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EFFECTS OF AN EVIDENCE-BASED PARENTING PROGRAM ON PHYSIOLOGICAL
MARKERS OF STRESS AMONG PARENTS AT RISK FOR PERPETRATION OF CHILD
ABUSE AND NEGLECT

by

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30303

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I first thank God for everything I have achieved. "O Lord of infinite prowess, my salutations to You... O soul of all, my obeisance to You from all sides indeed. You, who possess limitless might, pervade all; therefore you are all... the ruler of all, and worthy of all praise." *Srīmad Bhagavadgīta*, 11.40, 11.43

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Abstract

Introduction: Parental stress is an important risk factor for child maltreatment (CM) that can increase the likelihood of perpetration of abuse. Evidence-based, parent-training programs have shown a positive impact on preventing CM, and reducing self-reported parental stress. However, limited research among high-risk parents for CM perpetration has examined physiological correlates of stress, such as impaired cortisol, alpha-amylase, and dihydroepiandrosterone (DHEA). Because there are many challenges with validity of self-report measures, it is imperative to explore biomarkers as novel benchmarks of parental stress. Thus, the goal of this research was to conduct a quasi-experimental, mixed-methods and multidisciplinary study examining behavioral and physiological stress in response to a six-week, evidence-based program, SafeCare®, with a sample of at-risk mothers.

Methods: High-risk parents ($n=18$) were recruited from a children's hospital pediatric clinic in Atlanta, Georgia. Participants completed repeated within subject assessments of behavioral (self-report) and physiological (cortisol, alpha-amylase, DHEA) stress measures pre-and post-intervention. Acute cortisol and alpha-amylase were collected through Salivette® methods. Chronic cortisol was assessed using hair samples. DHEA was collected through passive drool samples. Participants also completed a qualitative interview at baseline. Correlational analyses were conducted to examine associations between self-reported parental stress and biomarkers. Paired *t*-test analyses were conducted to examine changes in self-reported stress and physiological markers pre-to post- intervention, as well as to examine participants' acute stress responses during a SafeCare® session in the presence of a home visitor. Qualitative analyses were conducted using line-by-line coding to examine feasibility of collecting biospecimens. In addition, themes on parental and general stress perceptions were examined.

Results: Participants were African American (M age=27.0 years, $SD=6.7$), and of low socioeconomic status (60% <\$20,000 annually), with 77%, reporting exposure to at least one lifetime traumatic event. Bivariate correlations indicated strong associations between self-reported stress and salivary cortisol levels ($r= -.70$, $p=.005$), as well as with alpha-amylase ($r=.74$, $p=.005$) among all participants at baseline. Correlations were also found between self-reported stress and alpha-amylase at follow-up ($r=.87$, $p<.05$) ($n=7$). Trends, although non-significant, were noted among completers towards decreased average self-report stress and improved salivary cortisol ($p=.08$) and alpha-amylase ($p=.08$). Participants with impaired salivary cortisol levels at baseline showed normalization post-intervention. No significant changes in participant acute stress levels were noted in the presence of the home visitor mid-intervention. Findings from qualitative interviews indicated that parents were generally willing to provide hair and salivary samples, but showed clear preference for Salivette methods over passive drool. While parents described many parental stresses addressed by SafeCare®, parents also described contextual factors such as socioeconomic status and other chronic stressors that contribute to parenting stress.

Conclusions: Study findings suggest that salivary cortisol and alpha-amylase are compelling neurobiological correlates of parental stress among high-risk parents for CM. Further, results support the short-term, positive effects of SafeCare® in potentially regulating physiological stress systems among at-risk parents. Given the feasibility noted in biomarker collection among participants, larger, and more rigorous studies should be conducted in the future to validate these results.

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Chapter 1.

INTRODUCTION AND STATEMENT OF PURPOSE

Introduction

Child maltreatment (CM) is a significant public health problem within the US. In 2013, approximately 3.9 million children were referred for CM, of which, 679,000 cases were substantiated (U.S. Department of Health and Human Services, 2015). The U.S. Department of Health and Human services reports that 91.4% of all perpetrators were parents. Parents at greatest risk for perpetrating abuse are those experiencing acute and chronic stressors, including low socioeconomic status, low household income (Chaffin, Kelleher, & Hollenberg, 1996), substance abuse (Ammerman, Kolko, Kirisci, Blackson, & Dawes, 1999; Chaffin et al., 1996; Walsh, MacMillan, & Jamieson, 2003), depression (Chaffin et al., 1996), trauma history, and significant levels of parental stress (Merrill, Hervig, & Milner, 1996). Parental stress is a particularly important risk factor since studies report that parental stress can lead to poor parent child interactions and heightened parent-child conflict. In turn, these outcomes directly increase the likelihood of future perpetration of abuse (Anthony et al., 2005; Halme, Tarkka, Nummi, & Åstedt-Kurki, 2006; Huth-Bocks & Hughes, 2008; Owen, Thompson, & Kaslow, 2006; Perren, Von Wyl, Bürgin, Simoni, & Von Klitzing, 2005; Rodgers, 1998). While studies of behavioral parent training programs, the recognized recommendation for CM prevention, have found positive outcomes on self-reported parental stress (Danforth, Harvey, Ulaszek, & McKee, 2006; Sharry, Guerin, Griffin, & Drumm, 2005), no known studies have explored effects of these programs on physiological biomarkers of stress. In addition, response bias challenges may the validity of self-report measures of stress. Thus, further understanding of other markers of parental stress, such as biomarker correlates, and their responses to behavioral parent training

programs, could advance the field; biomarkers may elucidate how such programs can have an impact on broad-based parental outcomes, including overall well-being.

Statement of Purpose

The objective of this paper was to report findings from a multidisciplinary project based in the fields of neuroscience and public health that assessed physiological stress responses to an evidence-based child maltreatment (CM) public health intervention. Specifically, this project examined how an evidence-based parent-training program, known to reduce self-reported parental stress, can affect physiological biomarkers of stress among a group of high-risk mothers for child abuse and neglect, by (1) Recruiting 18 mothers at risk of CM based on risk factors identified in research. Mothers were assigned to receive the Parent Child Interaction module of SafeCare®, an in-home evidence-based parenting program shown to reduce risk factors and self-reported parental stress associated with CM perpetration (Carta, Lefever, Bigelow, Borkowski, & Warren, 2013); (2) Conducting repetitive, within subject assessments that included self-report measures of parent stress and behavior, and physiological biomarkers including the hormones cortisol and DHEA, and salivary enzyme alpha-amylase. Steroid hormones and alpha-amylase were assessed pre- and post-intervention among 10 mothers. These biomarkers were compared to standard measures of self-reported stress typically implemented in parenting research.

Based on the existing literature, the aims of the study were as follows:

- 1) To test the hypothesis that mothers who report higher levels of self-reported stress and mental health symptomology will have impaired steroid hormone levels (i.e., cortisol, DHEA) and salivary alpha-amylase.

- 2) To test the hypothesis that parents who complete SafeCare® will show improvements in hormone production and salivary alpha-amylase, as well as in self-reported parental stress and mental health symptomology.
 - a. To further examine that parents who exhibit impaired biomarker levels (i.e., levels outside of standard physiological range) at baseline who complete SafeCare® will show normalization following the intervention.
- 3) To test the hypothesis that parents will experience increases in acute stress in the presence of a home-visitor at Session 3, mid-way through the intervention.
- 4) To assess the participants' willingness to provide physiological measures (i.e., salivary and hair samples) in a research project.
- 5) To examine maternal perceptions of general and parental stress.

Chapter 2.

REVIEW OF THE LITERATURE

Child Maltreatment Overview

Child maltreatment (CM) is a significant public health problem within the US. In 2013, approximately 3.9 million children were referred to child welfare and protective services for CM of which, 679,000 cases were substantiated (U.S. Department of Health and Human Services, 2015). The Centers for Disease Control and Prevention (CDC) defines CM as "Any act or series of acts of commission or omission by a parent or other caregiver that results in harm, potential for harm, or threat of harm to a child" (Leeb, 2008). Common forms of CM include emotional abuse, sexual abuse, physical abuse, neglect, and failure to supervise (Leeb, 2008). The U.S. Department of Health and Human Services (2015) reports that 91.4% of all perpetrators were parents, with a higher percentage of mothers (40.7%) than fathers (20.3%) acting alone. The literature is replete with studies of contextual factors that increase child risk for maltreatment, including low income, low parental education, and residing in communities with greater concentrations of disadvantage (housing stress, low social capital, lack of social support) (Kotch et al., 1997; Runyan, Wattam, Ikeda, Hassan, & Ramiro, 2002; Sidebotham, Heron, & Team, 2006). Parents at greatest risk for perpetrating abuse are those experiencing acute and chronic stressors, including part-time employment, low socioeconomic status, low household income (Chaffin et al., 1996), substance abuse (Ammerman et al., 1999; Chaffin et al., 1996; Walsh et al., 2003), depression (Chaffin et al., 1996), trauma history, and significant levels of parental stress (Merrill et al., 1996).

A Framework for Parenting among High-Risk Families for CM

In order to delineate the roles of the complex risk factors in explaining parenting among high-risk populations, Belsky (1980) proposed an ecological model that built upon Bronfenbrenner's theoretical ecological systems framework on human development (Bronfenbrenner, 1979). Belsky describes the interaction of four comprised of nested, contextual levels, each consisting of risk factors fostering CM risk. Specifically, emphasis is placed on interaction of the ontogenic level, consisting of the individual characteristics of parents and children contributing to family functioning and parenting roles (e.g., trauma history, psychopathology, parenting skills, personality traits); the microsystem, comprised of individuals (e.g., family, peers), institutions (e.g., schools), and the child's immediate environment (e.g., home environment, parental relationships, and neighborhood) that have the most direct impact on the child; the exosystem, consisting of processes taking place between multiple contexts, which do not directly involve children but have implications for their development (e.g., family's social support system, violence exposure, socioeconomic status); and the macrosystem, which describes the cultural beliefs and norms surrounding the child (e.g., attitudes and beliefs towards disciplinary methods, gender roles, family functioning, violence).

Primary Prevention Recommendations for CM

Behavioral parent training programs are recommended as the most impactful primary prevention approach for reducing risk of parent perpetrated CM (Hammond, Whitaker, Lutzker, Mercy, & Chin, 2006). Behaviorally based parent-training is based on social learning principles (Chronis, Chacko, Fabiano, Wymbs, & Pelham Jr, 2004) and includes components such as didactic instruction, modeling, and differential reinforcement (Serketich & Dumas, 1996). Behaviorally based parent-training programs attempt to teach parents effective child

management skills (Taylor & Biglan, 1998). Parents may be trained to minimize neglectful behavior and increase positive interactions with children by using playing techniques, reward systems and positive feedback. In addition, parents may be taught to set and follow clear rules and consequences for their children's behaviors and actions, and to use non-coercive discipline methods.

The SafeCare® model is an example of an evidence-based, behavioral parent-training program that focuses on reducing child neglect and abuse among families at high-risk of maltreatment (Lutzker & Bigelow, 2002). SafeCare® is conducted in the home environment and consists of three modules: health, home safety and parent child interactions (PCI). These modules address aspects of parenting behaviors (Lutzker & Bigelow, 2002), environmental, and healthcare risks, that are associated with CM (Lutzker & Bigelow, 2002). Each module follows a structured, seven-step process which includes: explaining the rationale for the behavior, demonstration of skills; practice of skills by the parent; observation and data collection of parental behavior by home visitors; positive and corrective feedback from the home visitor, additional parental demonstration of skills; and demonstration of skills to meet mastery criteria (Whitaker, Crimmins, Edwards, & Lutzker, 2008).

Several randomized trials have found benefit of SafeCare® relative to case management services or to a no treatment control, both in child welfare settings (after maltreatment has occurred) and in prevention settings (serve families at-risk for maltreatment). In the largest study to date, a statewide comparative effectiveness trial of SafeCare® in the Oklahoma child welfare system (Chaffin, Hecht, Bard, Silovsky, & Beasley, 2012), six service regions were matched and randomized to SafeCare® or to continue with services as usual. Over 2,100 families were enrolled in the study and were followed for six years, on average, following treatment. For the

child age group of 0-5 years, the primary target of SafeCare®, SafeCare® reduced CM recidivism by 26% (HR = .74) relative to services as usual. The authors concluded that SafeCare® prevented between 64 and 104 recidivistic reports per 1,000 cases relative to services as usual. Carta et al. (2013) conducted a two-site randomized trial that compared SafeCare® to a no services control group and found increased positive parenting skills, including parent-child interactions, and a higher rate of a more stimulating home environment for SafeCare® participants compared to controls. Findings from this same study indicated positive child level outcomes as well, including lower levels of externalizing behavior problems and increases in adaptive functioning (Bigelow, 2014). In a randomized trial by Silovsky and colleagues (2009) investigators found differences on a range of outcomes (parent social support, child abuse potential, parent depression) favoring SafeCare® as compared to usual services. In another randomized trial conducted in rural Oklahoma, researchers found greater service utilization, greater use of non-violent discipline, and fewer child protective services reports related to domestic violence, for SafeCare® versus services as usual (Silovsky et al., 2011).

