SES in either the

treatment (see Table 3.3) or control group (see Table 3.4) when considered separately, and there was no association between post-treatment parent depression symptoms and ACE scores within the treatment group (see Table 3.3).

Characterizing Parent CRP in Current Sample

Baseline parent CRP. To inform selection of covariates and to explore patterns of parent CRP in this socially disadvantaged sample, I first conducted descriptive analyses to visualize the range of CRP scores at baseline as they relate to parent sociodemographic characteristics. Zero-order correlations between baseline CRP and all other study variables are shown using imputed data in Table 2.1 and using complete cases in Table 2.2. Results did not differ across these analyses and specific statistical results described below are from the imputed data, unless otherwise noted. For ease of interpretation, all figures were produced using the observed data. Before excluding parents with extremely high CRP levels indicative of acute rather than chronic inflammation, CRP values in the current sample ranged from .00 mg/L to 22.00 mg/L, with 9 parents showing baseline CRP levels indicative of acute infection. Figures 1 shows distributions for parents' raw, DBS CRP concentrations at pre- and post-treatment. After excluding extremely high cases, the average parent CRP level reflected low levels of chronic inflammation (M = 2.39, SD = 3.17). However, considerable variability was observed and CRP levels associated with chronic, low-grade inflammation (i.e., between 3.0 mg/L and 9.9 mg/L) were present for 30 parents (20% of sample).

Table 3.1

Zero-Order Correlations Among Post-Treatment Parent CRP and Study Variables for Full Sample - Imputed Data

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Log Post-Tx CRP	___											
2. Post-Tx PSI: Dysfunctional	-.02	___										
3. Post-Tx PSI: Difficult	-.03	**.75****	___									
4. Post-Tx Depression	-.06	**.26****	**.44****	___								
5. Post-Tx BMI	**.30****	-.05	-.04	-.00	___							
6. Waist (cm)	**.46****	.09	.09	.01	**.42****	___						
7. Parent ACE score	-.11	-.02	.04	**.19***	-.07	-.06	___					
8. Cigarettes/Day	-.06	.03	.14	.13	.14	.09	**.24****	___				
9. Parent Age	**.22***	.03	.01	.13	.07	**.25****	.10	.06	___			
10. Parent Education	.11	-.11	-.10	-.07	.14	.14	.00	-.15	**.31***	___		
11. Subjective SES:	.13	-.01	-.17	-.01	-.02	-.09	-.09	-.03	.06	.05	___	
12. Subjective SES: U.S.	-.11	-.10	**-.17***	.01	.00	-.09	-.03	-.05	.03	**.29****	**.42****	___

Note. * = $p < .05$, ** = $p < .01$.

Table 3.2
Zero-Order Correlations Among Post-Treatment Parent CRP and Study Variables for Full Sample – Complete Cases

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Log Post CRP	___										
2. Post PSI: Dysfunctional	.15	___									
3. Post PSI: Difficult Child	.19	**.73***	___								
4. Post Depression Score	-.07	**.36***	**.51****	___							
5. Post BMI	**.56****	.00	-.01	-.07	___						
6. Waist (cm)	**.56****	.23	.20	.01	**.31***	___					
7. Parent ACE score	-.15	.12	.18	.21	-.12	-.06	_				
8. Cigarettes/Day	-.01	.02	.12	.07	.09	.00	.	___			
9. Parent Age	.18	.19	.25	.19	.07	.18	.	**.21***	___		
10. Parent Education	.09	-.07	-.15	-.08	.09	.14	.	-	**.25***	___	
11. Subjective SES:	.21	-.05	**-.27***	-.12	-.03	-.14	-	-.03	.06	.01	___
12. Subjective SES: U.S.	-.15	-.23	**-.41***	-.13	-.07	-.18	-	-.17	.01	**.32***	**.43****
13. Household Income	.14	-.09	**-.28***	-.22	.09	.04	-	-.14	.07	**.30***	**.19***

Table 3.3

Zero-Order Correlations Among Post-Treatment Parent CRP and Study Variables for Full Sample – Complete Cases

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Log Post CRP	___	-.10									
2. Post PSI: Dysfunctional	-.01	___									
3. Post PSI: Difficult Child	-.08	**.74****	___								
4. Post Depression Score	-.08	**.**19	**.39****	___							
5. Post BMI	**.50****	-.06	-.11	-.05	___						
6. Waist (cm)	**.45****	.01	-.03	-.09	**.55****	___					
7. Parent ACE score	-.16	-.04	.09	.05	-.15	-.16	___				
8. Cigarettes/Day	-.05	-.08	.08	-.03	.18	.05	**.30***	___			
9. Parent Age	**.25***	-.00	-.01	.01	.15	**.41***	.05	.03	___		
10. Parent Education	.10	-.04	.10	-.00	.20	.14	.01	-.11	**.38***	___	
11. Subjective SES:	.17	-.00	-.17	-.08	-.00	-.12	-.13	-.03	-.04	-.07	___
12. Subjective SES: U.S.	-.13	-.12	-.15	-.00	-.00	-.16	-.08	-.06	.04	**.29***	**.35****

Note. * = $p < .05$, ** = $p < .01$.

Table 3.4

Zero-Order Correlations Among Post-Treatment Parent CRP and Study Variables for Control Group

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Log Post CRP	___										
2. Post PSI: Dysfunctional Parent-	-.02	___									
3. Post PSI: Difficult Child	.06	**.77****	___								
4. Post Depression Score	-.02	**.36****	**.42****	___							
5. Post BMI	.15	-.03	.01	.01	___						
6. Waist (cm)	**.46***	.23	**.28***	.18	**.33***	___					
7. Parent ACE score	.00	.01	-.06	**.41****	.01	.10	___				
8. Cigarettes/Day	-.08	.14	.20	.20	.09	.11	.16	___			
9. Parent Age	.16	.08	-.14	.19	-.00	.01	.20	.09	___		
10. Parent Education	.15	-.05	**-.29***	-.17	.20	.15	-.02	-.22	.19	___	
11. Subjective SES: Community	.06	-.03	-.18	-.09	-.04	-.03	.01	-.03	.23	.22	___
12. Subjective SES: U.S.	-.07	-.07	-.20	.02	.01	.01	.06	-.05	.03	**.29***	**.56****

Note. * = $p < .05$, ** = $p < .01$.

Baseline CRP levels based on the imputed data were not statistically significantly different between mothers (n = 85, M = 1.80, SD = 2.00) and fathers (M = 1.33, SD = 1.66; see Figure 5), t (166) = -1.04, p = .30. Figure 6 shows mean CRP levels partitioned by parent race/ethnicity. Note that the cell sizes are small for non-White parents participating in the study (i.e., n = 101 White, 4 Hispanic/Latino/a, 3 Black/African American, 2 Pacific Islander 3 Native American, 36 Multi-ethnic or unknown). As depicted in Figure 7, pre-treatment CRP levels were not significantly associated with parent age, based on complete data [r (168) = .04, p = .59]. There was a statistically significant, positive association between a parent's baseline CRP and their waist circumference [r (168) = .47, p <.001] and BMI [r (168) = .53, p <.001], suggesting obesity was associated with higher CRP concentrations. CRP levels were not correlated with parent cigarette use [r (168) = -.05 p = .61], annual household income [r (168) = .02, p = .81], or, as shown in Figure 8, subjective ratings of their SES either within their own community [r (168) = -.05, p = .75] or relative to the entire U.S. [r (168) = -.05, p = .84].

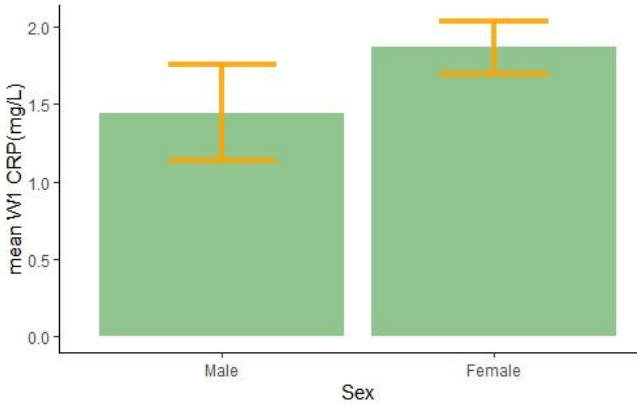


Figure 5. Mean baseline parent CRP levels by parent

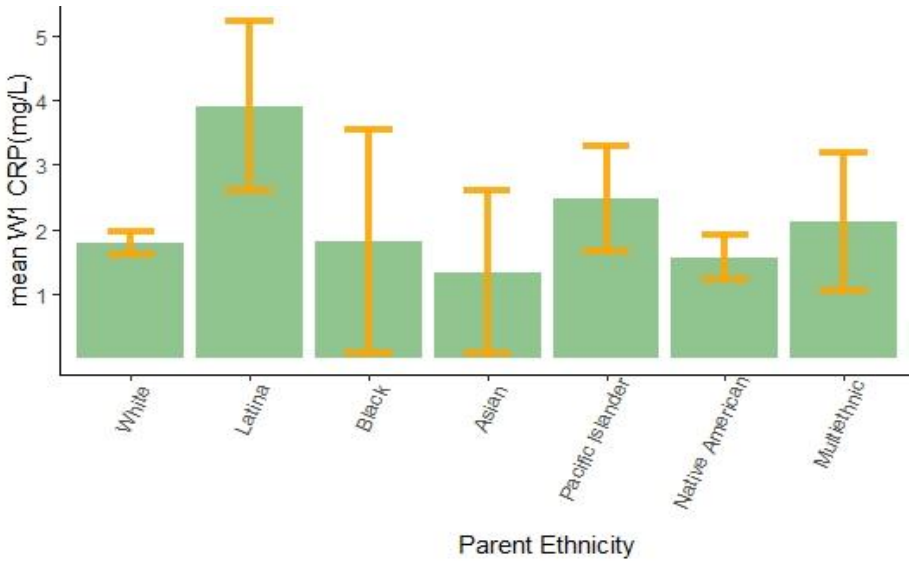


Figure 6. Mean baseline parent CRP levels by parent race/ethnicity.

