

CONTEXT, CORTISOL, AND EXECUTIVE FUNCTIONS  
AMONG CHILDREN EXPERIENCING HOMELESSNESS

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**Abstract.**

Homelessness represents a context of risk for child development. Yet, many homeless children show good develop outcomes, nonetheless. The processes of risk and resilience that contribute to this variability involve adaptive systems impacted by factors across levels of analysis, such as cortisol and physiology, executive functions (EF) and other aspects of psychological functioning, and parenting behavior and the family context. This study employs a resilience framework that is grounded in developmental-ecological theory and recognizes factors at multiple levels of analysis. The goal is to elucidate explanatory models of the processes of risk and resilience by incorporating relationships with cortisol, a component of physiological adaptive systems related to the stress response, self-regulation, and other functions.

Families in this study were all staying in an emergency homeless shelter and contained a child entering kindergarten or first grade. Children were separated from caregivers and completed a session of cognitive tasks that assessed executive functions and other abilities, followed by a session of parent-child interaction tasks. Saliva samples were collected throughout both sessions and assayed for cortisol concentrations. Parents reported on risk factors and stressful negative life events for each child. Initial levels of child cortisol were negatively related to EF, affirming a proposed inverted-U relationship between cortisol and cognition among this sample of high-risk children. Higher rates of stressful, negative life events were not related to cortisol, nor was positive parenting behavior. However, harsh, hostile, and insensitive parenting behaviors were related to higher levels of child cortisol, but only during the session when parent and child were together. There were no differences based on variables of interested when it came to changes in cortisol over either session. Results are discussed with respect to proposed mechanisms of the interface between cortisol, parenting and EF at different levels of analysis in the context of high developmental risk.

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## Context, Cortisol, and Executive Functions among Children Experiencing Homelessness

Homelessness represents a context of extreme poverty and multiple risks to child development. Groups of children who experience homelessness show lower levels of academic achievement and attainment, and higher levels of mental and physical health problems (Rog & Buckner, 2007; Samuels, Shinn, & Buckner, 2010). Many children who experience homelessness show good developmental outcomes, nevertheless. The processes of risk and resilience that contribute to the variability in individual outcomes are complex, occur across multiple levels of analysis, and unfold over time. These are the processes of successful adaptation or maladaptation. Understanding these processes has the potential to illuminate how differences emerge for children who experience poverty and high levels of adversity, with a promissory note to inform efforts at promoting competence and preventing maladaptive development.

Developmental risk and resilience emerge from many processes within and between systems, both within the organism and between the organism and many contexts (Cicchetti & Valentino, 2007; Masten, Cutuli, Herbers, & Reed, 2009). There is a robust tradition and literature on models of risk and resilience based on largely psychosocial factors (Luthar, 2006). Meanwhile, over the past several decades there has been a surge of research that explores possible influences of physiological factors on positive adaptation and maladaptation. These factors span multiple disciplines and traditionally considered 'levels of analysis,' such as molecular, genetic and cellular processes, physiological systems (e.g., neurobiological, endocrine, immunological), and their interaction with each other and with the social context and experience of the individual (Charney, 2004; Cicchetti & Blender, 2006; Curtis & Cicchetti,

2003; Masten, 2007). Appreciating how factors relate across levels of analysis, as well as ways in which they do not, may lead to different ways of conceptualizing good and poor outcomes as the product of interconnected processes.

The purpose of this investigation is to explore the processes of risk and successful adaptation in a very high-risk group of children as related to cortisol levels, one component of physiological functioning. Cortisol is a regulatory hormone that influences a variety of systems and functions, including the autonomic stress response, metabolism, immune function, and others. Cortisol also crosses the blood-brain barrier, allowing it to affect neural systems and functioning related to controlling emotion and behavior. Furthermore, cortisol levels and function can be impacted by stressful experience during development. Understanding the links between stress, cortisol, and developing cognitive abilities may hold implications for self-regulation skills that are important for success in the early school years (Blair, 2010; Blair, Granger, & Razza, 2005; Gunnar & Vazquez, 2006).