Independently implementing the PCI module with at-risk parents has also been shown in a randomized trial to reduce risk factors and self-reported parental stress associated with CM perpetration (Carta et al., 2013). This module specifically focuses on improving and increasing positive interactions between parents and children. Parents are taught to take care of infants (parent-infant interaction), and among toddlers and older children to manage child behavior by using positive interaction and planning skills (planned activities training).

Parental Stress as a Risk Factor

Parental stress is a particularly important risk factor portrayed in CM models. While CM risk is determined by the interplay of several aforementioned factors, evidence suggests that

high-risk parents often experience elevated levels of parental stress (Haskett, Ahern, Ward, & Allaire, 2006). Parental stress is considered a multifaceted construct that encompasses the harsh reactions, stress and difficulties that may occur under the demands of parenting and parent-child relationships in daily life (Abidin, 1990; Deater-Deckard, 2008; Zaidman - Zait et al., 2010). In a theoretical model by Abidin (1992), parental stress is thought to influence children's behavioral and emotional adjustments (1992). Evidence demonstrates that perceptions of high levels of maternal parental stress are associated with decreased response sensitivity to child needs (Hibel, Mercado, & Trumbell, 2012) as well as decreased social competency and heightened reports of behavioral problems among children in the home and school settings (Anthony et al., 2005; Levendosky & Graham-Bermann, 2000, 2001; Levendosky, Huth-Bocks, Shapiro, & Semel, 2003). Further, studies report that parental stress can lead to poor parent-child interactions and heightened parent-child conflict, which directly increase the likelihood of perpetration of abuse (Anthony et al., 2005; Halme et al., 2006; Huth-Bocks & Hughes, 2008; Owen et al., 2006; Perren et al., 2005; Rodgers, 1998). Such results suggest that higher levels of parental stress limit effective parenting strategies and parental ability to provide a nurturing environment for the child.

Challenges Associated with Measuring Stress in the Literature

Individual reception and responses to behavioral parenting programs are often diverse and may be a product of current stress levels among individuals entering such services. However, the current use and acceptance of perceived stress measures limits the ability to identify sub-populations who may be at risk poor intervention outcomes based on biological, environmental factors or an interaction of both (Marshall Jr, 2011). Within the violence prevention field, common methods of measures include self-report and observational measures to

validate the efficacy of CM prevention interventions. However, these measures are often flawed in their use. For example, use of self-report measures introduce the risks in response bias (Adams et al., 2005; Babcock, Costa, Green, & Eckhardt, 2004; King & Bruner, 2000; Paulhus & Vazire, 2007), delivery of honest reports, and individuals ability to have a clear understanding of what items are asking (Paulhus & Vazire, 2007; Schwarz, 1999). Research also indicates parental overestimation in behavioral changes on self-reports in response to parenting intervention (Forehand et al., 1982). Further, observational measures are often limited by an assessor's ability to get a sample of "real behavior" and rater reliability can be difficult to achieve and exposed to bias.

To fully evaluate the effectiveness of evidence-based interventions such as SafeCare® in reducing parental stress, there is a need to understand the impact of such interventions on multilevel stress effects, beyond behavioral changes. Because there are many challenges with the validity of self-report measures, further understanding of how other markers of parental stress respond to behavioral parent-training programs may advance the field in terms of how such programs can have an impact on broad-based parental outcomes among parents with varying stress levels. Thus, inclusion of new measures, less influenced by external biases, may strengthen and validate established prevention programs, such as SafeCare®, and increase confidence that such programs result in comprehensive improvements among targeted families.

Introduction of novel measures, such as biomarkers, may enable a novel classification system of stress levels among target populations, which may guide criteria for enrollment of parents in appropriate services and treatment based on initial levels of stress. In addition, inclusion of biomarkers in public health can establish better benchmarks for intervention

outcomes. Thus, this study was designed to address both behavioral and physiological responses to SafeCare® on parental stress and biological functioning among high-risk parents.

Although studies of behavioral parent-training programs, such as SafeCare® have found positive outcomes on reductions in self-reported parental stress (Danforth et al., 2006; Sharry et al., 2005), limited research exists on effects of these programs on psychophysiological functioning among parents. Furthermore, little knowledge is known on the correlations between physiological measures and self-report levels of stress. Such physiological measures may explicate the mechanisms between risk factors and disruptions in biobehavioral functioning (Repetti, Taylor, & Seeman, 2002).

Biological Measures of Stress

Several steroid hormones have been established as indicators that reflect mechanisms of the two major physiological stress response systems, the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS). The following sections present a discussion of stress research conducted on recognized hormone biomarker correlates of the HPA axis and SNS, particularly, cortisol, and alpha-amylase, respectively, as well as an additional steroid hormone: dihydroepiandrosterone (DHEA).

Cortisol. Cortisol is a steroid hormone synthesized and secreted by the adrenal cortex during periods of acute and chronic stress. The steroidal characteristics of cortisol mean that it is fat-soluble and can be secreted in saliva and accumulated in hair, therefore making it non-invasive to measure. It is an essential factor regulating HPA functioning, implicated by neuroendocrine models as the primary hormone that exacerbates debilitating effects of chronic stress among several physiological outcomes, including but not limited to diabetes (Björntorp &

Rosmond, 1999), obesity (Epel et al., 2000; Gluck, Geliebter, & Lorence, 2004), cancer (Carlson, Speca, Patel, & Goodey, 2004; S. Sephton & Spiegel, 2003; S. E. Sephton, Sapolsky, Kraemer, & Spiegel, 2000), arthritis (Catley, Kaell, Kirschbaum, & Stone, 2000; Heijnen & Kavelaars, 2005; Neeck, Federlin, Graef, Rusch, & Schmidt, 1990) and depression and schizophrenia (McEwen, 2000).

In early childhood, the developing HPA axis is under powerful social regulation (Levine, 2005; Tarullo & Gunnar, 2006). High and low circulating cortisol levels influence the manner in which neural circuits perceive and interpret environmental threats and the magnitude and duration of future stress responses (McEwen & Stellar, 1993; Tarullo & Gunnar, 2006). Both hyper- (elevated) and hypo (reduced)-cortisolism can reflect allostatic load, typically defined as the result of chronic exposure to fluctuating or heightened neural/neuroendocrine responses that emerge in response to chronic environmental challenges that are perceived as especially stressful.

Cortisol Studies of Stress among Maternal Populations. Within the maternal and child health literature, few studies have assessed cortisol stress regulation at the caregiver level in the context of CM. Two known studies have documented effects of factors associated with CM on cortisol regulation among a sample of employed women. In the first study, researchers assessed the effects of marital status, the number of children residing in a household and social support on daily cortisol excretion among participants. These researchers report higher levels of excreted cortisol being significantly associated with reports of having at least one child residing at home (Luecken et al., 1997). In the other published study, researchers found evidence of interaction effects between reports of increased parenting stress and job stress among a sample of 56

working mothers of preschool children in predicting levels of morning cortisol levels (Hibel et al., 2012).

Limited research has focused on cortisol responses to parenting programs among maternal populations. One known study has evaluated the effects of a coaching intervention on cortisol regulation among normal, healthy mothers of young children in Japan. This randomized controlled trial found that a 3-month group coaching program on self-management and stress cognition was associated with significant changes in cortisol levels and emotional intelligence among engaged parents (Ohashi & Katsura, 2015). Only one parenting intervention study has documented the influence of behavioral programs on reducing physiological stress among high-risk parents for CM. Toth et al. (2015) conducted two randomized controlled trials to examine effects of two theoretically-based preventive interventions, Child-Parent Psychotherapy, and Psychoeducational Parenting Intervention, on biobehavioral stress (self-report and cortisol) among maternal-child dyads, with Child Protective Service reports of CM neglect. Researchers noted significant decreases in parenting stress with engagement in interventions, as well as improved cortisol regulation one year post-intervention. These results provide early support for the ability of such programs to have long-term effectiveness for hormonal regulation, and associated health outcomes. However, given the paucity of research on stress regulation, more evidence is needed to support program influence on physiological functioning among high-risk parents.

Literature in maternal and child health has focused more expansively on child-level cortisol outcomes. For example, several studies have demonstrated varying, individual cortisol responses among children with exposure to risk factors for CM such as poverty (Lupie, King, Meaney, & McEwen, 2001) and maternal psychological symptomology (Bugental, Martorell, &

Barraza, 2003; Lupien, King, Meaney, & McEwen, 2000). Further, behavioral parenting research has examined and shown promise for improved cortisol stress responses to behavioral parenting programs at the child level (Bugental, Schwartz, & Lynch, 2010; Cicchetti, Rogosch, Toth, & Sturge-Apple, 2011; Dozier et al., 2006; Dozier, Peloso, Lewis, Laurenceau, & Levine, 2008; Fisher, Stoolmiller, Gunnar, & Burraston, 2007). For example, Fisher and Stoolmiller (2008) assessed self-reported stress among caregivers in relation to cortisol activity among a group of 117 caregiver-foster children preschoolers (ages 3-6) dyads participating in a randomized controlled trial. Caregiver-child dyads were assigned to receive a therapeutic intervention for caregivers, or a regular foster care services. Findings from this study illustrated that while foster parents assigned to the intervention showed reductions in self-reported stress, reductions in self-reported parental stress mediated the intervention effects on impaired cortisol activity among children. Thus, evidence supports that impaired cortisol levels among children may be modifiable with caregiver engagement in parenting interventions.

Fisher and Stoolmiller (2008) further suggest that parental uptake of skills in cognitive home visiting interventions can mediate the relationship between engagement in an intervention and child cortisol levels, in which positive parenting outcomes lead to improvements in child cortisol levels and child cognitive functioning. These results therefore, suggest that behavioral interventions that improve parental behaviors can reduce perceived parental stress and can therefore be effective strategies to physiological functioning among children. However, such studies are limited by the exclusion of cortisol levels among parents to validate perceived reductions in parental stress at a physiological level, as seen among children. While there is a clear role for cortisol in HPA axis regulation, further examination is also needed on other hormones and factors secreted during stress by the sympathetic nervous system (SNS), the

division of the autonomic nervous system that responds to stress. Thus, additional biological markers should be measured in efforts to obtain comprehensive, individual stress profiles.

Alpha-amylase. Salivary alpha-amylase (sAA), a salivary enzyme secreted by the parotid gland, has received increased attention as a biological stress indicator for clinically significant dysregulation of the SNS (Nater et al., 2006; Nater & Rohleder, 2009) leading to anxiety-related symptomology (Takai et al., 2004). sAA levels increase in response to acute psychosocial stress (Thoma, Kirschbaum, Wolf, & Rohleder, 2012) and in response to physical stress stimuli such as exercise, temperature, and psychological conditions (Bosch et al., 1996; Chatterton Jr, Vogelsong, Lu, & Hudgens, 1997; Chatterton, Vogelsong, Lu, Ellman, & Hudgens, 1996; Skosnik, Chatterton, Swisher, & Park, 2000; Takai et al., 2004). Growing evidence suggests increases in sAA may also be associated with chronic stress (Vigil, Geary, Granger, & Flinn, 2010; Vineetha, Pai, Vengal, Gopalakrishna, & Narayanakurup, 2014).

sAA Intervention Response. Evidence suggests that sAA is amenable to change following stress reduction programs and interventions. For example, Limm et al. (2010) conducted a randomized controlled trial among 174 males within the industrial workplace setting to examine changes in work stress in response to group-psychotherapy. Researchers found that participants who participated in the stress management intervention experienced greater decreases in sAA levels in comparison to participants of the waitlist control one year post-intervention (Limm et al., 2010). Similar reductions in sAA are noted among other randomized control trials among cancer survivors undergoing mindfulness training (Lipschitz, Kuhn, Kinney, Donaldson, & Nakamura, 2013), and among healthy couples going through an emotional-support intervention (Holt-Lunstad, Birmingham, & Light, 2008). However, no known studies within maternal and child health have examined sAA levels or responses to intervention.

DHEA. Research on other steroid hormones such as adrenal cortex produced DHEA, the precursor for sex hormones such as testosterone and estradiol, and psychosocial stress suggests an association between chronic stress experiences and impaired DHEA plasma levels. For example, Yehuda et al. (2006) conducted a cross-sectional study to assess the relationship between chronic post-traumatic stress disorder (PTSD) as a result of trauma exposure, and DHEA levels among a sample of 40 male veterans. Researchers found that increased DHEA levels were significantly associated with a diagnosis for current or lifetime PTSD. Furthermore, positive perceptions of coping and lower reports of symptom severity in the past month were predictive of DHEA levels among this group. Yehuda and colleagues describe that changes in DHEA levels may thus, play a role in degree of psychological recovery (Yehuda et al., 2006).