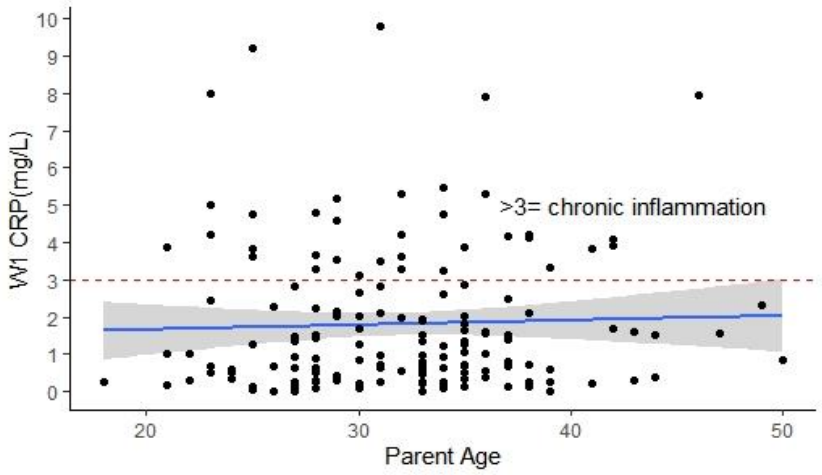


Figure 7. Associations between baseline parent CRP and parent age.

There was also a lack of associations in this sample between baseline measures of parent CRP and psychosocial risk indicators. Figure 11 shows mean CRP levels by parent ACE score; baseline CRP was not related to adverse childhood experiences [r (168) = -

.11, p = .25] or to their BSI depression symptoms [see Figure 10; r (168) = -.03, p = .76]. Further, baseline CRP concentrations were not associated with baseline parenting stress across either the PSI Dysfunctional Parent-Child Interactions subscale [r (168) = -.02, p = .63] or PSI Difficult Child subscale [r (168) = -.03, p = .61]. Zero-order correlations between baseline CRP and all other baseline study variables within the full sample are summarized in Table 2.1. I further explored whether associations between baseline CRP and psychosocial risk variables may vary across treatment group (shown in Table 2.3) and control group (see Table 2.4); however, the pattern of associations between CRP and other study variables did not differ within each treatment group, compared to full sample estimates.

Post-treatment parent CRP. At post-treatment, 3 parents displayed extremely high CRP indicative of frank infection (i.e., >10mg/L) and 20 parents (19.8% of sample) displayed CRP within a range indicating chronic, low-grade inflammation (i.e., between 3.0 mg/L and 9.9 mg/L; see Figure 1). After excluding those with extremely high levels, the mean post-treatment CRP level was 2.02 mg/L (SD = 2.39). Thus, a majority of parents showed post-treatment CRP concentrations within a healthy range, although elevations were, again, observed in a subset of parents.

Results drawn from imputed data are shown in Table 3.1 and results using complete case analysis are shown in Table 3.2; findings did not differ across datasets and correlation results reported below are from the imputed data results. Mean post-treatment CRP levels for mothers (M = 1.93, SD = 1.80) and fathers (M = 1.27, SD = 1.01; see Figure 12), were not statistically significant [t (166) = -1.64, p = .18]. Associations

between post-treatment CRP and parent age are displayed in Figure 13; based on the imputed data, post-treatment CRP levels were positively associated with parent age [r (168) = .22, p = .02]. Figure 14 shows mean levels of post-treatment CRP by parent race/ethnicity that appear somewhat variable across racial/ethnic groups. A one-way ANOVA using the observed data suggested post-treatment CRP did not vary significantly by parent ethnicity [F (6, 162) = .82, p = 56], although this analysis was underpowered due to small cell sizes.

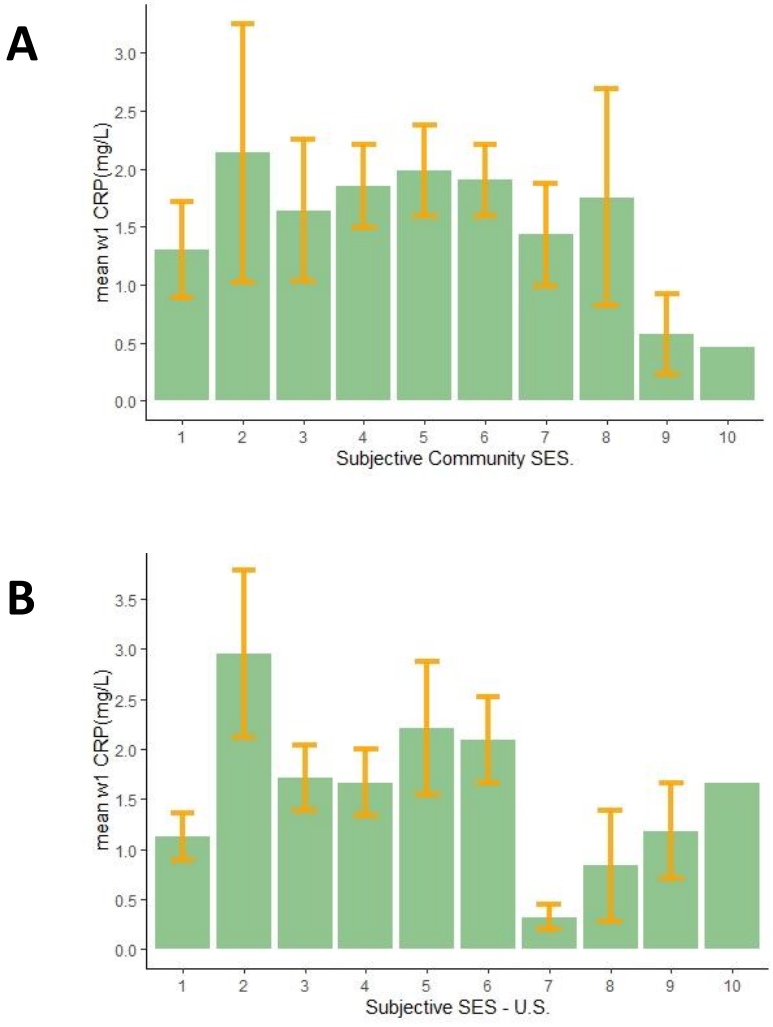


Figure 8. Mean baseline parent CRP levels by subjective ratings of SES in their community (A) and the United States (B).

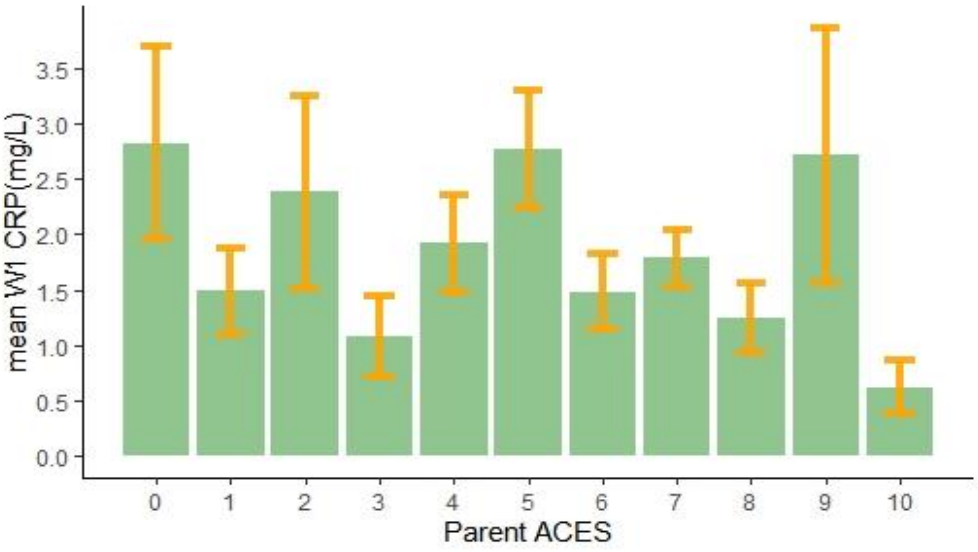


Figure 9. Mean baseline parent CRP levels by parent adverse childhood experience (ACE) score.

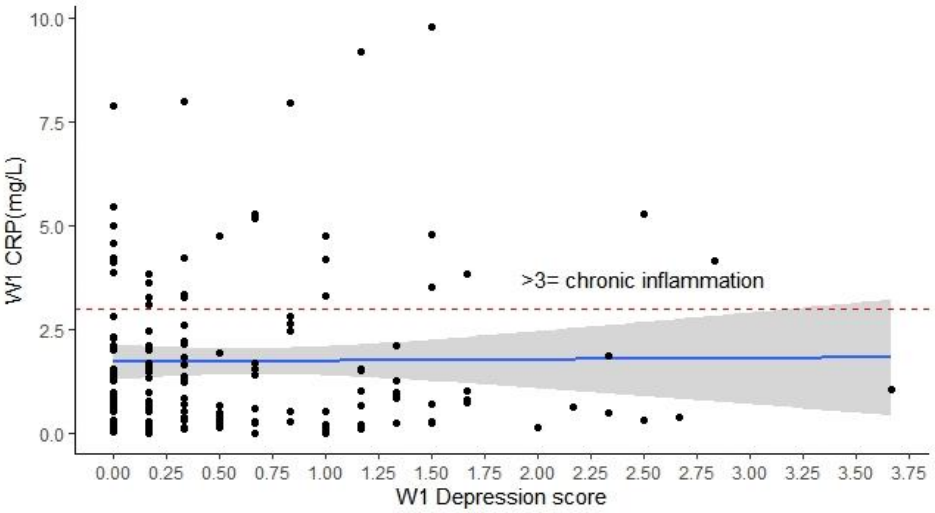


Figure 10. Associations between baseline CRP concentration and parent depression symptoms.