The conceptual organization of this study is grounded in developmental-ecological theory applied to a resilience framework with an acknowledgement of influential factors at multiple levels of the individual and her context (Bronfenbrenner, 1979; Curtis & Cicchetti, 2003; Luthar, 2006; Yates, Egeland, & Sroufe, 2003). Adaptation is impacted by external factors (e.g., family, school, community, culture, public policy) and internal ones (e.g., molecular/genetic disposition, structure and function of biological systems, psychological functioning) that interact, transact, and co-act in complex ways to influence functioning and development (Gottlieb, 1991). Past work has described a pathway that links parenting (an external system) with child cognitive self-regulation at the psychological level (internal system) and positive developmental outcomes

among homeless children (Buckner, Mezzacappa, & Beardslee, 2003; Herbers, 2011; Herbers et al., 2011). Yet, homeless children who experience higher levels of risk tend to show worse cognitive self-regulation skills (Obradović, 2007). The current study looks to further elaborate on these relationships by incorporating cortisol function, an aspect of the physiological stress response that has been linked to differences in experiencing adversity in early childhood and the preschool period, differences in executive functions related to self-regulation, and differences in parenting (Cutuli, Wiik, Herbers, Gunnar, & Masten, 2010; Wiik et al., under review). Consonant with the conceptual framework, incorporating factors at additional levels of analysis into existing explanatory models will elucidate the processes of resilience and maladaptation.

This study has four primary aims: (1) to affirm that salivary cortisol can be measured with a reasonable degree of fidelity in the shelter context with young children; (2) to test if executive functions relate to differences in dynamic cortisol function during a session of cognitive tasks; (3) to relate cortisol function to psychosocial factors (negative lifetime life events, parenting behavior) that may influence the development of physiological regulatory systems; and (4) to explore the relationship between cortisol and regulatory systems that are internal (e.g., executive function) and external (parenting behavior) during a session of parent-child interaction tasks.

## **Background**

The literature review is organized in 5 main sections. In the first section, I summarize the evidence on homelessness as a marker of high cumulative risk and stress for children. In the second section, I review the research on cortisol in the context of high risk and stress, including links between cortisol and parenting. The third section discusses the literature indicating a

central role for two key protective systems for adaptation and development in HHM children, namely parenting and executive functions related to self-regulation skills. The fourth section outlines proposed physiological mechanisms by which cortisol may be related to executive functioning. Finally, a summary and integration section comments on the apparent interplay between cortisol and other physiology, EF, parenting quality, and adversity that may contribute to developmental differences in competence among homeless children in kindergarten and first grade.

**Childhood homelessness in the United States.** Homelessness is a prevalent and serious threat to child development in the United States. While definitions vary across service agencies, families who are homeless generally ‘lack a fixed, regular, and adequate nighttime residence’ (U.S. Code, Title 42, Chapter 119, Subchapter I, § 11301). This includes, but is certainly not limited to, families staying in emergency shelter, ‘doubled up’ and staying with friends or relatives because of a lack of housing, and living in public or private spaces not ordinarily used as sleeping accommodations (e.g., in cars, parks, abandoned buildings, substandard housing). A relatively small percentage of homeless children and adolescents are unaccompanied by their families.

Families in emergency shelter represent an important and sizeable group for stakeholders in the wellbeing of low income children. According the U.S. Department of Housing and Urban Development (HUD) counts from October, 2007, through September, 2008, 20% of all persons staying in HUD shelters were children, representing 326,400 youth. Fifty-one percent of sheltered children were under the age of 6, while 85% were under the age of 13 (Samuels, et al., 2010; U.S. Department of Housing and Urban Development: Office of Community Planning

and Development, 2009). Even though the U.S. Department of Education reports that a minority of homeless children stay in shelters (21%) (National Center for Homeless Education (NCHE), 2009), the shelter context provides a window into adaptive processes as they are unfolding, and a point of contact for service providers looking to assist families with young children at high levels of risk. Children staying in shelter represent an important group for those interested in understanding the processes of risk and resilience in order to intervene and improve the lives of these children and families.

Homelessness almost always represents a context of extreme poverty, and it is accompanied by multiple other risk factors and stressful life events (Rog & Buckner, 2007). Gewirtz, Hart-Segos, and Medhanie (2008) found high rates of psychosocial risks and problems among 454 formerly homeless children in supportive housing programs around the Minneapolis-St. Paul area. Specifically, nearly half of parents had a substance use disorder, about half had a mental illness diagnosis, and 36.7% of children had an open child protection case. Additional research with homeless families reports high rates of other risk factors for poor developmental outcomes, including a high percentage of single parent families, a history of high residential mobility and instability, poor school attendance and increased rates of school mobility, family separations, low parental education, parental substance abuse, exposure to family conflict and violence, and other traumas and adversities (Cutuli, Wiik, et al., 2010; Rog & Buckner, 2007; Samuels, et al., 2010). Combined with the definitional certainty of a disruption in residential stability, homeless children tend to experience very high levels of risk, adversity, and stressful experiences in the context of poverty.