However, research on DHEA levels is limited, and has not been extended into the parenting literature. No known studies have examined DHEA outcomes in response to parenting interventions. Yet, evidence suggests that DHEA levels are modifiable with the introduction of psychological treatment. In a pre-post quasi-experimental study by Olf et al. (2007), researchers examined changes in several stress hormones including DHEA and cortisol, among 21 participants diagnosed with PTSD in response to 16 weekly sessions of brief eclectic psychotherapy. Significant improvements in DHEA and cortisol were found among participants responding positively to therapy (i.e., improvements in reported symptomology). Such findings provide evidence on the use of behavioral techniques in changing biological correlates of stress.

Purpose of Research

Given the limited research on biological correlates of stress in the parenting literature, there is a greater need to understand the relationship between perceived parental stress and

physiological correlates of stress known to have long-term impact on psychological disorders as well as chronic and infectious disease among adults. Research is therefore, needed to elucidate the impact of evidence-based practices, known to reduce parental stress, on known physiological biomarkers of stress. Furthermore, inclusion of novel stress measures will elucidate whether outcomes typically measured by such programs extend beyond parenting constructs; in this manner, inclusion biological measures may illustrate the potential of behavioral parenting programs to influence parent well-being.

Thus, this multidisciplinary study from the fields of neuroscience and public health aimed to evaluate ontogenic level factors, specifically psychophysiological profiles and responses, among parents engaged in an evidence-based CM public health intervention, SafeCare®. The objectives of this study were to use steroid stress hormones and a stress salivary enzyme to understand physiological stress among a high-risk parent population, and also in comparison to perceived, self-reported levels of stress by: (1) Recruiting 18 mothers at risk of abuse and neglect, based on risk factors identified in research, who received the PCI module of SafeCare® (2) Conducting repetitive, within subject assessments that included measures of parent stress and behavior (self-report), and physiological markers for cortisol, DHEA and sAA with the objective of comparing self-reported levels of stress to biomarker assessments.

Aims and Hypotheses. Based on the existing literature, the aims of the study were as follows:

- 1) To test the hypothesis that mothers who report higher levels of self-reported stress and mental health symptomology will have impaired steroid hormone (i.e., cortisol, DHEA) and sAA levels.

- 2) To test the hypothesis that parents who complete SafeCare® will show improvements in hormone production and sAA, as well as self-reported parental stress and mental health symptomology. Improvements were defined as positive changes towards mean values within the standard range of biomarkers.
 - a. To further test that parents who exhibit impaired biomarker levels (i.e., levels outside of standard physiological range) and who complete SafeCare® will show normalization following the intervention. Normalization was defined as positive changes from impaired biomarker levels to levels within standard reported ranges.
- 3) To test the hypothesis that parents will experience increases in acute stress (cortisol) in the presence of a home-visitor at session 3, mid-way through the intervention.
- 4) To assess the participants' willingness to provide physiological measures (i.e., salivary and hair samples) in a research project.
- 5) To examine maternal perceptions of general and parental stress.

Chapter 3.

METHODS

The Georgia State University's Institutional Review Board and Institutional Biosafety Committee approved this study.

Study Participants

Participants in this study included a convenience sample of eighteen African American mothers between ages 18 and 40 years ($M= 27.03$, $SD=6.66$). All participants were included in appropriate baseline descriptive analyses. Approximately half (56.5%) of participants had a high school education or less and were predominantly of low socioeconomic status, with 60% reporting annual household incomes of less than \$20,000. Approximately 44% of participants reported having one biological child. Sixty-one percent stated that at least three children were residing in their household. See Table 1 for a full list of demographics among participants. Ten participants completed the intervention and follow-up measures.

Table 1

Demographic Characteristics among Non-Completer and Completer Participants at Baseline

	All Participants (<i>n</i> =18)		Non Completers (<i>n</i> =8)		Completers (<i>n</i> =10)		Included Participants (<i>n</i> =14)		Excluded Participants (<i>n</i> =4)	
	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)
Age (years)	27.03 (6.66)		28.17 (3.57)		26.23 (8.28)		25.54 (5.22)		31.87 (9.29)	
Race (Black)		18 (100)		8 (100)		10 (100)		14 (100)		4 (100)
Education Level										
<High School		4 (23.50)		2 (28.6)		2 (20)		4 (30.80)		-
High School		5 (29.40)		2 (28.6)		3 (30)		3 (21.10)		2 (50)
Some College		6 (35.30)		3 (42.9)		3 (30)		5 (38.5)		1 (25)
Vocational		1 (5.90)		-		1 (10)		-		1 (25)
Graduate School		1 (50.9)		-		1 (10)		1 (7.70)		
Marital Status										

	All Participants (<i>n</i> =18)		Non Completers (<i>n</i> =8)		Completers (<i>n</i> =10)		Included Participants (<i>n</i> =14)		Excluded Participants (<i>n</i> =4)	
	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)
Single		9 (50)		5 (62.5)		4 (40)		9 (57.10)		1 (25)
Married		2 (11.10)		1 (12.5)		1 (10)		-		2 (50)
Partner		6 (33.30)		1 (12.5)		5 (50)		5 (35.70)		1 (25)
Other		1 (5.60)		1 (12.5)				1 (7.10)		-
Working Status										
Yes		3 (18.80)		1 (16.7)		8 (20)		3 (25)		-
No		13 (81.30)		5 (83.3)		8 (80)		9 (75)		4 (100)
Annual HH Income										
<\$15,000		8 (53.30)		4 (57.1)		5 (50)		7 (63.60)		1 (25)
\$15,000-30,000		5 (33.40)		1 (14.3)		5 (50)		3 (27.30)		2 (50)

	All Participants (<i>n</i> =18)		Non Completers (<i>n</i> =8)		Completers (<i>n</i> =10)		Included Participants (<i>n</i> =14)		Excluded Participants (<i>n</i> =4)	
	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)	<i>M</i> (<i>SD</i>)	<i>n</i> (%)
\$30,000+		2 (13.3)		2 (28.6)		-		1 (9.10)		1 (25)
No. of Birth Children	2.38 (1.66)		3.12 (1.96)		1.8 (1.23)		2.29 (1.78)		3.3 (1.15)	
No. Children in HH	2.77 (1.31)		2.75 (1.49)		2.8 (1.23)		2.78 (1.37)		3.33 (.58)	
No. Adults in HH	1.72 (.89)		1.35 (.517)		2 (1.05)		1.64 (.74)		2.33 (1.53)	

Recruitment

Participants were recruited through an established community partner with Georgia State University, Hughes Spalding Children's Hospital. Hughes Spalding is located in downtown Atlanta and provides comprehensive pediatric care and community resource referrals for families at-risk. Hughes Spalding has a long-standing relationship with the National SafeCare® Training and Research Center (NSTRC), located within the School of Public Health at Georgia State University, and has provided family referrals for three federally funded studies. NSTRC is the national purveyor for SafeCare® program, and conducts national and international training and research related to SafeCare®.

Inclusion/ Exclusion Criteria

Participants in this study were restricted to mothers' ≥ 18 years of age. Mothers included both biological and custodial caregivers, with children between 0-5 years of age. Mothers were also required to have custodial rights with the target child for the SafeCare® intervention. Exclusion criteria included participant's acknowledgement of a diagnosis of biologic or medical conditions, and/or consistent use of steroid medications likely to interfere with hormone measures at time of recruitment.

Intervention Procedures

Eligible and consenting participants received the PCI module of SafeCare®. The intervention included six home visiting sessions with a SafeCare® home visitor. The PCI intervention is a highly structured behavioral parenting program that is delivered with fidelity to at-risk parents and targets parent risk factors related to perpetration of physical abuse and neglect. The intervention focused on building a positive relationship between parent and child,

and reducing problematic child behavior. Home visitors assessed parenting skills and activities that presented most challenges for mothers when interacting with their children. Over the intervention, home visitors provided instruction and model activities that mothers could practice at home. Parents learned to structure activities with their children, while reducing problematic behavior and reinforcing positive behavior. At the end of all sessions, home visitors reassessed parenting skills. Parents received \$10 per session.

Assessment Procedures

Trained research assistants scheduled and conducted two in-home assessments at baseline and post-intervention at week 8. Research assistants also collected biomarker data at these in-home assessments and obtained biomarker data mid-intervention (described later). Participants were reimbursed \$50 for their time for each assessment battery, and \$20 for biomarker collection mid-intervention. Additionally, at the end of the baseline assessment, participants were asked to complete a qualitative interview to discuss: their opinions about the feasibility on collection of physiological measures, how to make this process more efficient in future research trials, and their perceptions of general and parental stress.

Self-Report Measures. Prior to and following the intervention (i.e., baseline and post-intervention assessments), parents completed a battery of self-report measures (Table 2).

Table 2

Self-Reported Measures Administered at Pre- and Post-Intervention Assessments

Outcome	Measure	# Items
Control variables	Demographic Information Form: <i>constructed for the study and used to collect basic demographic information on all participants</i>	
Self-reported Mental Health Symptomology and Parental Stress	Posttraumatic Stress Diagnostics Scale (PDS) (Foa, Cashman, Jaycox, & Perry, 1997): <i>to evaluate trauma exposure and related trauma symptomology</i>	53
	Brief Symptom Inventory (BSI) (Derogatis & Melisaratos, 1983): <i>to evaluate mental health symptomology (depression, anxiety, global distress symptomology)</i>	49
	Parenting Stress Inventory (PSI-SF) (R. Abidin, .R., 1995): <i>to examine parent stress.</i>	36

Demographic Information Form. Participants provided information on gender, age, race, marital status, educational attainment, yearly income, employment status, and household size.

Current Perceived Stress. Parents were asked to rate their stress levels at the time of assessment, using a 6-point Likert scale item ranging from “not at all stressed” to “extremely stressed”.

Parenting Stress Inventory (PSI-SF) (Abidin, 1990). The PSI-SF is a 36-item self-report measure used to assess parenting stress among parents with children between 3 months to 10 years of age. The PSI-SF consists of three subscales (parental distress, parent-child dysfunctional interaction, difficult child characteristics), each consisting of 12 items. Caretakers were asked to score each item using a 5-point Likert scale ranging from 1 (“strongly agree”) to 5 (“strongly disagree”). A summary score was used to determine total stress among caretakers, in which

higher summary scores would indicate higher levels of perceived stress (potential scores ranging from 0-180). Summary scores at assessments 1 and 2 ranged from 48-141, and 42-117, respectively. The PSI demonstrated good internal consistency for this sample (Assessment 1, $\alpha = .94$; Assessment 2 $\alpha = .95$).

Brief Symptom Inventory (BSI) (Derogatis & Melisaratos, 1983). The BSI is a 53-item instrument with nine symptom-specific subscales (somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoia, psychoticism). Mothers rated each item a five-point Likert scale ranging from 0 (“not at all”) to 4 (“extremely”). All items were summed to assess global distress symptomology among participants. These items demonstrated excellent internal consistency (Assessment 1, $\alpha = .95$; Assessment 2, $\alpha = .95$). The 6-item depression subscale was used to assess maternal depressive symptomology. These items demonstrated adequate internal consistency (Assessment 1, $\alpha = .81$; Assessment 2, $\alpha = .81$). The 6-item anxiety subscale was used to assess maternal anxiety symptomology. These items demonstrated adequate internal consistency at Assessment 1, but poor internal consistency at Assessment 2 ($\alpha = .81$; Assessment 2, $\alpha = .34$).

Posttraumatic Diagnostic Scale (PDS) (Foa et al., 1997). The PDS assesses PTS symptomology in accordance with the *Diagnostic and Statistical Manual of Mental Disorders 4th Edition-Text Revision* (American Psychiatric Association, 2000). The PDS has demonstrated good psychometric properties (Foa et al., 1997). Thirteen participants completed the PDS measure.

Trauma Screen. Part I of the PDS consists of 12 dichotomous items and one explanatory item (“Which event bothered you the most?”). Items were used to assess exposure to stressful and traumatic life events among participants (e.g., witnessing a serious accident, fire or

explosion). Scores were summed to obtain a summary score (potential scores ranging from 0 to 12), with higher scores indicating greater levels of exposure to traumatic events.

PTS symptomology. Frequency of maternal PTS symptomology was assessed using Parts III and IV of the PDS. For each item measuring PTS symptoms, mothers responded from 0 (“not at all or only one time”) to 3 (“5 or more time a week/almost always”) according to self-reported frequency of problematic occurrence during the past month. Scores for Parts III and IV were summed to yield an overall summary score of PTS symptoms among mothers, with potential scores ranging from 0-51, in which higher scores indicate increased severity in PTS symptomology. Clinical symptom severity ranges were identified (i.e., mild = 1-10; moderate = 11-20; moderate-severe = 21-35; severe = 36 and greater) (McCarthy, 2008). The PDS demonstrated adequate internal consistency for this sample ($\alpha = .89$).