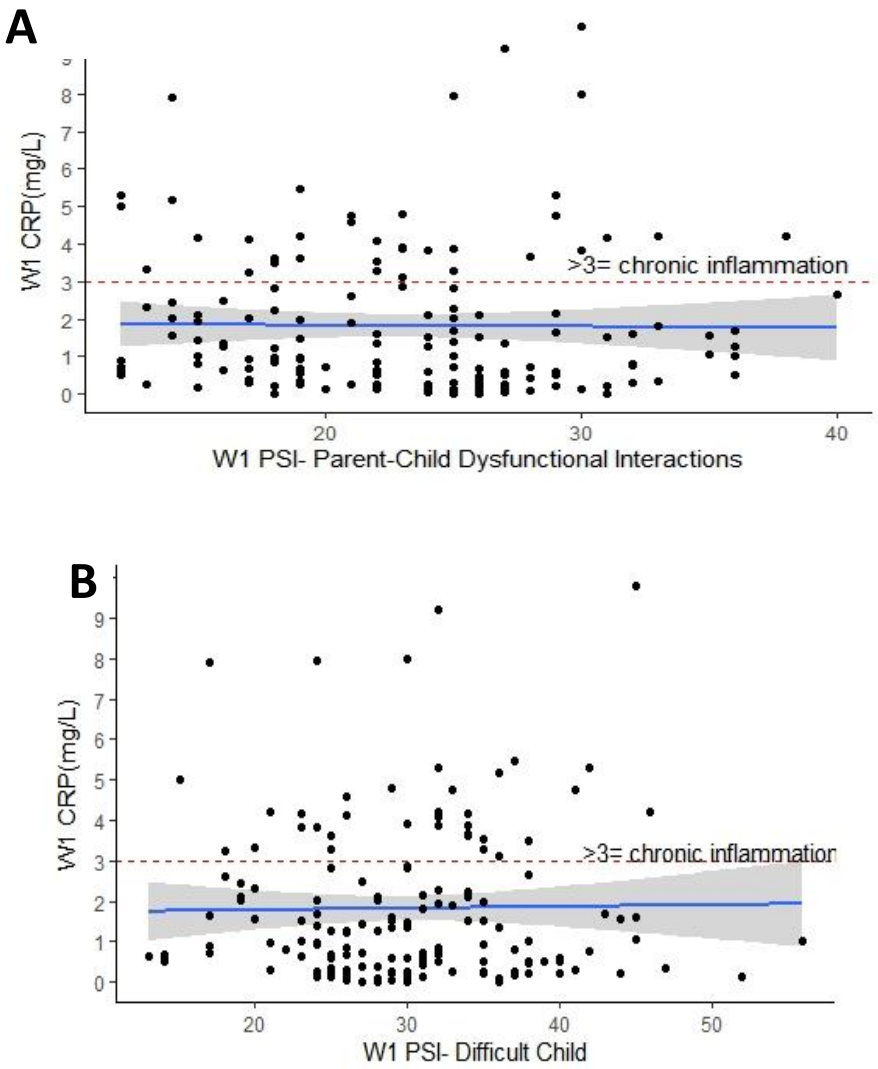


Figure 11. Associations between baseline parent CRP levels and parenting stress indices.

Results drawn from imputed data are shown in Table 3.1 and results using complete case analysis are shown in Table 3.2; findings did not differ across datasets and correlation results reported below are from the imputed data results. Mean post-treatment CRP levels for mothers (M = 1.93, SD = 1.80) and fathers (M = 1.27, SD = 1.01; see Figure 12), were not statistically significant [t (166) = -1.64, p = .18]. Associations

between post-treatment CRP and parent age are displayed in Figure 13; based on the imputed data, post-treatment CRP levels were positively associated with parent age [r (168) = .22, p = .02]. Figure 14 shows mean levels of post-treatment CRP by parent race/ethnicity that appear somewhat variable across racial/ethnic groups. A one-way ANOVA using the observed data suggested post-treatment CRP did not vary significantly by parent ethnicity [F (6, 162) = .82, p = 56], although this analysis was underpowered due to small cell sizes.

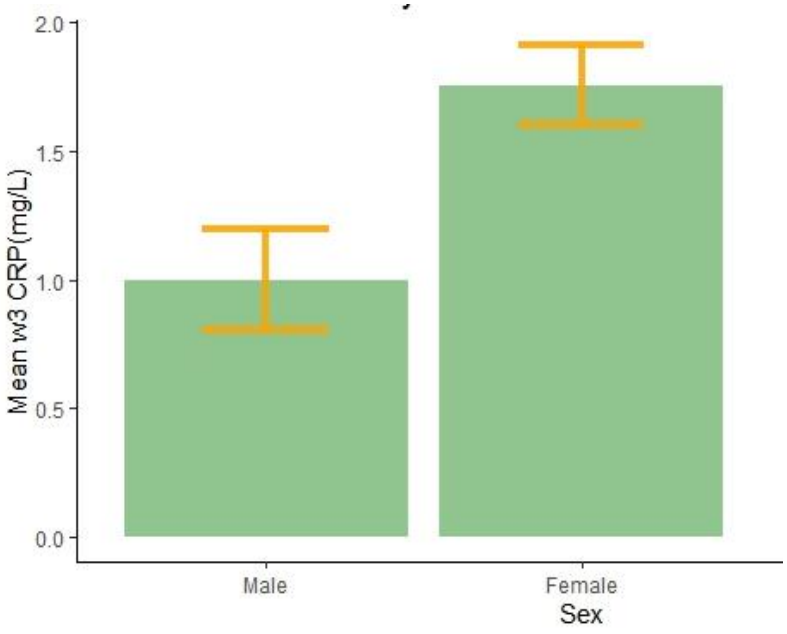


Figure 12. Mean post-treatment CRP levels by parent sex.

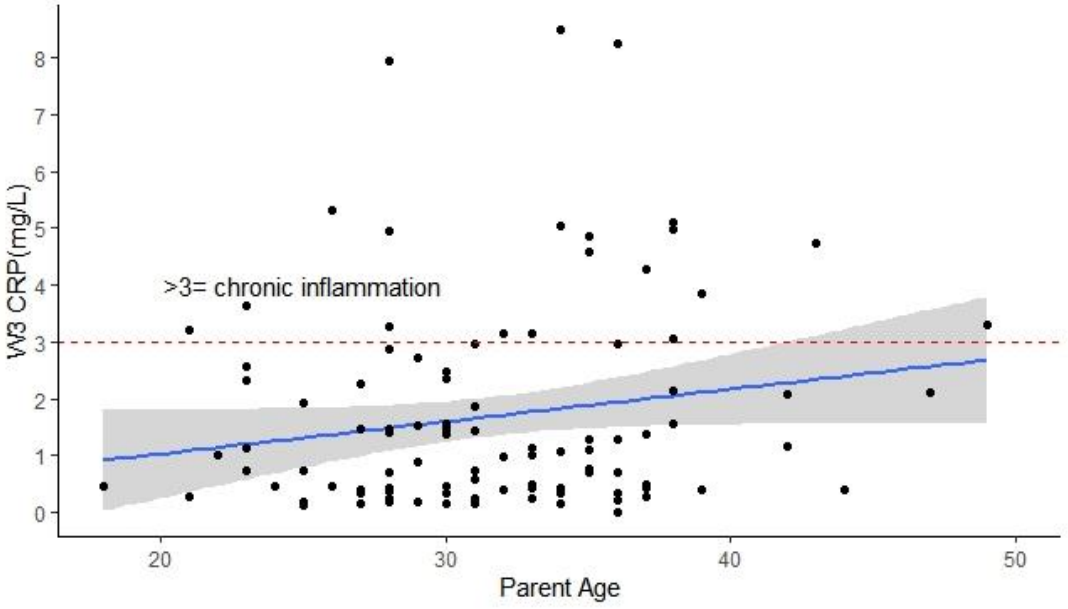


Figure 13. Mean post-treatment CRP levels by parent age.

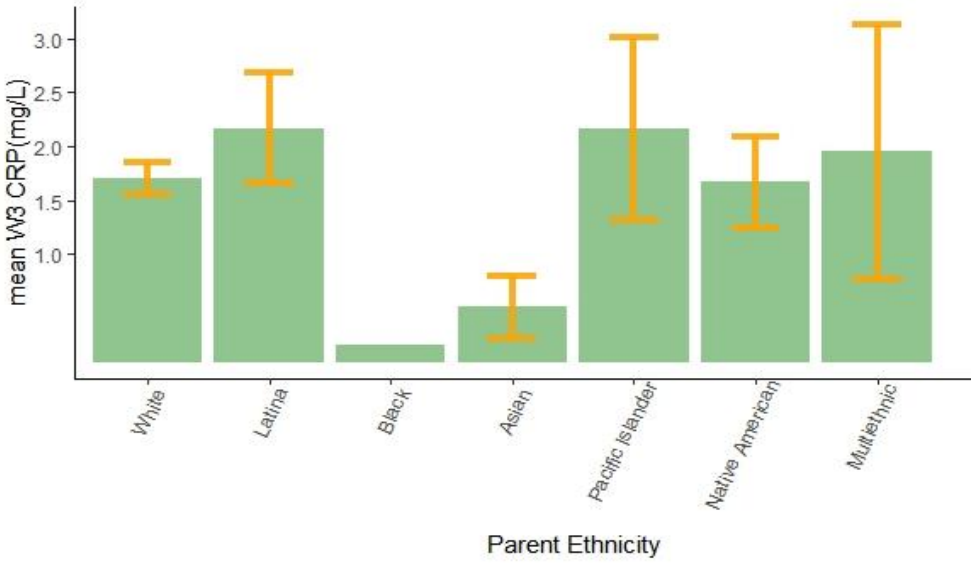


Figure 14. Mean post-treatment parent CRP levels by parent race/ethnicity.