Children who experience homelessness are at increased risk for a number of poor outcomes, including poor mental health, low levels of competence and self-esteem in multiple domains, and increased rates of chronic illness and susceptibility to disease (Rog & Buckner, 2007; Samuels, et al., 2010). This may not be surprising given the large number of concomitant adversities and stressful experiences that accompany childhood homelessness; while the experience of homelessness itself may also contribute to some of the poor developmental outcomes evidenced in this group (Cutuli et al., Under Review; Masten, Miliotis, Graham-Bermann, Ramirez, & Neemann, 1993; Rafferty, Shinn, & Weitzman, 2004). Given the prevalence of childhood homelessness and its links to poor outcomes, the effects of homelessness on children likely contribute to well-established socioeconomic disparities in achievement, mental health, and disease (Obradović et al., 2009).

Homeless children are at especially high risk for low levels of academic achievement and attainment (Buckner, 2008; Obradović, et al., 2009; Samuels, et al., 2010). Few studies have investigated school readiness or academic achievement among homeless children specifically in the early school years, although preschoolers staying in shelters tend to have lower scores on standardized tests of cognitive development relative to national norms and to housed, low-income children (Bassuk & Rosenberg, 1990; Garcia Coll, Buckner, Brooks, Weinreb, & Bassuk, 1998; Rescorla, Parker, & Stolley, 1991; Wood, Valdez, Hayashi, & Shen, 1990). Analyses of standardized testing in the 1<sup>st</sup> grade using all available data from Minneapolis Public Schools find that homeless and highly mobile students underperform stably housed, low income peers. This affirms that academic disparities are in place by the early grades (Herbers et al., in preparation). Furthermore, these gaps persist at least through the eighth grade (Cutuli, et al., Under Review).

Even though homeless children underperform more advantaged children as a group, considerable variability exists in key developmental outcomes when considering individuals. For example, about 43% to 63% of homeless or highly mobile (HHM) students in Minneapolis Public Schools scored within or above a standard deviation of standardized test norms in reading and/or math achievement over multiple school years, reflecting academic resilience (Cutuli, et al., Under Review; Obradović, et al., 2009). Buckner, Mezzacappa, and Beardslee (2003) used a sample of extremely low income 8 to 17 year olds (those with a history of homelessness were overrepresented) to find that 29% displayed resilience considering assessments across multiple domains, including good mental health, adaptive functioning, and competencies in multiple areas. Obradović (2010) found that 41% of homeless 4 to 7 year olds showed resilience by attaining at least average levels of academic functioning and peer competence without clinical levels of psychopathology. Meanwhile, in the early 1990s Masten's group documented notable percentages of sheltered homeless children in Minneapolis who 'beat the odds' and show resilience across a number of domains (Masten & Sesma, 1999). Together these findings suggest that while the processes of risk predominate at the group level for homeless children, sizeable percentages of resilient individuals do well nonetheless. The existence of these children presents the opportunity to explore the processes of protection and resilience among homeless youth.

#### **Incorporating Cortisol Function in Models of Adaptation and Maladaptation.**

Appreciating the role of developing cortisol function in models of adaptation and maladaptation may help to explain the processes that contribute to achievement and health disparities for individuals who experience childhood homelessness and low socioeconomic (SES) status. Like

with any developmental outcome or characteristic, developing physiological systems appear to co-act, interact, and transact with each other, with the child's psychosocial environment (including family systems, parenting behavior, and the parent-child relationship), with developing cognitive and psychological functioning, and with factors at other levels to produce developmental change and eventuate in a level of functioning at any given point in time (Gottlieb, 1991). While investigations at any single level of analysis hold great importance, expanding accounts to include factors at multiple levels of analysis has potential to further understanding of the complexities of developmental processes (Cicchetti & Valentino, 2007; Curtis & Cicchetti, 2003; Masten, 2007).

This study seeks to add cortisol functioning to models linking stress, parenting, and child executive functions. Cortisol, a glucocorticoid, is the product of a series of hormones produced by the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis generally, and cortisol in particular, plays a central role in maintaining and restoring homeostasis in multiple physiological systems, including metabolism, immune functioning and inflammation, and cession of the sympathetic stress response. Cortisol levels follow a circadian rhythm under basal conditions where concentrations reach their daily peak during the first hour after waking up and then gradually decline over the course of the day.