Functional Impairment. Part IV of the PDS was used to assess levels of daily life impairment among caregivers as related to their trauma exposure. Nine items were summed to obtain a summary score (potentially ranging from 0 to 9). Past research examining functional impairment has utilized the following cut-off ranges: no impairment=0; mild=1-2; moderate=3-6; severe=7-9 (Howgego et al., 2005).

Physiological Measures. Additionally, the physiological measures, described in Table 3 below, were taken as the mother participants progressed through the intervention. Research assistants reminded parents the day prior to, and the day of assessments to refrain from eating or drinking within 2 hours of their assessment.

Table 3

Physiological Samples Schedule

Baseline/ Pre-Intervention	Session 3 of Intervention	Post-Intervention
Week 1	Week 4	Week 8
Cortisol (saliva and hair)	Cortisol (3 saliva samples):	Cortisol (saliva and hair)
DHEA	-Start of session -10 minutes after session -20 minutes after session	DHEA
sAA		sAA

Salivary Biomarker Collection. Cortisol, sAA and DHEA were obtained from saliva samples at baseline, and post-intervention at week 8. At week 4 of the intervention only (Session 3), participants were asked to provide three additional salivary samples to assess acute cortisol at the beginning of the intervention session, 10 minutes after the session ends and 20 minutes after. Cortisol is known to show in saliva after 15 minutes from presentation of a stimulus (Clemens Kirschbaum & Hellhammer, 1994). Thus, the sample taken prior to the session was a proxy measure of anticipation to the challenge. Samples collected after the session were interpreted as responses to the intervention session.

Salivary samples for cortisol and sAA were collected using a Salivette® procedure. Protocol required that participants chew a roll-shaped salivary swab for approximately 1-2 minutes. Once the roll was saturated, participants then dropped the swab directly from their mouths into a specialized vial that was securely closed with a stopper. Research assistants attempted to collect all salivary samples consistently at the same time (1-3:30pm), when cortisol levels appear stable. However, at assessment 1, 2 participants provided saliva samples between 3:30-4pm. At assessment 2, 1 participant provided saliva at between 3:30-3:50pm.

DHEA was collected using passive drool collection kits. Participants were asked to drool into cut plastic straws leading into specialized vials for saliva collection that were securely closed. At least 1 ml of saliva was required for analyses. Studies have indicated changes in DHEA levels among healthy adults in as little as four weeks (McCarty, Barrios-Choplin, Rozman, Atkinson, & Watkins, 1998).

Research assistants collected the samples and labeled all vials with the times and dates that samples were collected. Samples were transported to the Georgia State Neuroscience Institute, at room temperature, where biomarkers were extracted from the saliva samples to concentrations (See Physiological Measures Laboratory Methods Section for in-depth description).

Hair. Hair samples were collected to measure cortisol as an indicator of chronic stress and to determine the stress profile for the previous 2-3 month period. Hair is considered a reliable and stable measure of cortisol production (Cirimele, Kintz, Dumestre, Gouille, & Ludes, 2000; Raul, Cirimele, Ludes, & Kintz, 2004), and chronic stress (Russell, Koren, Rieder, & Van Uum, 2012) Hair growth is assumed to occur at a rate of 0.6-1.4 cm/ month (Saitoh, 1969). Therefore, hair samples of 2-3 cm may provide a 2-3 month estimation to chronic stress exposure. Hair samples were cut with clean scissors from the posterior vertex of the head, and as close to the scalp as possible. This region has been shown to have the lowest coefficient of variation for cortisol levels in comparison to other areas (Sauvé, Koren, Walsh, Tokmakejian, & Van Uum, 2007). Tape was used to indicate the hair end taken from the scalp. Hair samples from each individual were placed in labeled and sealable plastic baggies or envelopes at room temperature. Samples were taken to the Carruth Lab at Georgia State University where hair

cortisol was extracted and measured (See Physiological Measures Laboratory Methods Section for in-depth description).

Qualitative Interview. At the end of the first assessment session, parents were asked to give opinions about the physiological measure assessments. Parents were asked to discuss how burdensome these measures were to complete and provide recommendations for making the data collection more efficient in future project. In addition, parents were also asked to comment on personal definitions of general stress and parental stress. Interviews took approximately 10-15 minutes to complete.

Physiological Measures Laboratory Methods

Participants were classified as “abnormal” if salivary cortisol and sAA levels fell outside of standard ranges on biological outcomes (e.g., salivary cortisol levels < 0.053 µg/mL; sAA levels: <40.0 u/mL, > 94.2 u/mL as determined by Salimetrics).

Salivary Cortisol. The following procedures were for 50 uL samples following the Salimetrics LLC (Carlsbad, CA) kit directions. First pH was determined for the assay diluent samples: acidic samples turn the pH indicator yellow and alkaline samples turn the indicator purple. Samples outside the <3.5 or >9.0 pH range were artificially increased or decreased. All Salivette® samples were vortexed and centrifuged at 1500g (3000 rpm) at room temperature before assaying. All readings were made at 450 nM. All reagents were allowed to come to room temperature before use. Mixed samples were incubated at room temperature for 45 minutes before being added to the assay plate wells. The template included 6 standards: µg/dL: 3.0, 1.0, 0.333, .0111, .037, and .012. High and low cortisol known samples were used as controls and non-specific binding (NSB) wells and the no anti-cortisol antibody wells were used as blanks.

Procedure. A 1X wash buffer was prepared by diluting wash buffer 10 fold with room temperature distilled water (100 mL of 10X to 900 mL of distilled water. Twenty-four milliliters of assay diluent was placed into disposable tube for conjugate dilution, and was subsequently mixed. Twenty-five microliters of standards, controls, and unknowns were pipetted into appropriate wells, in duplicate. Twenty-five microliters of assay diluent were pipetted into 2 wells to serve as ZERO wells. Twenty-five microliters of assay diluent were pipetted into each NBS well. A 1:1600 dilution of the conjugate was prepared: (15 uL of the conjugate to 24 mL of assay diluent prepared in first step) and mixed immediately. Two hundred microliters were pipetted into each well using a multichannel pipette. The plate was then mixed on a rotator for 5

minutes at 500 rpm, and was then incubated for an additional 55 minutes at room temperature. The plate was then washed 4 times with 1 X Buffer by pipetting 300 uL of the wash buffer into each well and then discarding the liquid over the sink. The plate was blotted thoroughly on paper towels before turning right side up. TMB solution (200 mL) was added to each well, and then mixed on the plate rotator for 5 minutes at 500 rpm. The plate was then incubated in the dark at room temperature for an additional 25 minutes. Fifty microliters of STOP solution were added. The plate was then placed on the rotator for 3 minutes at 500 rpm. The bottom of plate was wiped with water-moistened lint free cloth and subsequently dried. Plate readings were done at 450 nm (Program in iMark Analysis), and within 10 minutes of adding the STOP solution.

SAA. sAA samples were analyzed using the EIA – Liquid Phase alpha-Amylase Saliva Assay, IBL International kit # RE80111. Ten microliters of each sample of saliva collected via salivettes were used and diluted in buffer and centrifuged at 3000 x g room temperature for 10 minutes. All components were allowed to reach room temperature before gently swirling to mix.

Sample preparation: Sample buffers were diluted as a 1:10 dilution. This dilution buffer (DB) was used for the standards (4 mL), controls (6.4 mL) and samples (3 mL per sample) dilutions. Next, the controls were mixed with 200 µL of dilution buffer and were left to stand at room temperature for 15 minutes. To make stock standards, the lyophilized stock was reconstituted by adding 200 µL diluted sample. Five-milliliter conical tubes were used to pre-dilute the samples and controls. Either 10 µL sample or 10 µL reconstituted control was then added to the 3 mL diluted buffer and was mixed well.

sAA measurement: Ten microliters of standards, controls, and samples were pipetted into respective wells, followed by 200 μ L of Substrate Solution into each well. Mixtures were mixed on plate shaker at 300 rpm gently to avoid creating air bubbles. Mixtures were then incubated for 3 minutes at room temperature. The first measurement was taken after 3 minutes at 405 nm optical density (reference was 600-690). Mixtures were incubated for an additional 5 minutes at room temperature, followed by a second measurement at 405 nm.

Hair Cortisol. Preparation of hair samples: Hair samples were prepared following a standard published protocol (Sauvé et al., 2007; Yamada et al., 2007). A minimum of 10mg of hair was weighed and finely cut into small pieces using sharp surgical scissors and then placed into a disposable glass scintillation vial containing 1mL of methanol. The vials were sealed and incubated for 24 hours at room temperature on a rotating shaker (Lab-Line Maxi rotor). After incubation, the supernatant was collected and put into disposable glass 13 x 100 mm culture tubes (Fisher) and evaporated in a dry bath (Thermolyne Dri-Bath) using a sample concentrator until dry. The samples were then re-suspended in 150-250 μ L of pH 8.0 phosphate buffered saline (PBS). Samples were mixed using a vortex for 1.5 minutes until well mixed. The cortisol in each sample was measured using Salimetrics LLC Salivary Cortisol kit (Carlsbad, CA) as described above for saliva and following manufacturer's directions with the reagents provided.

Participants were classified as "chronically stressed" if hair cortisol levels were above the published range considered normal for adult human hair (Sauvé et al., 2007) of 17.7 – 153.2 pg/mg.

DHEA (Passive Drool). All procedures were conducted following the standard Salimetrics LLC (Carlsbad, CA) DHEA ELISA kit. All reagents were allowed to come to room

temperature before use per manufacturer's instructions. All passive drool samples were vortexed and centrifuged at 1500 g (3000 RPM) before assaying. The plate template is below and all readings were made at 450 nM.

The wash buffer (100 mL of 10x Wash buffer + 900 mL room temperature dH₂O water) was prepared immediately prior to diluting the samples. Tips were changed between each dilution when preparing the serial dilutions of the standard. Eighteen milliliters of the assay diluent was pipetted into a disposable tube and set aside for the enzyme conjugant step.

Procedure. Fifty microliters of standards, controls and unknowns were pipetted into appropriate wells in duplicate. Fifty microliters of assay diluent were pipetted into 2 wells to serve as the ZERO wells. Fifty microliters of assay diluent were pipetted into each NSB well (blanks). The enzyme conjugate was then diluted at 1:1500 by adding the conjugate to the assay diluent and pipetted into each well.

An adhesive cover was placed over the plate, which was then mixed on a rotator for 5 minutes at 500 rpm. The plate was then incubated at room temperature for a total of 3 hours. The plate was then washed 4 times with Wash Buffer. Three hundred microliters were pipetted with a multichannel pipette into each well. Liquid was flipped into a sink. After each wash, the plate was thoroughly blotted on paper towels before turning upright.

Two hundred microliters of TMB solution was added to each well with a multichannel pipette. The plate was then mixed on a rotator for 5 minutes at 500 rpm and incubated in the dark at room temperature for an additional 25 minutes. STOP solution (50 μ L) was added to the plate with a multichannel pipette. The plate was again mixed on a rotator for 3 minutes at 500 rpm. Efforts were made to ensure all wells turned yellow. Mixing was resumed if any green color

remained. The bottom of the plate was wiped with a water- moistened lint free cloth and subsequently dried. Plates were read in a plate reader at 450 nm within 10 minutes of adding STOP solution.

Data Analysis Plan

Given the exploratory nature of this study, quantitative and qualitative data were analyzed using an Embedded Design mixed-methods approach (Creswell, Plano Clark, Gutmann, & Hanson, 2003). Qualitative data collection was embedded within a primarily quantitative study in order to answer independent research questions that could not be answered through quantitative data.

Quantitative Analyses. All quantitative analyses were conducted with IBM SPSS Statistics Software version 20 (IBM Corporation, 2011). Means, frequencies and clinical significance were examined among applicable study items for all participants (see Tables 1,4). Four parents were excluded from relevant data analyses because of invalid data (3 identified outliers for salivary cortisol and/or sAA levels) and missing data (1 incomplete data on hormone levels). Descriptive data were obtained to examine differences between completers and non-completers, and excluded participants.

Bivariate correlation analyses were conducted between all biomarkers and self-report variables for participants at baseline and follow-up. Bivariate correlations were also conducted among a sub-sample of participants at baseline, who reported exposure to at least one traumatic event ($n=8$). Correlations were not conducted at follow-up for these participants with trauma exposure, given sample size restrictions ($n=5$).

Paired *t*-tests were conducted among participants who completed the intervention to determine significant, within subject differences in biomarker levels, self-reported stress levels, and mental health symptomology from baseline to follow-up. Seven of ten completing participants with complete data were included in these analyses.

Qualitative Data Analyses. Data for qualitative analyses included transcripts of audio-recorded semi-structured interviews among participants. Line-by-line coding and thematic analysis were used to analyze all transcripts. One graduate research assistant read and openly coded transcripts. Derived codes from these transcripts were compared for consistency and overlap. Codes were grouped into themes. A codebook integrating all themes and associated statements was created.