Similar to patterns observed in baseline data, post-treatment parent CRP showed a highly significant, positive association with post-treatment BMI [r (168) = .30, p <.001]. Parents' waist circumference data were not available at post-treatment. Figure 15 maps associations between CRP and subjective SES ratings relative to a parent's community (panel A) and to the entire United States (panel B). Post-treatment parent CRP was not associated with any measured SES indicators [household income: r (168) = .11, p = .54; subjective community SES: r (168) = -.13, p = .18; subjective U.S. SES: r (168) = -.11, p = .20]. Mean post-treatment CRP levels by parent ACE score are shown in Figure 16. This appears to show higher CRP scores among parents with exposure to no adverse childhood experiences. However, there is not a statistically significant zero-order correlation between post-treatment CRP and parent ACE score [r (168) = -.11, p =.31] and the number of parents reporting no exposure to ACEs was quite small (n =3). Post-treatment CRP did not show associations with post-treatment depression symptoms [see Figure 17; r (168) = -.06, p = .51] or with parenting stress [see Figure 18; Dysfunctional Parent-Child Interactions: r (168) = -.02 p = .86; Difficult Child: r (168) = -.03, p = .83]. Associations between post-treatment CRP concentrations and other study variables did not differ when considered separately among treatment group parents (see Table 3.3) and control group parents (see Table 3.4).

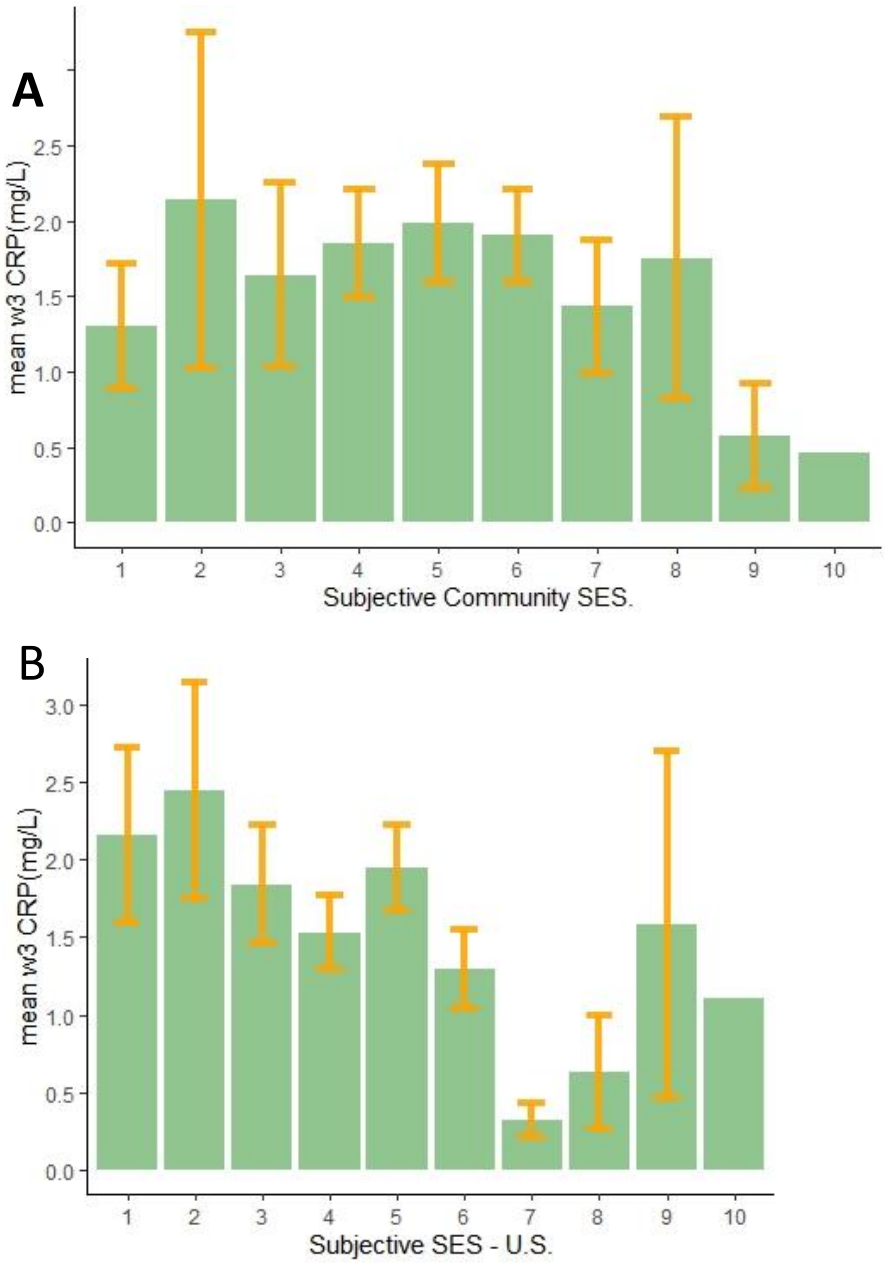


Figure 15. Mean post-treatment CRP levels by parent subjective SES ratings within community (A) and United States (B).

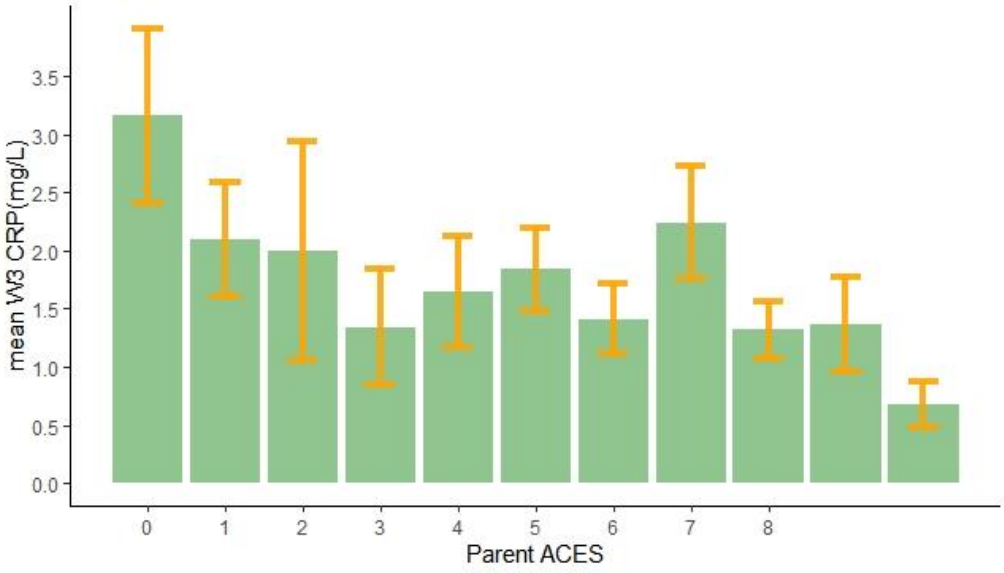


Figure 16. Mean post-treatment parent CRP level by adverse childhood experiences score.

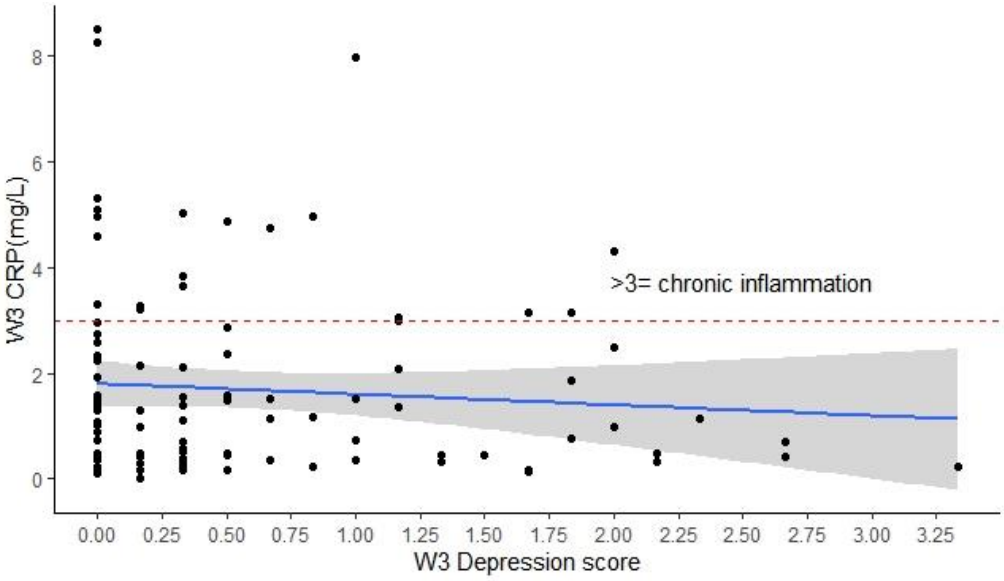
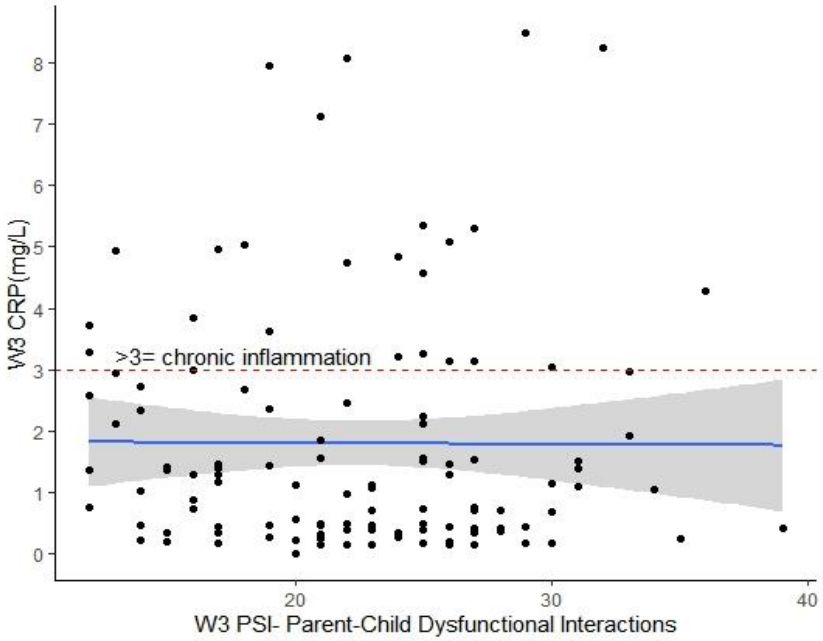


Figure 17. Associations between post-treatment CRP and parent depression score.



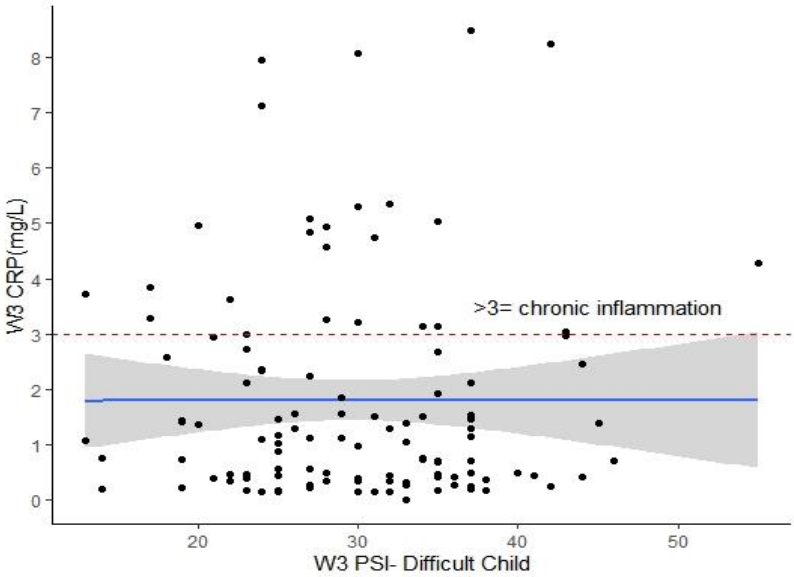


Figure 18. Associations between post-treatment parenting stress indices and parent CRP.

Main Analyses

Based on these preliminary results, I determined to include parent waist circumference as a covariate in all main analyses, along with parent age, consistent with prior empirical work linking waist circumference and age with chronic inflammation, but not with the intervention condition (PCIT vs. SAU). I also included pre-treatment CRP levels as a covariate in order to examine PCIT treatment effects on post-treatment CRP, controlling for variation in CRP concentration at study-entry.

Aim 1

To test the hypothesis that PCIT would reduce parent CRP, I conducted a series of one-way analysis of covariance (ANCOVA) tests in SPSS v. 26 to assess differences in post-treatment parental CRP between the control and intervention groups, controlling for parent age and waist circumference. I completed Levene's test and confirmed that the assumption for homogeneity of regression was met, as well as assumption of multivariate normality. I assessed for multicollinearity and found that associations among included covariates were within acceptable limits (.04 to .50; see Table 2). To investigate possible sex differences in treatment response, I performed each ANCOVA test separately for male and female parents, in addition to the full sample. In order to address potential bias associated with multiple imputation, I have reported analyses below both using the imputed data as well as a more conservative, complete case analysis using observed data.

Intention-to-treat approach. I first conducted ANCOVA tests using an intention-to-treat (ITT) approach, comparing post-treatment CRP among treatment-assigned parents with control group-assigned parents, controlling for baseline CRP levels,

parent waist circumference, and parent age. Results with the imputed data revealed no statistically significant difference between treatment and control groups on parent post-treatment CRP [F (1, 160) = .01, p =.36, $\eta^{2 =}$ .00]. Pre-treatment parent CRP was a statistically significant covariate [F (1, 160) = 25.29, p <.001, $\eta^{2 =}$ .14], as were parent waist-circumference [F (1, 160) = 16.81, p <.001, $\eta^{2 =}$ .10] and age [F (1, 160) = 5.10, p = .03, $\eta^{2 =}$ .03]. There were also no significant differences between treatment and control group post-treatment CRP when considering mothers [F (1, 138) = .28, p =.60, $\eta^{2 =}$ .00] and fathers [F (1, 17) = .39, p =.54, η^2 = .02] separately. For mothers, pre-treatment CRP [F (1, 138) = 21.53, p <.001, $\eta^{2 =}$ .14] and waist circumference [F (1, 138) = 15.56, p <.001, $\eta^{2 =}$ .10] were statistically significant covariates, but age was not [F (1, 138) = 4.11, p = .05, η^2 = .03]. For fathers, none of the covariates were statistically significant, including pre-treatment CRP levels [F (1, 17) = .3.64, p =.07, η^2 = .18], waist circumference [F (1, 17) = .01, p =.94, η^2 = .00], or age [F (1, 17) = 4.02, p =.06, η^2 = .19]. Results using complete case analysis also revealed no significant differences in post-treatment parent CRP between the PCIT and control groups [F (1, 80) = .11, p =.74, η^2 = .00]. In this model, pre-treatment CRP [F (1, 80) = 33.52, p <.001, η^2 = .30] and waist circumference [F (1, 80) = 5.81, p =.02, η^2 = .07] were statistically significant covariates, although parent age was not [F (1, 80) = .1.19, p =.74, η^2 = .02].

I also conducted a repeated measures ANOVA assessing change in parent CRP from pre- to post-treatment, controlling for parent waist circumference and parent age. Time was modeled as a within-subjects factor and treatment group was modeled as a between-subjects factor. Results indicated that the time * treatment group interaction was not statistically significant, F (1, 83) = .50, p = .48, η2 = .01. After controlling for parent

age and waist circumference, change in CRP levels from pre- to post-treatment was not significantly different among parents in the PCIT and SAU control groups. Thus, there was no evidence that PCIT lowered parent CRP over time.

Further, a post-hoc analysis of variance revealed no statistically significant differences in post-treatment parent CRP among those in the PCIT (M = 1.79, SD = .19) and SAU control group (M = 1.47, SD = .26), F (1, 102) = .96, p =.33, ηp2 = .01, after controlling for parent age and waist circumference. In this model, parent waist circumference was a statistically significant covariate, F (1, 102) = 26.28, p <.001, ηp2 = .21, parent age was not, F (1, 102) = 1.20, p =.28, ηp2 = .01.

Per-protocol treatment-engager approach. I next used a per-protocol approach to test hypothesis 1 by assessing differences in post-treatment parental CRP among parents in the control group and a treatment engager group, defined by those parents assigned to the intervention group who completed at least one treatment session. Analyses here were not conducted separately for mothers and fathers given small *n* for fathers after partitioning by engagement group. Using the imputed data, the treatment-engager model revealed no statistically significant differences in post-treatment CRP between treatment engagers and controls [F (1,134) = .46, p = .50, η^2 = .00]. Baseline CRP was a statistically significant covariate in this model [F (1,134) = 16.10, p <.001, η^2 = .11] as were parent waist circumference [F (1,134) = 9.72, p = .002, η^2 =.07] and age [F (1,134) = 4.66, p = .03, η^2 = .03]. There was also not a significant effect of PCIT using the observed data [F (1,66) = .00, p = .99, η^2 = .00]. In this model, baseline CRP [F (1,66) = 20.54, p <.001, η^2 = .24] and waist circumference [F (1,66) = 7.43, p = .01,

η^2 = .10] were statistically significant covariates, parent age was not [*F* (1,66) =1.91, *p* = .17, η^2 = .03].

Per-protocol treatment-completer approach. Finally, I conduced a set of ANCOVAs using a per-protocol approach that compared post-treatment parent CRP among control group-assigned parents and parents who completed the full course of PCIT treatment, defined as completing all of the CDI phase and at least 3 sessions of the PDI phase of treatment. Using imputed data, the treatment-completer analysis showed no statistically significant differences in CRP levels between parents assigned to the control group and those who completed PCIT [*F* (1,95) = .34, *p* = .56, η^2 = .00]. In this model, parent baseline CRP was a statistically significant covariate [*F* (1,95) = 9.90, *p* = .002, η^2 = .09] as was parent waist circumference [*F* (1,95) = 6.04, *p* = .02, η^2 = .06]; parent age was not a statistically significant covariate [*F* (1,95) = .71, *p* = .40, η^2 = .01]. Using complete cases, there was also not a statistically significant difference observed between PCIT completers and controls [*F* (1,48) = .02, *p* = .90, η^2 = .00]. Pre-treatment CRP [*F* (1,48) = 8.15, *p* =.01, η^2 = .15] and waist circumference [*F* (1,48) = 8.22, *p* =.01, η^2 = .15] were statistically significant covariates, whereas age was not [*F* (1,48) = .26, *p* =.61, η^2 = .01].

Aim 2

I conducted mediation analyses using Hayes' PROCESS macro v. 3.5 (Hayes & Preacher, 2013) to assess indirect effects of PCIT on post-treatment parent CRP, through PSI parenting stress. Analyses were conducted separately for PSI subscales Difficult Child and Parent-Child Dysfunctional Interactions. Results provided no evidence of

indirect of PCIT on parent CRP through either PSI Difficult Child subscale (Figure 19) or PSI Parent-Child Dysfunctional Interactions parenting stress symptoms (Figure 20).

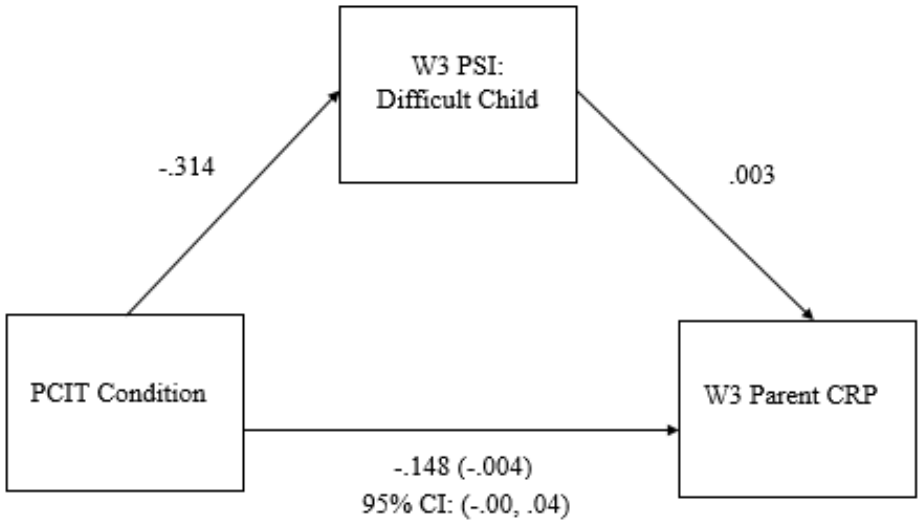


Figure 19. Mediation model for indirect effects of PCIT on parent CRP through parenting stress – Difficult Child subscale.