Chapter 4.

RESULTS

Descriptive Statistics

Baseline Self-Report Measures. (See Table 4)

Parenting Stress. Data from the PSI were used to assess levels of perceived parenting stress. The mean total parenting stress score among all participants at baseline was 78.06 ($SD=26.35$). At baseline, those parents who were non-completers reported higher levels of parenting stress in comparison to those who went on to complete SafeCare®, $M= 82.0$ ($SD=29.0$). Approximately 19% ($n=3$) of participants met clinically significant levels of stress (i.e., raw score >90) (R. R. Abidin, 1995) at baseline. Two of these participants completed the intervention.

Mental Health Symptomology. Data from the BSI at were used to assess depressive, anxiety and global distress symptomology and clinical significance for these symptoms (Table 2). Data from the PDS were used to assess the severity of PTS symptomology among 13 participants (this measure was added to the protocol later in the study).

On average at baseline, clinical levels of global distress symptomology were observed among all participant groups. Among all participants, 36%, 21% and 57% met clinically significant levels of depressive, anxiety, and global distress symptomology, respectively. Among completers at baseline, 20%, 10% and 40% met clinically significant levels of depressive, anxiety and global distress symptomology respectively. Symptomology levels were higher on average among non-completers in comparison to completers.

On average, most mothers (77% all participants, 70% completers) reported experiencing at least one traumatic event. Most commonly reported events included sexual assault by someone known, (40%), and non-sexual assault by someone known (30%). The mean level of PTS symptoms was $M=22.56$ ($SD=11.71$), indicating moderate-severe levels of PTS symptomology. Two parents (1 completer) exhibited severe clinical symptom levels. Participants on average reported mild levels of functional impairment ($M=2.60$, $SD=2.41$). Forty percent of trauma-exposed parents reported moderate to severe levels of functional impairment.

Biomarker Measures. (See Table 4)

Cortisol Levels. Baseline Salivary Acute Cortisol. Among all participants at baseline, mean salivary cortisol levels were $.08 \mu\text{g/dL}$ ($SD= .046$). Salivary cortisol levels were higher on average among non-completers in comparison to completers at baseline. Among completers at baseline, mean cortisol levels were $.076 \mu\text{g /dL}$ ($SD=.052$).

Chronic Cortisol. Hair samples were used to obtain 3-month estimates of chronic or cumulative cortisol levels at baseline and follow-up assessments. Among all participants at baseline, mean chronic cortisol levels were $84.86 \mu\text{g /ml}$ ($SD= 35.93$). Among completers at baseline, mean cortisol levels were 83.23pg /mL ($SD=49.70$).

sAA levels. Saliva taken from Salivettes were used to extract sAA. Among all participants at baseline, mean sAA levels were above the upper range for normality ($> 94.2 \text{ u/mL}$), $M=102.22 \text{ u/mL}$ ($SD= .046$). On average, non-completers exhibited sAA levels within the normal range at baseline. Among completers at baseline, mean sAA levels were above normal, $M= 111.70 \text{ u/mL}$ ($SD=51.11$).

DHEA levels. DHEA levels were obtained from participants' passive drool samples. Among all participants at baseline, mean DHEA levels were 192.69 $\mu\text{g/mL}$ ($SD=190.74$). DHEA levels were higher among non-completers on average, in comparison to completers at baseline. Mean DHEA levels among completers at baseline were 204 $\mu\text{g/mL}$ ($SD=161.15$).

Table 4

Descriptive Information for Study Variables among All Participants

Variables (possible score range)	Baseline Participants <i>M (SD)</i>	Non-Completers Baseline <i>M (SD)</i>	Completers Baseline <i>M (SD)</i>
Parenting Stress (0-180)	78.06 (26.35)	82.0 (29.0)	75.0 (25.10)
Global Distress Symptomology (0-5)*	.93 (.65)	1.18 (.60)	.83 (.67)
Depressive Symptomology (0-5)*	.79 (.85)	1.13 (.77)	.67(.88)
Anxiety Symptomology (0-5)*	.50 (.63)	.94 (.70)	.32 (.53)
Trauma Experiences (0- 12)**	1.69 (1.60)	1.67 (1.15)	1.70 (1.76)
PTS Symptomology (0- 51)**	22.56 (11.70)	23.67 (12.34)	22.0 (12.54)
Functional Impairment (0- 9)**	2.60 (2.41)	3.00 (2.65)	2.43 (2.51)
Cortisol (saliva) $\mu\text{g/dL}\ddagger$.081 (.046)	.086 (.04)	.076 (.07)
Cortisol (hair) $\mu\text{g/mL}\ddagger$	84.85 (42.14)	86.76 (35.9)	83.23 (49.70)
sAA $\mu\text{g/mL}\ddagger$	102.22 (56.28)	92.73 (63.57)	111.69 (51.11)
DHEA $\mu\text{g/mL}\ddagger$	192.69 (190.78)	247.83 (257.53)	137.55 (73.79)

*Note. Average scores reported; **PDS data obtained from 13 participants (3 non-completers, 10 completers); \ddagger biomarker levels reflect non-excluded participants

The following is a discussion of the results, as they relate to prescribed hypotheses and aims of this study:

1) *To test the hypothesis that mothers who report higher levels of self-reported stress and mental health problems will have impaired steroid hormone levels (i.e., cortisol, DHEA) and alpha-amylase.*

Bivariate correlations between biomarker levels (salivary cortisol, hair cortisol, sAA, DHEA) and self-reported measure scores (parental stress, mental health symptomology: global distress, depressive and anxiety symptomology) were conducted among all participants at baseline (Table 5), completers at baseline (Table 6) and follow-up (see Table 7), and among participants experiencing at least 1 traumatic event (Table 8).

Baseline (Assessment 1)

Cortisol, sAA, DHEA: Demographics and Parental Stress.

Biomarkers. Among all participants, acute salivary cortisol was significantly and negatively correlated with sAA levels. No other significant correlations were noted between biomarker measures (see Table 5).

Parental Stress. Among all participants, acute salivary cortisol levels were significantly and negatively correlated with parental stress scores. sAA was significantly and positively correlated with parental stress scores. No significant correlations were found between DHEA levels and parental stress (see Table 5). Among completers at baseline (Table 6), acute salivary cortisol levels were trending towards a significant, negative correlation with parental stress scores. ($p=.056$). Although no significant correlations were found between sAA and parental stress scores at baseline, visual inspection of the data showed a positive, linear relationship between these variables (see Figure 1).

Cortisol, sAA, DHEA- Mental Health Symptomology. Among all participants (Table 5), acute cortisol levels were trending towards a significant, negative correlation with global distress symptomology ($p=.08$). No significant associations were found between biomarker levels and mental health symptomology among completers at baseline (see Table 6).

Correlations among Self-Report Measures. Among all participants, global distress symptomology was significantly and positively correlated with depressive and anxiety symptomology (Table 5). Among completers at baseline (see Table 6), perceived parental stress was significantly and positively correlated with global distress symptomology and depressive symptomology. Global distress symptomology was significantly and positively correlated with anxiety and trending towards a positive correlation with depressive symptomology among completers at baseline.

Follow-Up (Assessment 2) (see Table 7)

Cortisol, sAA, DHEA- Parental Stress. No significant relationships were observed among participant biomarker levels at Assessment 2. sAA levels were significantly and positively correlated with parental stress scores. No significant correlations were found between DHEA levels and parental stress.

Cortisol, sAA, DHEA- Mental Health Symptomology. sAA levels were significantly and positively associated with global distress and depressive symptomology at Assessment 2.

Correlations among Self-Report Measures. Perceived parental stress levels were significantly and positively correlated with global distress and depressive symptomology at follow-up. Global distress symptomology was significantly and positively correlated with both anxiety and depressive symptomology.

Table 5

Correlation Matrix for Biomarker Levels and Self-Report Measures among All Participants at Baseline (n=14)

Variable	1	2	3	4	5	6	7	8	9
1. Acute Cortisol levels-Saliva	-	-.14	-.61*	.13	-.70**	-.54‡	-.35	-.36	-.15
2. Chronic Cortisol Levels- Hair		-	-.31	-.23	.13	-.09	.11	.001	.50
3. sAA levels-Saliva			-	-.14	-.74**	-.26	-.07	-.10	-.41
4. DHEA levels				-	-.34	.24	.08	.37	-.01
5. PSI					-	.41	.44	-.07	.01
6. Global Distress Symptomology						-	.73*	.80**	.36
7. Depression Symptomology							-	.54	.48
8. Anxiety symptomology								-	.72‡
9. Stress Levels (0-5)									-

‡ .05 < p ≤ .08; * p < .05; ** p < .005

Table 6

Correlation Matrix for Biomarker Levels and Self-Report Measures for Completers at Baseline (n=7)

Variable	1	2	3	4	5	6	7	8	9
1. Acute Cortisol levels-Saliva	-	-.20	.44	.82*	-.74‡	-.50	-.41	-.16	.01
2. Chronic Cortisol levels-Hair		-	-.74‡	-.50	-.01	.07	.09	.30	.09
3. sAA			-	-.14	.66	.45	.43	-.06	-.41
4. DHEA levels				-	-.66	-.53	-.58	-.49	-.36
5. PSI					-	.91**	.87*	.23	.20
6. Global Distress Symptomology						-	.75‡	.79*	.42
7. Depression Symptomology							-	.46	.54
8. Anxiety symptomology								-	-.74‡
9. Stress Level (0-5)									-

‡ .05 < p < .06, * p < .05, ** p < .005

Table 7

Correlation Matrix for Biomarker Levels and Self-Report Measures at Follow-Up (n=7)

Variable	1	2	3	4	5	6	7	7	8
1. Acute Cortisol levels-Saliva	-	-.16	.19	-.48	.31	.46	.29	-.21	-.44
2. Chronic Cortisol levels-Hair		-	-.46	-.42	-.11	-.30	-.22	-.21	.09
3. sAA levels			-	-.08	.87*	.91*	.79*	.23	-.19
4. DHEA levels				-	-.35	-.22	-.31	-.48	.50
5. PSI					-	.96**	.87*	.42	-.05
6. Global Distress Symptomology						-	.89**	.33	.07
7. Depression Symptomology							-	.33	.23
8. Anxiety symptomology								-	-.17
9. Stress Level (0-5)									-

* $p < .05$, ** $p < .005$

Trauma Exposure

Cortisol, sAA, DHEA and Trauma. Trauma exposure data were collected from thirteen participants. Among participants who reported exposure to at least 1 traumatic event ($n=8$) (see Table 8), sAA was significantly and positively associated with PTS symptomology and functional impairment. sAA was also significantly and positively correlated with self-reported parental stress and trending towards a positive correlation with both salivary ($p=.05$) and chronic cortisol ($p=.07$). Salivary cortisol was significantly and negatively correlated with self-reported

stress. Both salivary cortisol and sAA were significantly and positively associated with global distress symptomology at baseline. PTS symptomology was significantly and positively correlated with functional impairment levels and trending on positive correlations with parental stress levels ($p=.078$).

Table 8

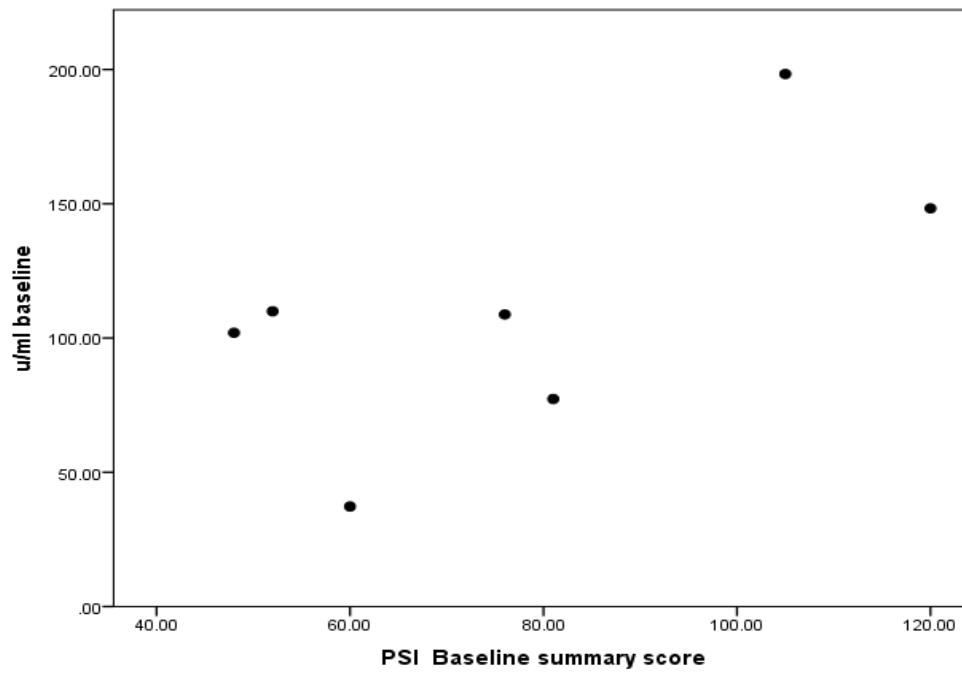
Correlation Matrix for Biomarker Levels and Participants with Trauma Exposure (n=8)

Variable	1	2	3	4	5	6	7	8	9	10	11
1. Acute Cortisol levels-Saliva	-	.28	-.70†	-.14	-.21	-.40	-.60	-.81*	-.48	-.20	-.21
2. Chronic Cortisol levels-Hair		-	-.72†	-.53	-.35	-.62	-.38	-.18	.08	.02	.74†
3. Baseline sAA levels			-	.17	.35	.76*	.76*	.75*	.47	.14	.32
4. Baseline DHEA levels				-	-.23	.13	-.12	-.16	-.19	-.43	-.01
5. Total number of trauma events					-	.67	.75*	.27	.39	.47	-.08
6. PTS symptomology						-	.93*	.70†	.59	.44	.38
7. Functional Impairment							-	.74*	.45	.38	.15
8. Parental Stress Scores								-	.68†	.55	.41
9. Global Distress Symptomology									-	.73*	.72*
10. Depression Symptomology										-	.21
11. Anxiety symptomology											-

† .05 < p < .08; * p < .05

Figure 1

Trends between sAA and Self-Report Stress at Baseline among Completers



2) *To test the hypothesis that parents who complete SafeCare® will show improvements in hormone production and alpha-amylase, as well as self-reported parent stress and mental health symptomology.*

a. To further examine that parents who exhibit impaired biomarker levels (i.e., levels outside of standard physiological range) and who complete SafeCare® will show normalization following the intervention

Pre-Post Intervention Differences (See Tables 9 & 10)

Within subject differences were assessed among the seven completers with physiological readings falling within 3 standard deviations of biomarker means. At Assessment 2 (Table 9), mean salivary cortisol levels increased to .18 µg /dL ($SD=.09$). Mean salivary cortisol levels for completers demonstrated an increase trend towards normative values from baseline ($t [6] =-2.09$, $p=.08$). Four participants exhibited hypocortisolic profiles at baseline ($<.053$ µg/dl). Three of these participants completed follow-up assessments. All completers exhibited normal salivary cortisol profiles at follow-up. Mean chronic cortisol levels (hair) showed minimal change at follow-up, $M= 83.08$ pg/mL ($SD=45.01$).

Mean sAA levels were within the normal range, $M= 90.75$ u/mL ($SD=61.40$) at Assessment 2. A trend in decreased sAA levels was observed ($t [6] =2.08$, $p=.08$). Six of seven completers at baseline participants exhibited sAA levels outside of the normal range at baseline ($1 \leq 40.0$ u/mL, $5 > 94.2$ u/mL). Three participants continued to exhibit levels outside the normal range ($1 \leq 40.0$ u/mL, $2 > 94.2$ u/mL). No significant changes or trends in DHEA were noted at follow-up.

Among all other variables measured at Assessment 2, including parental stress and mental health symptomology (global distress, depressive and anxiety symptomology), non-significant decreases were observed (see Table 10). Among completers at baseline included in analyses ($n=7$), the mean self-reported parental stress score was 77.43 ($SD=27.09$), and 73.43 ($SD=27.45$) at follow-up. No changes in clinically significant levels of parental stress were noted. At follow-up, average global distress symptomology levels decreased below clinical cutoffs. Similar decreases were observed for depressive symptomology ($M=.57$, $SD=.75$). Clinical levels of global distress and depression decreased from 57% ($n=4$) to 28% ($n=2$), and 43% ($n=3$) to 28% ($n=2$) participants, respectively, from baseline to follow-up. No participants reported clinical levels of anxiety at Assessment 2.

Table 9

Mean Baseline and Follow-up Cortisol, sAA and DHEA levels

	Baseline All ($n=14$) <i>M</i> (<i>SD</i>)	Baseline (Non- Completers) ($n=7$) <i>M</i> (<i>SD</i>)	Baseline (Completers) ($n=7$) <i>M</i> (<i>SD</i>)	Follow-Up ($n=7$) <i>M</i> (<i>SD</i>)	<i>t</i>	<i>df</i>	<i>p</i> -value
Cortisol (saliva) $\mu\text{g/dL}$.081 (.046)	.086 (.04)	.076 (.07)	.18 (.093)	-2.09	6	.08 [‡]
Cortisol (hair) pg/mL	84.85 (42.1)	84.78 (35.93)	83.23 (49.70)	83.74 (45.01)	-.13	6	.90
sAA u/mL	102.22 (56.28)	92.73 (63.57)	111.69 (51.11)	90.57 (61.40)	2.08	6	.08 [‡]
DHEA pg/mL	192.69 (190.78)	247.83 (257.53)	178.83 (73.79)	204.50 (161.15)	-.521	3	.64

[‡] Trending

Table 10

Differences in Self-Report Measures among Completers Following the Intervention (n=7)

Variables (possible score range)	Completers Baseline <i>M (SD)</i>	Completers Follow-Up <i>M (SD)</i>	<i>t</i>	<i>df</i>	<i>p</i> -value
Parenting Stress (0-180)	77.43 (27.09)	73.72 (27.45)	1.07	6	.33
Global Distress Symptomology (0-5)*	1.03 (.68)	.65 (.56)	1.82	6	.12
Depressive Symptomology (0-5)*	.90 (.88)	.57 (.75)	1.65	6	.15
Anxiety Symptomology (0-5)*	.45 (.59)	.33 (.36)	.70	6	.51

*Average score reported

3) *To test the hypothesis that parents will experience increases in acute stress in the presence of a home-visitor at Session 3, mid-way through the intervention.*

Session 3: Cortisol Levels

Eleven usable samples taken before session 3 began were available for analyses. However, six samples taken immediately after session 3 were usable for analyses. No usable data was available for the third time point taken at session 3. Thus, paired *t*-tests were used to examine within subject differences in acute cortisol levels at T1 and T2 for the six participants with complete data (Table 11).

At T1, average cortisol levels among participants was .21 µg /dL (*SD*=.11). A non-significant increase in cortisol at T2, was observed .25 µg /dL (*SD*=.30).

Table 11

Mean Salivary Cortisol Levels Before and During Session 3

	Session 3- T1 (<i>n</i> =6)	Session 3- T2 (<i>n</i> =6)	<i>t</i>	<i>df</i>	<i>p</i> -value
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)			
Salivary Cortisol Levels (µg /dL)	.21 (.11)	.25 (.30)	-.47	5	.66

Qualitative Analyses

4) *To assess the participants' willingness to provide physiological measures (i.e., salivary samples) in a research project.*

Ten of twelve conducted qualitative interviews were used for analyses. Excluded interviews were corrupted files and could not be used. Several themes were noted among participants with regards to feasibility of biomarker collection, as well as with interpretations of the meaning of parental stress. Data on project feasibility and biospecimens collection are presented first, followed by participant perceptions of general and parental stress.

Passive Drool. The majority of participants (77%) experienced discomfort when providing passive drool samples. When asked how they felt about providing this saliva sample, most participants commented on the awkwardness in using the straw and vial, for example:

“Um it was kind of weird but it was okay *laughs*; Well the spitting in the tube part was kind of weird and I felt like I was like a felon or something”

Another noted:

“Ew *laughs* I wouldn’t do it again mm. but the part was difficult... is making the saliva go in the tube because it at first it wasn’t going in...”

Two participants noted that they did not find the experience to be uncomfortable. No participants felt that providing drool was intrusive.

Salivette. More than half of participants experienced some discomfort in soaking the cotton roll under their tongue. When asked about the experience, several provided comments, such as:

“..It was uncomfortable though. Putting in your mouth and got to roll it around for 3 minutes I mean, it was long”

“That hurt a little bit”

However, some participants expressed a preference to the Salivette® over the passive drool sample methods.

Hair Sample. The majority of participants felt comfortable providing hair samples. For example, participants who were satisfied with providing hair samples made similar remarks, stating:

“I felt okay with it”

“That was, that was nothing, it was okay, I’m fine with that.”

Two participants expressed discomfort, and skepticism. When asked how she felt about providing a hair sample, one participant commented:

“Very upsetting, my hair got cut. I’m 30, my hair could be lost soon, I need all of that.”

Another participant stated:

“I’m tripping on the hair sample, because... what y’all need with the hair sample, that’s what I wanna know”

Participation in Research Collecting Biospecimens. When asked whether they would agree to participate in another study using similar biomarker collection methods, all participants agreed. All participants also noted that given the opportunity, they would also recommend this project to their friends.

Two participants expressed interest in understanding in the research process. One mother stated:

“Yes, I’m interested in knowing how, spitting in the tube, soaking the cotton swab, or cutting my hair, how.. what does it say about my stress level”

Another stating:

“Providing a sample of hair, I’m interested in knowing what the results are going to be”

Another participant noted:

“Mmhm, because there wasn’t really anything bad about it I mean if I could help I would.”

While participants agreed to participate in similar research in the future, two participants noted their hesitation in providing drool samples. For example, one mother said:

“ I mean I’m comfortable with cutting of hair and swabbing of cheek but spitting in the tube thing, I didn’t like that”.

When asked whether she believed her friends would be interested, one mother stated:

“Yes, the only thing that would gross them out is the same thing grossed me out, the saliva part”.

Improvements and Recommendation. Recommendations for the biomarker collection were directed at collecting passive drool samples. Some participants did recommend that the size of mouth piece be adjusted to improve the experience. For example, one participant stated:

“... maybe if the tube could be just a little bigger on the top, it would be okay...”

Another suggested:

“with the spitting in the tube thing, I think a straw would be better than the little thing that you had, so that it would come out, the straw would have been better.”

In addition, another participant noted difficulty building sufficient levels of saliva to insert into the tube:

“ um I felt at first it seemed like I couldn’t really get the spit inside but I, I just kinda built up a lil’ of spit to get it in there.”

One participant described discomfort in providing the sample in front of researchers.

5) To Examine Maternal Perceptions of General and Parental Stress

General Stress. When asked to provide an interpretation of what general stress means, approximately half of parents remarked stress to stem from feelings of frustration, and a lack of situational control.

For example, one parent described:

“Ooh, it means that a lot of things has hit that button to the point where you wanna scream kick and cuss, but you have to count to 10 and take deep breaths.”

Similarly, another participant indicated:

“To me it means like when you’re frustrated or stuff is hard for you to handle”

Another stated:

“The word stress to me means, like you’re not able to accomplish some of your goals.”

Another parent stated stress developing the pressure of multiple responsibilities:

“Um stress is I feel like it’s something that, um how can I get the word out, it’s basically like a lot on your um, like a lot on your chest, a lot on your plate like a heavy weight on your shoulder, it’s I mean, it’s basically what I feel like it is to me... trying to get a lot of things done at one time.”

Some participants (30%) shared that feelings stress can result from the worry over financial constraints and living arrangements. For example, when asked to provide her interpretation of stress, one parent stated:

“Well you can stress and worry about anything, your rent, you know not having food not being able to pay the bills so I think just worrying a lot can become very stressful.”

Another parent described the stress arising from her current living arrangements:

“I’m stressed out, like right now, I’m ready go move out... and I’m stayin’ with someone. I hate going through that, so that how the situation is I’m stressed cuz I gotta

have a roof over my children heads, so its making me stressed and when you being at somebody else spot it make u uncomfortable.”

Two parents simply described stress as being a negative emotion.

Parenting Stress. The majority of parents (80%) stated that parenting accounted for only a little stress in their daily lives. One parent commented that parenting accounted for all of her daily stress. When asked to describe types of parenting stresses that can be experienced, approximately half of participants commented on stress arising from fussy behaviors during activities such as feeding, bathing, dressing and sleeping:

For example, one parent described the stress arising during dressing:

“He doesn’t like putting on clothes, he likes to be naked. *laughs* And he gets fussy and he tries to move and leave... Because I’m just trying to put his clothes on and he doesn’t understand that it’s not so bad if you do it quick. It makes it harder because he takes up a lot of time when he’s moving around and being fussy.... but I don’t mind that, just sometimes it gets frustrating, nothing that I can’t handle though.”

Half of parents also described stress resulting from the difficulties in multitasking daily activities while caring for young children. When asked to elaborate on her experiences, one mother described:

“Yeah um trying to get things done, trying to cook or get ready to go somewhere,...um that’s another thing for me also, trying to get ready trying to get dressed to go somewhere and get her dressed, it’s like I have to wake up two hours early just to get to where im going. Yeah that um, that’s kind of stressful”

Two mothers described the advantages of having additional support to balance responsibilities.