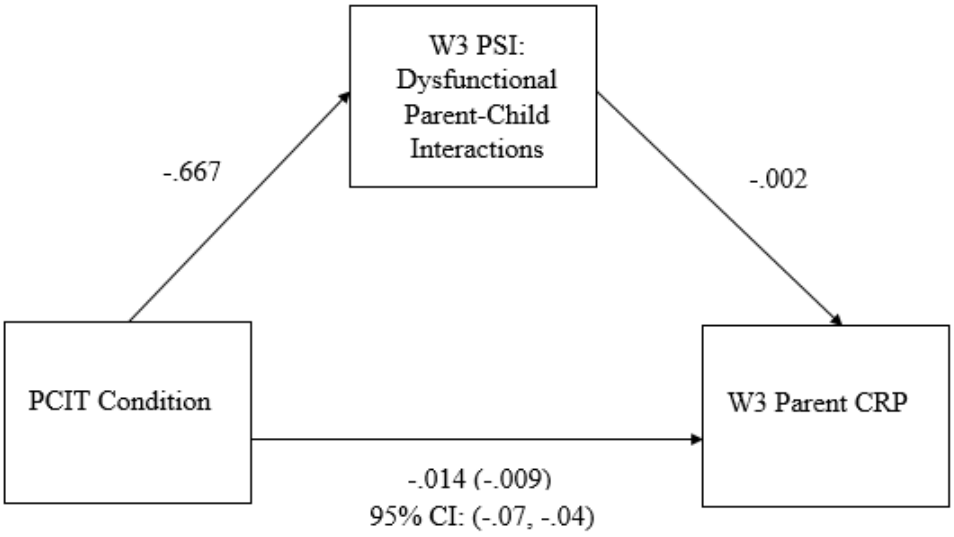


Figure 20. Mediation model for indirect effects of PCIT on parent CRP through parenting stress – Dysfunctional Interactions subscale.

Aim 3

To test the hypothesis that the effect of PCIT on parental CRP would vary based on parental characteristics, I next conducted a series of regression moderation analyses using Hayes' PROCESS macro v. 3.5 (Model 1; Hayes & Preacher, 2013). Because PROCESS requires complete data and does not accommodate imputed datasets, complete case analysis was used for each moderation analysis described below. All interaction terms were derived from centered variables. I assessed multicollinearity of variables in each moderation model and found them to be within an acceptable range, (i.e., .10 to .50; see Table 2.1).

First, I ran a hierarchical regression model to assess whether effects of PCIT on post-treatment parent CRP varied by parent sex, controlling for baseline CRP levels, parent age, and waist circumference. The overall model was statistically significant [$R^{2 =}$ .50, F (6, 78) = 13.30, p < .001]. In step 1, parent baseline CRP, age, and waist circumference were included. Baseline CRP was a statistically significant predictor of post-treatment CRP [b = .36, t (78) = 4.63, p <.001] as was parent waist circumference [b = .01, t (78) = 6.03, p <.001], but parent age was not [b = .00, t (78) = 1.27, p = .21]. In step 2, there was no significant main effect observed for either parent sex [b = .11, t (78) = 1.51, p = .13], or PCIT condition [b = .02, t (78) = .42, p = .68]. In the final step, an interaction term between PCIT and parent sex did not predict post-treatment CRP levels [b = .07, t (78) = .52, p = .60], nor explain additional variance in post-treatment parent CRP [ΔR^2 = .00., F (1, 78) = .52, p = .47]. Thus, parent sex did not moderate the effect of PCIT on CRP, (see Table 4.1). To explore whether inclusion of covariates may have limited statistical power to detect interaction effects in this small set of complete cases, I

additionally performed each moderation model without covariates. As shown in Table 4.2, null main effects of PCIT group and parent sex and null effect of the interaction term were also observed when covariates were excluded.

Table 4.1

No Evidence Parent Sex Moderates effect of PCIT on CRP, Covariates Included

Variable	B	SE	*t*	Sig.	95% Confidence Interval for B	
					Lower	Upper
(Constant)	-.49	.15	-3.34**	.00	-.78	-.20
Treat Group	.02	.04	.42	.68	-.07	.10
Sex	.11	.07	1.51	.13	-.03	.25
PCIT X Sex Interaction	.07	.13	.52	.60	-.20	.34
Baseline Log CRP	.37	.08	4.78**	.00	.22	.53
Waist Circumference	.01	.00	6.03**	.00	.00	.01
Age	.00	.00	1.27	.21	-.00	.01

Note. * = $p < .05$, ** = $p < .01$.

Table 4.2

No Evidence Parent Sex Moderates effect of PCIT on CRP, Covariates Excluded

Variable	B	SE	*t*	Sig.	95% Confidence Interval for B	
					Lower	Upper
(Constant)	.35	.02	14.60**	.00	.30	.40
Treat Group	.01	.05	.10	.92	-.09	.10
Sex	.12	.08	1.52	.13	-.04	.29
Treat X Sex Interaction	.17	.16	1.06	.29	-.15	.48

Note. * = $p < .05$, ** = $p < .01$.

I next conducted a hierarchical regression model to assess whether effects of PCIT on post-treatment parent CRP varied by parent age, controlling for pre-treatment CRP and waist circumference. The overall model was statistically significant, [R^2 = .50, $F(5,79) = 13.33$, $p < .001$]. In step 1, baseline parent CRP levels [b = .36, t (79) = 4., p <.001] and parent waist circumference [b = .01, t (79) = 6.35, p <.001] were revealed to be statistically significant predictors. In step 2, there was no significant main effect for either parent age [b = .00, t (78) = 1.20, p = .23] or PCIT condition [b = .02, t (78) = .54, p = .59]. In the final step, the parent age X PCIT interaction term was not a significant predictor of post-treatment CRP [b = -.01, t (78) = -1.49, p = .23], and did not explain additional variance in CRP levels [ΔR^2= .00., F (1, 78) = .64, p = .42], revealing that parent age did not moderate the effect of PCIT on parent CRP (see Table 5.1). Null results across treatment group main effect, age main effect, and their interaction term were also obtained when this analysis was conducted without covariates (see Table 5.2).

Table 5.1

No Evidence Parent Age Moderates Effect of PCIT on CRP, Covariates Included

Variable	B	SE	*t*	Sig.	95% Confidence Interval for B	
					Lower	Upper
(Constant)	-.57	.26	-3.41**	.00	-.90	-.24
Treat Group	.02	.04	.54	.59	-.06	.11
Age	.00	.00	1.20	.23	-.00	.02
PCIT X Age Interaction	-.01	.01	-1.49	.23	-.00	.01
Log Baseline CRP	.36	.08	4.63**	.00	.21	.52
Waist Circumference	.01	.00	6.35**	.00	.01	.01

Note. * = $p < .05$, ** = $p < .01$.

Table 5.2

No Evidence Parent Age Moderates Effect of PCIT on CRP, Covariates Excluded

Variable	B	SE	*t*	Sig.	95% Confidence Interval for B	
					Lower	Upper
(Constant)	.35	.02	14.74**	.00	.30	.40
Treat Group	.03	.05	.61	.55	-.07	.13
Age	.01	.00	2.04*	.04	.00	.02
Treat X Age Interaction	-.00	.01	-.29	.78	-.02	.02

Note. * = $p < .05$, ** = $p < .01$.

Finally, I performed a moderation analysis to examine whether intervention effects on parent post-treatment CRP varied by levels of parent ACE score, controlling for pre-treatment CRP, parent waist circumference, and age. The overall model was statistically significant [$R^2 = .49$, F (6,78) = 11.50, $p < .001$]. In step 1, pre-treatment CRP [$b = .38$, t (78) = 4.72, $p < .001$] and parent waist circumference [$b = .01$, t (78) = 5.75, $p < .001$] were significant predictors of CRP, although parent age [$b = .01$, t (78) = 1.38, $p = .17$] was not. In step 2, no significant main effect was found for either PCIT condition [$b = .02$, t (78) = .42, $p = .68$] or parent ACE scores [$b = -.01$, t (78) = -1.06, $p = .29$]. The interaction term between PCIT and parent ACE scores was not a significant predictor of post-treatment CRP [$b = -.00$, t (78) = -.03, $p = .98$] and did not explain added variance in post-treatment parent CRP levels [$\Delta R^2 = .00.$, F (1, 78) = .25, $p = .62$]. Therefore, parent ACE scores did not moderate the effect of PCIT on parent CRP (see Table 6.1). Null results for main effect of treatment group and parent ACE score as well as their interaction were also obtained when this analysis was performed without covariates

Table 6.1

No Evidence Parent ACE Score Moderates Effects of PCIT on CRP, Covariates Included

Variable	B	SE	*t*	Sig.	95% Confidence Interval for B	
					Lower	Upper
(Constant)	-.66	.20	-3.27**	.00	-1.06	-.26
Treat Group	.02	.04	.42	.68	-.07	.10
ACE Score	-.01	.01	-1.06	.29	-.02	.01
Treat X ACEs Interaction	-.00	.02	-.03	.98	-.03	.04
Log Baseline CRP	.38	.08	4.72**	.00	.22	.54
Waist Circumference	.01	.00	5.75**	.00	.00	.01
Age	.01	.00	1.38	.17	-.00	.01

Note. * = $p < .05$, ** = $p < .01$.

Table 6.2

No Evidence Parent ACE Score Moderates Effects of PCIT on CRP, Covariates Excluded

Variable	B	SE	*t*	Sig.	95% Confidence Interval for B	
					Lower	Upper
(Constant)	.35	.02	14.94**	.00	.31	.40
Treat Group	.01	.05	.23	.82	-.09	.10
ACE Score	-.02	.01	-1.43	.16	-.03	.01
Treat X ACE Interaction	-.02	.02	-.99	.32	-.06	.02

Note. * = $p < .05$, ** = $p < .01$.