For example, in response to being asked on the stresses of parenting, one mother stated:

“Just being able to get up and go without having to worry about finding a babysitter and stuff like that... Trying to find a babysitter, for your child and it might be a day where you can't find one and you trying to figure out what you're gonna do to get to work.”

She continued to state:

“Oh yeah um, well, when she cries I try to pick her up and make her bottle at the same time but sometimes I make her a container of milk and I just have to, to sometimes keep her on the bed and let her cry and hurry and make it and um it's really hard when I don't have my sister or someone to hold her while I go make that or while I try to eat something I have to just sit down and starve a little just to rock her to sleep or, I have to wait to eat basically, or yeah.”

Lastly, another mother described the role her older son, who was incarcerated at the time of the interview, in sharing caregiving responsibilities:

“I'm used to him [son] being here with to help me out, you know what I'm saying, wish I can handle my thing but he's a lotta help with my daughter, you feel me?”

Some parents described the stress that occurs when providing parental instructions. One parent stated:

“When you keep asking a child to do something over and over again and they just won’t do it... the things, that homework, because he knows how to do it, but he act like he don’t know nothing.”

Another parent simply stated:

“Having her, to keep telling her to stop, don’t do this don’t do that.”

Some parents described contextual factors as being part of parenting stress. When asked what could cause parenting stress, one parent commented:

“if they ain’t got no income and anything... financial, and they ain’t got no stable place to be at,...that’s what causes stress. That will cause stress when you got lot going on.”

Another parent described her current distress over being able to provide support and finances for her child:

“Yes, not being able to feed her, the exact, you know enough, cuz money is very tight and money is going to bills, and you know I mean she comes first, but we need a roof over our heads too so not being able to provide for her”

One parent stated her fears of keeping her children safe as her daughters grow:

“Okay, if you have girls you have to worry about them going out there and getting this one person that whisper that nice something in they ear and you become a momma. That’s my worry every day all day, and basically how every day I try to figure out how I’m going to survive with them, meaning that financially as they get older things expensive so it gets hard sometimes to figure it out, but then you come around to it, so...”

Chapter 5.

DISCUSSION/RECOMMENDATIONS/CONCLUSIONS

The purpose of this exploratory, feasibility study was to assess physiological and behavioral stress, and mental health profiles among high-risk mothers for CM. This project focused on assessing associations between self-reported stress and mental health symptomology with physiological measures, including, cortisol, an established stress biomarker. Apart from cortisol, little scientific research has been conducted on other salivary measures of stress. Given the paucity of this literature on additional biomarkers, alpha-amylase and DHEA were added to this study as exploratory markers for perceived stress. This study was also conducted to assess effects of a 6-session, evidence-based SafeCare® PCI intervention, known to reduce risk of CM and parental stress, in regulating biobehavioral stress among participants. Particularly, analyses were conducted to assess whether SafeCare® completers would exhibit positive changes in the aforementioned physiological marker levels, as well as in self-reported stress and mental health symptomology following the intervention. Findings from this study demonstrated the potential of salivary cortisol and sAA to be strong correlates of self-report measures for parental stress in violence prevention research. sAA was also strongly associated with mental health symptomology among trauma-exposed participants. While non-significant, trends in improved levels and scores for physiological and self-report measures were observed among participants who completed the intervention.

In the present study, one of the exploratory hypotheses was unfounded. No patterns emerged between DHEA and other outcome variables. Given the limited research on DHEA in the literature, these results were not surprising. These results indicate that DHEA may not be an appropriate surrogate marker for physiological stress among this high-risk sample. The

remainder of this discussion will therefore, focus on cortisol and alpha-amylase. All findings are discussed in detail in the following sections.

Examining Relationships between Physiological and Self-report Measures

Findings from this study showed varying interrelations between salivary cortisol, parental stress and mental health outcomes in response to the SafeCare® PCI intervention. For example, lower levels of cortisol were associated with higher levels of self-reported perceived stress among participants, prior to intervention engagement. Of particular interest, parents with hypocortisolic profiles, or blunted cortisol levels below the normal physiological range, reported clinically significant levels of parental stress at baseline. Such results would support that salivary cortisol may be an appropriate, objective marker for clinical levels of perceived stress in such populations. Within this sample, no parents demonstrated hypercortisolic profiles (i.e., cortisol levels above accepted physiological levels). Thus, relationships between abnormally high salivary cortisol and parental stress scores could not be assessed.

This research also assessed additional exploratory markers for acute stress, sAA, as well as a measure of chronic stress using cortisol derived from hair. Results indicate that sAA may be a novel and strong correlate for parental stress, and mental health symptomology among high-risk parents. Like salivary cortisol, parents with impaired sAA levels (hyper) also reported clinically significant levels of parental stress at baseline. These patterns were maintained at follow-up. Correlations observed between sAA levels and perceived stress, global distress and depression, strengthen the argument and importance of including sAA as a biological stress and mental health parameter for SNS functioning. While these results must be interpreted with caution, given the small sample of participants, changes in sAA reactivity may reflect more the

immediate changes seen in self-reported psychosocial measures following the intervention, as compared to salivary cortisol. Collectively however, findings suggest that both physiological markers are correlates of parenting behavior among high-risk parents.

Baseline findings from the current study contrast with previous research observing no correlation between salivary cortisol and sAA measures (Chatterton et al., 1996; Granger et al., 2006; Nater et al., 2006; Wolf, Nicholls, & Chen, 2008). However, post-intervention findings did show no correlation between these variables. These results could suggest that sAA and cortisol are more highly correlated during periods of acute stress (i.e., prior to intervention), but less so when acute stressors have decreased (post-intervention). Thus, it may be plausible to use sAA and cortisol as distinct or non-redundant parameters to independently measure changes and activity of different physiological stress response systems, SNS and HPA functioning, respectively (Wolf et al., 2008). However, additional research among a larger group of participants is needed to evaluate the relationship between sAA and salivary cortisol. Furthermore, examining sAA and salivary cortisol over each intervention session, may better inform the point at which systematic responses diverge.

Exploratory analyses looking at correlations between sAA levels and 3-month cumulative/ chronic cortisol (i.e., extracted from hair) among completers at both assessments showed trending associations between these biomarkers. No known studies have examined relationships between these markers. These findings are therefore, novel. The potential association should be given more consideration in future research to gain greater understanding of the complexity of physiological stress regulation.

In this study, no relationship was found between chronic cortisol levels and any behavioral outcome at either assessment. Further, no correlations were found also between hair and salivary cortisol levels. Research on the relationship between cumulative and acute cortisol is limited and mixed, with some evidence suggesting moderate associations between the two biomarkers (van Holland, Frings-Dresen, & Sluiter, 2012) and other findings showing no associations (Sauvé et al., 2007; Steudte et al., 2011).

Collectively, the lack of associations in the present study between chronic cortisol and other outcomes could suggest that acute and chronic cortisol serve different functions, where salivary cortisol is a more appropriate correlate of self-reported measures of acute stress. Given that chronic cortisol is a measure of a 3-month estimation of systemic cortisol, this biomarker may be a more effective correlate for self-report measures capturing retrospective stress. For example, in one study by Kalra et al. (2007), hair cortisol levels were correlated with self-report measures among 25 pregnant women who reported on the Perceived Stress Scale (S. Cohen, Kamarck, & Mermelstein, 1983), a validated self-report questionnaire assessing individuals' stress experiences within the previous month.

With regards to participants who reported trauma exposure, several noteworthy findings emerged. The strong correlations between sAA, PTS and other mental health symptomology among trauma-exposed participants at baseline are in line with the few, existing studies showing strong associations with sAA and chronic stress. For example, in a study by Rohleder et al. (2008), chronic shame experiences (experienced for several months) and depressive symptomology were significantly and independently associated with sAA levels among 56 females between ages of 15 and 19 years. Similarly, Vigil et al (2010), report higher basal sAA levels among 62 females exposed to Hurricane Katrina two-months post-disaster, in comparison

to a control group reporting no trauma exposure. Thus, findings from the current study support the potential of using sAA response patterns as correlates for not only acute stress, but also chronic stress in these high-risk populations. However, further data are needed to explore these associations. An additional finding of interest was the trending relationship observed between sAA and both salivary and hair cortisol among trauma victims. Such data highlights the importance of data collection on both SNS and HPA functioning to understand stress profiles and stress outcomes among this vulnerable population.

With respect to cortisol, no correlations between salivary and cumulative cortisol with PTS or other mental health symptomology were observed. However, upon closer, visual inspection of these data, three of four parents reporting severe levels of PTS, reported hypocortisolic salivary profiles. Chronic stress exposure has been established as associated with lower levels of HPA activity (i.e., lower levels of cortisol production), which may reflect desensitization of the HPA stress response (Flinn, Quinlan, Decker, Turner, & England, 1996; Miller, Chen, & Zhou, 2007). Collectively, these findings provide support for the relationship between symptom severity and impaired hormonal regulation as measured through saliva. However, these results should be interpreted in light of visual methods used and lack of significance tests. Given the small number of completers with trauma exposure, limited assessment of biomarker and psychosocial outcomes could be made among this group at follow-up. However, preliminary evidence (described below) showing the amenable nature of the measured biomarkers could lend early support for SafeCare® to have positive impact on biobehavioral functioning that may extend to trauma victims.

Biobehavioral Responses to SafeCare®

While no significant correlations were found between cortisol and self-reported parental stress among participants who completed the intervention, it is important to note that trends towards significant improvement in salivary cortisol regulation were seen collectively among this group. Of great interest, all parents with hypocortisolic profiles fell within normal limits of cortisol production at completion. Thus, lack of correlations between salivary cortisol and perceived stress post-intervention may not be of great value when considering the plausible positive intervention effects on physiological hormonal regulation. Like salivary cortisol, average sAA levels in this study showed trends towards significant improvement among completing participants. While not all participants showed normalization, sAA levels did show improvements towards the standard range for most participants. Altogether, these findings not only suggest physiological plasticity in adulthood, but the impact of SafeCare® on shaping stress regulatory systems.

Although non-significant, decreases in perceived stress levels, global distress, depressive and anxiety symptomology were found among all participants. However, one participant following the intervention reported an increase in anxiety levels, as well as a sizeable increase (>10 points) to her already clinically significant parental stress levels. This mother reported several changes in her home environment over the course of her intervention involvement including temporary psychiatric admission to hospital care after completing the SafeCare®, which may have led to the noted increase her psychosocial stress. Of interest, this participant showed improvement in salivary cortisol production at follow-up. While these data are exploratory in nature, findings from this participant and other completers provide compelling

evidence that SafeCare® engagement may overall assist mothers in improving biobehavioral outcomes.

In the one known study to date by Toth and colleagues (2015) examining the impact of an evidence-based parenting intervention on stress hormone regulation and parental stress among high-risk mothers, comparable findings were demonstrated with their measured hormone of interest, salivary cortisol. Notably, however, in the aforementioned study the intervention was much lengthier and intense in nature (one-year) and, consequently, the assessments in the study were conducted at longer time-intervals. Data from the current study indicate that intervention effects, both physiological and behavioral, may be observed from parenting programs of shorter duration. Nonetheless, taken together, both studies collectively underscore potential of evidence-based parenting interventions to influence both behavioral and physiological functioning among high-risk mothers and highlight the potential for positive short and long-term health implications in reducing stress-related symptomology.

With regards to hair cortisol levels in the current study, a minimal change in cumulative cortisol was seen among parents following the intervention. However, the short time interval between pre and post assessments (<2 months) likely limited the breadth of change that could be observed post-assessment. To more accurately assess changes in chronic cortisol levels in the future, additional follow-up measures would be needed at least three months post-intervention to collect new hair.

Acute Stress Response to Home Visitors

In examining acute salivary cortisol stress participant responses to home visiting sessions, no significant increases in acute stress were observed in the presence of the home-visitor. While

average cortisol levels did increase at follow-up, this change was non-significant. Therefore, the hypothesis that home visitors pose psychosocial stress was not supported. The slight variation in average salivary cortisol at follow-up may reflect normal physiological fluctuations, or a possibility that participants experienced minimal stress during the session with home-visitors. However, since this assessment of acute stress was conducted mid-way through the intervention, rapport may have already been established between participant and home-visitor. Thus, acute stress responses to home-visitors may be better captured earlier in the intervention, such as during Session 1, a period when rapport building is in early stages between parent and home-visitor. However, in light of evidence from this study and additional support from psychological stress research showing sAA to be a highly sensitive marker to changes in acute stress (Chatterton et al., 1996; Thoma et al., 2012), the latter enzyme may be a more efficient measure of stress responses than salivary cortisol among participants in the future. sAA was included in this study as exploratory measure to assess pre- and post-intervention differences and was therefore not considered to measure changes in acute stress at Session 3.