CHAPTER IV

DISCUSSION

Summary

The current study aimed to investigate effects of PCIT on a biomarker of chronic inflammation (i.e., CRP) among child welfare-involved parents, to test parenting stress as a possible mediator of treatment effects, and to explore parent characteristics (i.e., age, sex, exposure to adverse childhood experiences) that may moderate effects of PCIT on CRP. Given that brief, behavioral therapies (i.e., mind-body therapies and cognitive behavior therapy; Bowen & Irwin, 2016; Lopresti, 2017) have been shown to confer inflammatory benefits in adults and one study documented lower inflammation among youth following a parent training program (Miller et al. 2014), I hypothesized that PCIT would reduce parent chronic inflammation. Based on broad knowledge that the quality of key relationships influences physical health (Kiecolt-Glaser et al., 2010; Umberson et al., 2010) and limited evidence that relational stress may predict levels of inflammatory. markers over time, at least in adolescents (Chiang et al., 2012; Fuligni et al., 2009), I posited that PCIT would reduce parent inflammation through lowering parenting stress, a well-documented outcome of PCIT (Thomas et al., 2017). I also sought to explore parent characteristics and risk factors that may moderate effects of PCIT on CRP, and expected that treatment effects may be amplified among older parents, female caregivers, and parents with greater exposure to adverse childhood experiences. Overall, study hypotheses were not supported. Results provided no evidence that post-treatment CRP was lower among parents randomized to PCIT, relative to the family services-as-usual control, and no evidence that parenting stress levels were related to parents' CRP

concentrations at either study entry of post-treatment. Moreover, there was no evidence of treatment effects on CRP when parent age, parent sex, or parent ACE scores were considered as moderators.

Patterns of CRP in Current Sample

A range of parent CRP levels were observed in the current sample, including levels indicative of chronic, low-grade inflammation. At baseline, although a majority of parents displayed low, healthy levels of CRP, indicating an absence of peripheral inflammation, CRP concentrations suggesting chronic, low-grade inflammation (i.e., > 3.0 mg/L but <10.0 mg/L) were displayed by approximately one third of parents. Further, parents reported high rates of sociodemographic characteristics and health factors typically associated with chronic, low-grade inflammation. Over half of study parents were obese and reported daily cigarette use. Parents were also predominantly low-income (i.e., nearly half reported receiving financial aid from Department of Human Services) and reported high rates of adversity in their own childhoods (nearly three-quarters reported four or more ACEs). A large, significant, positive association was observed between parents' CRP levels and their BMI and waist circumference, showing that, consistent with prior research (Brooks et al., 2010), obesity was a strong predictor of chronic inflammation in this sample. However, parent CRP was not significantly related to any other parent characteristics (i.e., age, sex, race/ethnicity) or common psychosocial risk factors for inflammation (i.e., income, subjective SES, ACE score, cigarette use, depression symptoms).

This lack of associations is surprising, given the literature base documenting that chronic inflammation is related to psychosocial risk factors such as childhood adversity

(Nusslock & Miller, 2016; Lob, Lacey, & Steptoe, 2019) and current, low SES (Gruenewald et al., 2009). One possible explanation for the lack of associations between parent CRP and psychosocial risk factors in our sample is the high rate of parent medication use prior to collecting dried blood spots (DBS). Although data were excluded from the main analyses for parents who reported use of major anti-inflammatory medications (e.g., NSAIDs, acetaminophen, antihistamines), over two-thirds of parents reported use of any medication prior to the post-treatment assessment, and many parents reported multi-medication use. Thus, while steps were taken to attempt to control for parent medication use, substantial medication use remained among parents who were retained in the study, which may have obscured true patterns of chronic inflammation in this sample. This notion is in line with previous studies that have reported null effects of behavioral interventions and failed to find associations between CRP and psychosocial variables when participants reported high rates of medication use (Antoni et al. 2009; Claesson et al., 2006; Hermanns et al., 2015).

Behavioral Interventions to Reduce Inflammation

No previous study has examined potential impacts of PCIT on parental inflammation. While the current investigation was a novel effort, findings from other behavioral interventions shown to impact inflammatory processes in adults can offer insight regarding the conditions under which CRP changes might be expected. Mind-body therapies (MBTs), including yoga, Tai Chi, Qigong, and meditation-based therapies such as Mindfulness-Based Stress Reduction, have been shown through randomized controlled trials to reduce circulating CRP (Chen, 2010; Lavretsky et al., 2011; Malarkey et al., 2013) as well as cellular markers of inflammation (Irwin et al., 2014, 2015;

Kiecolt-Glaser et al. 2014) and pro-inflammatory gene expression (Black et al., 2013; Bower et al. 2014). Inflammation lowering effects of MBTs have been recorded in healthy adults (Chen, 2006), adults receiving complementary treatment for medical conditions (Chen, 2010; Oh, et al., 2012), and older adults (Irwin & Olmstead, 2012). However, a number of other MBT studies have returned null results on inflammation (see Bower & Irwin, 2014 for a review). Cognitive behavioral therapy (CBT) has also been shown to reduce circulating inflammatory markers in adults receiving treatment for depression or medical condition-related stress (e.g., Huebner et al., 2016; Moreira et al., 2015), although findings in this literature are mixed as well (see Lopresti, 2017 for a review). What are the mechanisms by which MBTs and CBT impact inflammation, and how might these differ from treatment mechanisms of PCIT?

Mechanisms of Behavioral Interventions that Lower Inflammation

Broadly speaking, MBTs are thought to operate through reducing stress reactivity (Bower & Irwin, 2014). Although MBTs encompass a diverse set of practices, active ingredients shared across interventions have been identified as breathing practice, focused attention, and physical movement (Bower & Irwin, 2014). There is some evidence that physical movement may be a critical component for reducing CRP in particular; one meta-analysis of MBT effects reported CRP reductions only in trials employing yoga, Tai Chi, or Qigong, but not meditation interventions (Morgan et al., 2014). Further, across modalities, MBTs share an emphasis on daily practice of stress reduction techniques (Bower & Irwin, 2014). Less is known about potential mechanisms by which CBT may reduce inflammation. CBT generally focuses on improving coping with stress and negative emotion, and it has been proposed (Lopresti, 2017) that CBT

may lower inflammation either through the use of coping strategies which reduce stress (Kang et al., 2011), or through lifestyle improvements that result from positive coping practice. For example, engagement in daily pleasurable activities may serve to increase physical activity levels (Sin, Graham-Engeland, & Almeida, 2015) and relaxation practices may lead to improved sleep (Irwin, Olmstead, & Carroll, 2016). Indeed, some of the largest effect sizes for intervention-driven CRP reductions in Lopresti's 2017 review were observed in studies of CBT for Insomnia (CBT-I; Chen et al., 2011; Irwin et al., 2014, 2015). Thus, although the primary therapeutic goal of MBTs and CBT is to reduce stress (reduce stress reactivity in MBTs, improve coping with stress in CBT), their effects on inflammation may, in fact, be driven by improvements in health behavior such as sleep and physical activity that are either directly targeted (as in Yoga, Qigong, Tai Chi, and CBT-for Insomnia) or indirectly influenced by teaching positive coping strategies (as in CBT).

In contrast to MBTs and CBT, which work to reduce stress through a broad range of lifestyle improvements, PCIT more narrowly targets the quality of the parent-child relationship by increasing positive parenting behaviors and reducing harsh parenting behaviors. If improvements in sleep and physical activity are, in fact, critical components of MBTs and CBT for inflammation, then it could be that a strictly psychosocial intervention, such as PCIT, is unlikely to affect immune processes. More research on these interventions is needed with study designs that allow for possible mechanisms to be compared in order to disentangle the effects of stress reduction versus health behavior improvement on inflammation. However, there is evidence that MBTs also have the capacity to target neural mechanisms involved in threat reactivity processing (Creswell &

Lindsay, 2014), suggesting that intervention-driven improvements in stress reactivity may influence inflammatory processes. For example, MBTs have been shown to decrease sympathetic nervous system activity and increase parasympathetic nervous system activity, both at rest and in response to a stressor (Audette et al., 2006; Creswell & Lindsay, 2014: Motivala et al., 2006). There is also some evidence that meditation practice is associated with decreased activity in the amygdala (Golden & Gross, 20101; Taren et al., 2015), a brain region involved in responding to threat. Such intervention-related improvements in neural threat reactivity and sympathovagal balance may in turn lower inflammation through reduced adrenergic signaling (Nusslock & Miller, 2016), leading to the post-MBT reductions in circulating CRP seen in some studies (Bower & Irwin, 2016). Thus, while changes in health behavior likely account for some of the effects on inflammation seen in CBT and MBTs, evidence of changes in physiological stress responses systems that operate upstream from the immune system seem to suggest that intervention-driven improvements in stress reactivity also play a role in lowering inflammation.

Furthermore, findings by Miller and colleagues (Miller, Brody, Yu, & Chen, 2014) have demonstrated that a strictly psychosocial parenting intervention can lower inflammation, albeit among adolescents, rather than parents. In their longitudinal trial of Strong African American Families, a 7-week positive parenting program delivered to low-income families in the rural South, they found that youth of parents who received the intervention showed lower levels of six pro-inflammatory cytokines at an 8-year follow-up, relative to controls. These findings are also consistent with some evidence from observational, longitudinal studies which show that adolescents' reports of stress or

conflict in close family and friend relationships correspond to levels of inflammatory markers over time (Chiang et al, 2012; Fuligni et al., 2009). In light of this evidence, why were inflammation reductions not observed from PCIT, an intervention known to show robust effects in strengthening the parent-child relationship (Thomas et al., 2017)? The treatment goals of PCIT are similar to SAAF and include improving parent-child relationships and building child socioemotional competency (Brody et al., 2004; Eyberg et al., 1995). PCIT is a more intensive parenting intervention, delivered individually with live coaching versus the group format used in SAAF. PCIT intervention dosage is higher also higher compared to SAAF; in the current study, families received an average of 12 treatment sessions compared to the 7 sessions reported by Miller et al. (2014).