Feasibility of Biomarker Collection

Given the importance of identifying non-invasive methodology that can be used in behavioral research among high-risk populations, qualitative research was conducted among participants to assess feasibility of collecting various biospecimens among this high-risk population. Findings demonstrate that while some methods such as passive drool, provided discomfort, parents were amenable to participating in similar research salivary methods in the future. Based on these results, efforts should be made to find alternative methods to collecting passive drool in the future. However, since no significant correlations or trends emerged between DHEA (i.e., collected through passive drool) and psychosocial variables, biomarker collection in

the future could be limited to Salivette® methods to capture biomarker surrogates for HPA and SNS stress responses (e.g., cortisol and sAA, respectively). Comments from participants also warrant exploration of a smaller saliva swab (Salivette®) to make the saliva collection process more comfortable. In addition, given that some participants noted difficulties in producing saliva, mechanisms to enhance salivary flow rate should be considered in the future. Altogether, however, parental reports demonstrated that biospecimens collection is a feasible process, thus supporting the use of salivary aids and hair as alternatives to invasive blood specimens in future biobehavioral research.

Examining Parental Definitions of Stress

Qualitative interviews were conducted primarily to understand parental perspectives of factors that contribute to parenting stress in comparison to questions commonly used self-report measures assessing parental stress. Analyses of transcribed interviews suggested several themes on parental perceptions of stress. Notably, the majority of parents felt that little of their daily stress was attributable to parenting, which may be reflected in the non-clinical average of perceived parental stress scores. While parents described several daily activities (e.g., bathing, dressing, eating) that may be addressed using measures such as the PSI, several parents also described more chronic concerns regarding social support, financial and housing circumstances that interfere with parenting. Research suggests that mothers living in at-risk circumstances experience cumulative trauma that negatively impact their own hormonal physiology (Brand et al., 2010; Bublitz & Stroud, 2013) as well as the parenting responsiveness and behavior with their own children (Banyard, Williams, & Siegel, 2003; Bugental et al., 2010; Chemtob, Gudiño, & Laraque, 2013; L. R. Cohen, Hien, & Batchelder, 2008; Lang, Gartstein, Rodgers, & Lebeck, 2010). Specifically, mothers negatively affected by instability in their proximal environments,

respond to children with increased stress and a decreased ability to provide sensitive parenting (Corapci & Wachs, 2002; Deater - Deckard et al., 2009; Matheny, Wachs, Ludwig, & Phillips, 1995). Thus, the qualitative findings from this study illustrate the multidimensional nature of stress that can accompany the demands of parenting. In light of the discussion of chronic, situational factors described to affect parental stress, future studies should consider inclusion of additional measures of general stress that can affect this population. Based on the quantitative findings from this study, however, SafeCare® may be effective in reducing some of the parenting and general stress concerns voiced during interviews. Improvements in physiological biomarker regulations also support that while parents experience a battery of stressors in their daily lives, biological indicators are amenable to change with this behavioral parenting intervention.

Strengths of the Current Study

This feasibility study is the first to examine multiple acute and chronic physiological responses to an evidence-based parenting intervention. Assessing multiple stress responses provided novel information not only on the HPA axis (i.e., acute and chronic stress), but also on markers capturing SNS functioning. The use of saliva samples allowed for simple and non-invasive sample collection on acute biomarkers from participants. Particularly, this project included novel instrumentation, the Salivette® (vs. passive drool), to assess acute cortisol as well as sAA levels. Studies evaluating cortisol production via Salivette® in response to psychotherapeutic interventions have been conducted among children (Brotman et al., 2007; Fisher et al., 2007), adolescents (Gunlicks-Stoessel, Mufson, Cullen, & Klimes-Dougan, 2013), and individuals with post-traumatic stress syndrome (Young & Breslau, 2004; Young, Tolman, Witkowski, & Kaplan, 2004), history of child abuse (Koopman et al., 2003; Pierrehumbert et al.,

2009), acute stressors (Gaab et al., 2003; Gaab, Sonderegger, Scherrer, & Ehlert, 2006; Hammerfald et al., 2006) and adults with work-related burnout (Mommersteeg, Keijsers, Heijnen, Verbraak, & van Doornen, 2006). However, no known study has assessed cortisol via a Salivette device among parents at risk of CM.

Similarly, while most existing research in maternal and child health has focused on saliva to capture short-term excretion of cortisol, fewer studies have examined chronic levels of stress as a result of the lack stable biomarkers. Thus, the use of hair provided a simple, but efficient mechanism to retrospectively explore long-term chronic stress hormone reactivity in this population. Another strength of this study was the short duration of SafeCare®'s PCI module, lasting approximately 6 weeks, which allowed follow-up assessments to depict changes in measures that could occur over a short time interval.

Limitations

Several limitations must be considered in light of the exploratory nature of this research. First, this quasi-experimental study was conducted with the goals of obtaining pilot data to support the in-depth investigation of physiological functioning and biobehavioral stress responses in the future. However, the implemented design lacked a comparison group, and was conducted among a small sample of parents. Lack of statistically significant findings may have resulted from limitations in power. Second, sample collection was conducted generally between 1-3:30 pm, when cortisol levels are considered most stable and at baseline levels. However, a few parents provided samples after this window (3:30-4pm). Further, no data was collected on wake-times for participants, which may also have affected the diurnal pattern of cortisol production, and therefore, the relative stability of levels during the time of collection. Third, while research assistants asked parents to confirm their non-use of steroid hormone and other

medications that may interfere with salivary assays, this assessment was conducted at baseline only. Further, one parent reported being pregnant during this study. While pregnancy can alter physiological regulation, this variable and other potential confounders were not controlled for in analyses given the small sample size. Similarly, no data were collected on covariates such as BMI (Dockray, Susman, & Dorn, 2009; Putignano et al., 2001), hair washing (Hamel et al., 2011), and smoking status (Badrick, Kirschbaum, & Kumari, 2007; C Kirschbaum, Wüst, & Strasburger, 1992), which may interfere with cortisol levels. Fourth, changes in biobehavioral outcomes were measured at a 1-week follow-up among parents who completed the PCI module. Thus, the observed findings cannot speak to long-term impact of engagement on physiological and behavioral intervention outcomes.

Future Directions

Given this preliminary support for SafeCare® in improving stress regulation, research studies should be replicated in a larger sample to examine the short- and long-term effects of SafeCare® on biobehavioral health. More rigorous methodology (i.e., randomized controlled trial) in a high-powered study is needed to support the correlations seen between physiological and self-report measures, as well as to establish causal associations between SafeCare® participation and improved biobehavioral outcomes. Given the exploratory nature of the conducted study, no goals were proposed to test for mediating roles on the relationship between SafeCare® enrollment and intervention outcomes, moderating roles on the relationship between perceived stress and parenting outcomes, nor the influence of protective and other risk factors on parenting outcomes. However, a larger sample size, and highly powered study can help clarify such mechanisms in the future.

Future studies should also include multiple, longer-term follow-up assessments to continue to monitor changes in stress levels as well as maintenance of parenting skills delivered by SafeCare®. This methodology will allow for the examination of trends in stress regulation among parents who continue or discontinue SafeCare® parenting skills. Furthermore, additional research is needed to identify and control for potential confounders such as common medications that may affect biomarker levels. Similar analytical consideration must be given to self-report of biological or medical conditions that may interfere with physiological measures and readings.

Inclusion of children in these studies should also be considered in the future to assess the biobehavioral effects of SafeCare® on maternal-child dyads, as well as to examine associations between early childhood adverse experiences and well-being. Shonkoff and colleagues (2012) proposed an ecobiodevelopmental (EBD) framework, which considers the complex relationships amongst biology, ecology, and health/development. The framework incorporates a taxonomy of stress, which includes: 1) *positive stress*, or common stress that is brief and of mild intensity, 2) *tolerable stress*, or stress that is higher threat that may interrupt daily activities, and takes longer to resolve, and 3) *toxic stress*, or stress that is intense and unresolved and leads to strong, frequent and prolonged activation of the body's stress response systems (Herman-Smith, 2013; Sparrow, 2007). Under the EBD perspective, toxic stress in young children's lives leads to physiologic responses that impair well-being throughout life. Central to the EBD framework is a focus on initiatives that can build sensitive and responsive parenting practices that can buffer stress and support healthy child development (Coley, Lynch, & Kull, 2015). SafeCare® may be one such example that could lead to an interruption in toxic stress among young children. Given the compelling foster care research (previously mentioned) demonstrating measurable changes in the cortisol levels of young children in response to parenting interventions, inclusion of this

young population in future research will fully explain how SafeCare® can reduce behavioral and emotional triggers for stress among parents, enhance the quality of PCI, and consequently interrupt sources of toxic stress risk among these children.

Generally missing from studies conducted to date among children as well as maternal populations, are genetic indices of stress. Examining multiple markers of stress, in addition to cortisol and sAA, will contribute to a comprehensive stress profile among maternal-child dyads engaged in SafeCare®. For example, future research can consider telomeres, repetitive DNA sequences that protect chromosomal ends (Epel et al., 2004; Parks et al., 2009; Tyrka et al., 2010). Truncated telomere length represents a biological marker for cellular aging (Enokido et al., 2014) and has been described as a ‘psychobiomarker’ linking stress and disease (Epel, 2009; Epel et al., 2004; Ornish et al., 2008). There are no published and replicated studies demonstrating that telomere length is reversible in human or *in vivo* models. No research to date has examined this outcome among adults or children in response to CM intervention efforts. However, there is a strong rationale for including telomere length measures for mothers, as those with truncated telomeres may have a long history of chronic stress and may respond differently to SafeCare®.

In addition to telomere length, research on epigenetic sciences suggests gene-environmental interactions lead to stable changes in gene expression and silencing, and subsequent cell functioning (Zhang & Meaney, 2010). One such epigenetic event, DNA methylation occurs can alter gene functioning and lead to long-term changes within DNA segments that regulate HPA activity. This epigenetic modification can subsequently alter stress responsiveness that can be sustained into adulthood (Meaney & Szyf, 2005). Understanding that chronic stress may lead to both hypocortisolic and hypercortisolic profiles supports examining

different global DNA methylation patterns and genetic susceptibility. Furthermore, this genetic information can allow researchers to further explore how these factors may influence cortisol levels and possible responses to interventions.

Implications and Conclusion

Important implications can be drawn from this exploratory study. Findings support the continued examination of salivary cortisol and sAA as new, relevant correlates for parental stress among high-risk maternal populations for CM, as well as for possibly mental health symptomology among those with trauma history (i.e., sAA). Most importantly, data trends provide preliminary support for the use of evidence-based practices as an approach to potentially mitigate negative physiological regulation among high-risk mothers; high-risk mothers who completed the intervention showed trends towards normalization of salivary cortisol and improvements sAA levels at the follow-up assessment.

A critical question then arises, on the mechanism driving the noted improvements in salivary biomarker regulation among parents following intervention engagement. SafeCare® sessions include an explanation of target skills, modeling of target skills by home visitors, practicing of the target skills by the parent, and feedback from the provider about mother mastery and competence in skills. Favorable changes in psychosocial and biological outcomes at follow-up may result from changes in self-efficacy and the uptake and mastery of PCI skills over the course of the intervention. Research has shown self-efficacy as an important mediator of relationships between maternal parenting and psychosocial outcomes, such as mental distress (Halpern & Mclean, 1997; Jackson & Huang, 2000; Jones & Prinz, 2005). Research among evidence-based programs suggests that improved behavioral outcomes and decreased risk of

abuse may result from reduced negative parent-child interactions (Chaffin et al., 2004). While these pathways to change must be explored, the quasi-experimental design and exploratory nature of this study did limit the type and scope of data analyses that could be performed. However, the observed improvements in physiological and psychosocial functioning among parents following SafeCare®, lend support to continue this line of research in the future, and to examine plausible mechanisms leading to improvements biobehavioral outcomes.

In conclusion, this innovative pilot work provided unique and noteworthy biobehavioral findings for the fields of violence prevention and maternal and child health. There is a dearth of research on the relationship between physiological measures and perceived or self-reported measures among high-risk adult populations for CM. The common use and analysis of self-report measures of stress in research has limited the understanding of complex stress responses to intervention. Use of multiple markers of stress in this study produced more comprehensive psychobiological stress response profiles among this high-risk population that would not be obtained by measuring self-report measures alone. Thus, this study was able to demonstrate the multidimensional nature of parental stress among a high-risk population.

In addition, positive salivary biomarker responses to SafeCare® provided evidence of physiologic plasticity among a high-risk adult population in response to a public health intervention known to reduce self-report levels of stress and CM perpetration. Such results also elucidate the potential of SafeCare® to affect physiological regulatory systems in a population known to experience several environmental hardships. Given the noted feasibility of collecting multiple methods of biospecimens, these methods should be utilized in violence prevention research to not only continue exploring the biobehavioral profiles of high-risk populations, and responses to public health interventions, but also to help identify effective evidence-based

strategies that contribute to positive psychosocial and physiological outcomes among these high-risk populations.

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