One explanation is that improvements in the parent-child relationship may be more salient for the immune systems of children, compared to caregivers. Developmental science has established the critical importance of the quality of early caregiving for the developing neuroendocrine stress response systems of children (Kuhlman, Chiang, Horne, & Bower, 2017; Nusslock & Miller, 2016). Although it is not yet clear at which stage in development the effects of the early environment on immune response may manifest, there is evidence that exposure to maltreatment (Coehlo, Viola, Walss-Bass, Brietzke, & Grassi-Oliveira, 2014) or harsh parenting (Brody, Yu, Beach, Kogan, Windle, & Philibert, 2014) in early and middle childhood impacts immune responding and may lead to chronic inflammation by adolescence (Brody et al., 2014). Thus, there is a strong empirical and theoretical basis for Miller and colleagues' 2014 finding that a positive parenting program delivered in middle childhood protected against immune dysregulation for vulnerable youth over the following 8 years. In contrast, research has

not delineated whether parents' stress response systems are more or less impacted by the quality of interactions with their child, compared to the quality of other key relationships, or to more global levels of psychosocial stress. It may be that changes in the quality of the parent-child relationship have less impact on the stress response systems (and, in turn, inflammatory processes) of adult caregivers, relative to the profound developmental significance the quality of this relationship has for a child. This notion appears to be supported by the fact that each of the findings reviewed here showing significant associations between the quality of the parent-child relationship and inflammation, either as observed over time (Chiang et al., 2012; Fuligni et al., 2009; Marin et al., 2009) or driven by intervention (Miller et al., 2014), involved adolescents or young adult offspring. However, the paucity of research examining the effect of the parent-child relationship on *parent* stress responding makes it difficult to draw conclusions here.

Another notable difference between the current study and Miller and colleagues' 2014 study is the length to follow-up for testing intervention-related effects on inflammation. Miller et al. (2014) found treatment effects on youths' inflammation 8 years following treatment, whereas we found no effects on CRP immediately following treatment completion. Thus, an alternative explanation is that, within the current study design, CRP was simply assessed too early to allow for potential changes to be detected. PCIT is known to have powerful effects on improving parenting behavior (Batzer et al., 2018; Cooley et al., 2014) and reducing child problem behavior (Thomas et al, 2017). However, it is possible that behavioral gains evident at post-treatment do not translate into corresponding neurobiological improvements until later. While it is unknown how long after competing PCIT changes in CRP would be expected to emerge, findings from

the mind-body therapy (MBT) literature offer clues about the time course of intervention-driven changes in inflammation. As reviewed by Bower and Irwin (2014), effects immediately following an MBT intervention are most often reported on inflammatory gene-expression pathways (e.g., Black, Irwin, Olmstead, Crabb Breen, & Motivala, 2014; Creswell et al., 2012), whereas effects on circulating pro-inflammatory markers appear to emerge over a longer period. For example, in their RCT assessing effects of a 12-week yoga intervention on inflammation among 200 breast cancer survivors, Kiecolt-Glaser and colleagues (2014) found no effect on Il-6, TNF, and IL-1 immediately following treatment, but did observe reductions at a 3-month follow-up (Kiecolt-Glaser et al., 2014). Moreover, because CRP production is regulated in part by IL-6, CRP changes occur slightly further downstream in the inflammatory process and may not be detectable as early as changes in IL-6 and other inflammatory cytokines. Illustrating this, among the small subset of MBT trials reviewed by Bower & Irwin (2014) that focused on Tai Chi among older adults, IL-6 reductions were observed in one study as early as 16 weeks post-intervention (Irwin & Olmstead, 2012), whereas Tai Chi-related reductions in CRP were observed in a similar study after 24 weeks (Lavretsky et al., 2011). Taken together, evidence suggests that the brief longitudinal design of the current study may have limited our ability to accurately assess intervention-related CRP changes. It is possible that CRP changes could have been observed in the current sample if measured via proinflammatory gene expression, or that changes in circulating CRP may have emerged if assessed at a 6- or 12-month follow-up.

Conclusions

Hypotheses in the current study were not supported; I found no evidence that PCIT impacted parent CRP levels at post-treatment and no evidence that parents' CRP levels were associated with their parenting stress. Based on literature review of other behavioral interventions shown to impact inflammation (Bower & Irwin 2014: Lopresti, 2017), it is possible that PCIT does not affect inflammation because it does not involve improvements in health behaviors such as sleep and physical activity or because it narrowly focuses on improving the parent-child relationship, rather than reducing stress more broadly. Reducing stress reactivity, in particular, appears to be a critical mechanism driving the neuroendocrine changes in mind-body therapies that lead to lower inflammation (Bower & Irwin, 2014). Alternatively, it may be that the brief longitudinal design here did not allow for CRP changes to be captured over the period in which they may emerge. However, much remains to be learned about potential benefits of PCIT for parents. Future studies should assess whether PCIT reduces stress reactivity and examine potential effects of PCIT on other physiological systems, such as the HPA-axis or autonomic nervous system, to learn whether positive behavioral outcomes of PCIT may confer benefits on more upstream processes in the body's response to stress. Results of such studies could help determine whether continued investigation of PCIT and chronic inflammation are warranted.

Although parent CRP levels were related only to parent BMI and waist circumference in the current study, parents did show high rates of several other risk factors for chronic inflammation, such as daily cigarette use, elevated parenting stress and depression symptoms. Moreover, these psychosocial risk factors were higher among

parents who reported greater exposure to adverse childhood experiences or lower subjective SES. This highlights how multiple risk factors for poor health outcomes can accumulate among socially disadvantaged individuals, such as parents involved with child welfare. PCIT is known to be an effective intervention for reducing child maltreatment (Chaffin et al., 2011), one critical public health problem experienced by child welfare-involved families. Continued research is needed to identify preventive interventions that can target the range of health problems these families may face, in order to reduce the burden of stress-related disease in these populations.

Study Strengths

The current study used data from a randomized controlled trial (RCT) design, which bolstered internal validity in examining the impact of PCIT on parent low-grade inflammation. Collection of parent measures at pre- and post-treatment allowed the ability to assess CRP changes across a brief time period, while controlling for some possible confounds. The study is also strengthened by the high-risk nature of the sample of child-welfare involved parents. With Miller and colleagues' work as an important exception (Miller et al., 2014) this population is underrepresented in research on behavioral treatments and inflammation, despite the fact that they may be disproportionately burdened by stress-related illness. Finally, the use of both ITT and per-protocol analytic approaches maximizes the external and internal validity of conclusions here, by allowing for possible treatment effects to be assessed both under the most stringent conditions as well as under conditions most generalizable to community implementation of PCIT.

Study Limitations

A number of limitations in the current study should be noted. First, I relied on a single biomarker of chronic inflammation, CRP. Collecting a full panel of pro-inflammatory cytokines (e.g., IL 1β, -6, -8, and -10, tumor necrosis factor α, IFN- γ) would have provided a more complete picture of chronic inflammation in the sample and strengthened internal validity. The brief longitudinal design also constrained our ability to detect possible changes in CRP, as we were not able to assess for changes that could have emerged at 3- or 6-months after parents completed PCIT, as has been documented in some MBT trials (e.g., Lavretsky et al, 2011). In future studies, researchers should consider whether there are ethical ways to limit participant medication use prior to collection of inflammatory markers.

Further, there are several potential confounds in the current study. As described above, high rates of parent medication use may have obscured true patterns of inflammation in study parents and thus interfered with my ability to detect treatment effects or associations between psychosocial risk variables and parent CRP. There are also a number of health-related variables which were not assessed here but may have contributed to parents' CRP levels, such as underlying parent health conditions, sleep quality, diet, frequency of physical exercise, and use of alcohol or caffeine. Although a number of measures of psychosocial risk were included (e.g., objective income and subjective SES, parent occupational and housing status) at pre- and post-treatment, we did not include a measure of other stressful life events parents may have experienced in the course of the study, such as death or incarceration of a loved one or new material hardships (e.g., loss of transportation). Researchers should incorporate measures of

stressful life events in future studies examining chronic inflammation, especially when using samples of low-income families, given they are known to experience stressful life disruptions more frequently (Brown et al., 2016).

External validity may also be limited by several sample characteristics. First, participating parents were primarily White. Higher levels of chronic inflammation have been documented among non-white ethnic groups, most consistently among Black/African American and Hispanic/Latino/a individuals (O'Connor et al., 2009), and so the relatively homogenous makeup of our sample may have limited our ability to detect effects of PCIT on CRP, especially given evidence that behavioral interventions may have greater impact on reducing inflammation among participants with higher baseline levels of inflammation (Morgan et al., 2014). Sex is another important demographic factor influencing inflammation (Khera et al., 2005) and, because fathers made up a small portion of the sample and, it was difficult to assess how results may have differed by parent sex. Finally, although this study's focus on an understudied sample of child welfare-involved parents is an important contribution to the literature, relatively high levels of psychosocial risk observed across the sample (e.g., only 3 parents reported no exposure to ACEs) may have limited statistical ability to uncover associations between psychosocial risk variables and parent CRP. The above sample characteristics also impact generalizability of the current results, which may not apply to non-White ethnic groups, men, or to lower-risk, community samples.

Future Directions

To understand whether PCIT has the ability to confer anti-inflammatory benefits, more research is needed using longitudinal designs with follow-up assessments at least 3 months after treatment completion that can measure inflammatory markers when intervention-related changes may emerge. To capture a more complete picture of low-grade inflammation, researchers should also collect a full panel of circulating anti-inflammatory cytokines in addition to CRP, and consider assessing cellular inflammatory markers or inflammatory gene expression pathways, as there is evidence treatment effects may be more readily observable in these measures of inflammation. Further, researchers should assess health behaviors such as physical activity and quality of sleep at pre- and post-treatment in order to disentangle treatment-related changes in inflammation driven by stress reduction versus lifestyle improvements.

Behavioral interventions that do lower inflammation and confer benefits across neuroendocrine stress response systems appear to work at least in part by reducing stress reactivity (Bower & Irwin, 2014). It is unknown whether the significant parent behavior changes observed in PCIT may be accompanied by broader improvements in stress reactivity, and not only improvements in parenting stress. The larger parent grant from which the current study is drawn aims to discover whether PCIT may improve biobehavioral indices of parent self-regulation, including via autonomic responding, and so will help to address this question.

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