Beyond these basal patterns, cortisol levels also change in response to a stressful experience (including both somatic and psychological stress) or in ambiguous situations where stress is anticipated. In young children, separation from parents and interacting with an unfamiliar adult have produced stress reactions and elevations in cortisol levels (Blair, Granger, et al., 2005; Nachimas, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). Stressors activate the HPA



axis initiating a cascade of endocrine responses beginning with the production of corticotrophin releasing hormone (CRH) from the hypothalamus. CRH then initiates a release of adrenocorticotrophic hormone (ACTH) from the anterior pituitary, which, in turn, activates other components of the sympathetic nervous system resulting in sympathetic arousal commonly referred to as the “fight-or-flight” response. ACTH also travels to the adrenal glands where it stimulates the production of cortisol. Cortisol works to interfere with the processes of sympathetic arousal in multiple physiological systems. Cortisol also readily crosses the blood-brain barrier to influence the functioning of neurological structures. Notably, cortisol curtails the activity of the HPA-axis at the levels of the hypothalamus and pituitary to encourage a return to basal levels through a process of negative feedback inhibition (Gunnar & Quevedo, 2007; Sapolsky, Romero, & Munck, 2000).

**Cortisol and Stress in Childhood.** Experiencing stress has been implicated in lasting alterations in HPA axis functioning and cortisol levels, especially if the stress is chronic or repeated and occurs early in life (Gunnar & Vazquez, 2001, 2006; Lupien, McEwen, Gunnar, & Heim, 2009). Chronic over- or under-activity of stress response systems results in physiological alterations, called allostatic load (McEwen, 1998). In the most general sense, stress response systems mobilize the organism to react in the face of threat or perturbation. In most cases, this protects the integrity and safety of the organism; it is beneficial to be able to run away from danger or to feel upset when you are doing something wrong. However, activation of these systems comes at a cost, as the stress response mobilizes some systems and suppresses others. Once the stress has passed, physiological systems operate to counteract this arousal and return to baseline modes of functioning (homeostasis). Experiencing repeated or chronic stress may

impair components of the stress response with respect to the initial arousal or the return to homeostasis. These alterations may be mediated by exposure to dysregulated levels of stress hormones, at least in part.

Individuals from low SES backgrounds tend to experience more stress that is more severe in nature (McLoyd, 1998), with homeless children facing even greater risk from recent stressful life experiences (Masten, et al., 1993). Furthermore, children from lower SES families tend to show elevated basal levels of cortisol (Fernald, Burke, & Gunnar, 2008; Lupien, King, Meaney, & McEwen, 2000, 2001). These differences are also present in adulthood: individuals from low SES upbringings show higher levels of cortisol and lower expression of GR genes compared to others who experienced high SES in childhood (Miller et al., 2009). Longer durations of past poverty exposure appear to be related to higher basal cortisol levels in preschoolers (Essex, Klein, Cho, & Kalin, 2002) and young adolescents (Evans & Kim, 2007). Poverty appears to represent a context of high allostatic load with lasting consequences for cortisol function.

The phenomena of hypercortisolism versus hypocortisolism further underscores the complexity in the relationship between psychosocial factors and developing endocrine systems. Hypercortisolism refers to the situation wherein cortisol is produced in excess compared to what would be expected in some context or situation, and sometimes this term is extended to generally higher levels of daily cortisol. Hypocortisolism, in contrast, refers to a blunted cortisol response or lower levels of daily cortisol (Gunnar & Vazquez, 2001, 2006). Hypocortisolmic patterns have been identified in some samples of children who experience high levels of risk, with the belief that these patterns mark a down-regulation of previously chronically high levels

of cortisol (Fries, Hesse, Hellhammer, & Hellhammer, 2005; Heim et al., 2000). Hypocortisolmic patterns have been found in children who experience extreme deprivation, such as extended periods in orphanages with very low levels of care (Gunnar, 2000; Gunnar & Vazquez, 2001) or experiencing neglect and/or certain other types of child maltreatment (Bruce, Fisher, Pears, & Levine, 2009; Cicchetti & Rogosch, 2001a; Dozier et al., 2006). Repeated exposure to stressful negative life events also seems to predict hypercortisolmic patterns or processes that may eventually result in hypocortisolism (Bevans, Cerbone, & Overstreet, 2008; Gustafsson, Anckarsater, Lichtenstein, Nelson, & Gustafsson, 2010). Such findings tend to assert or imply that the effect of repeated stressful negative life events may be less sensitive to the type to events, but more sensitive to the number or duration of events. This view is consonant with the idea of allostatic load.

Nevertheless, risk factors literally tied to low-income status (e.g., a lack of physical resources) tend to be relatively poor predictors of SES-related differences in cortisol functioning among low-SES children (Cutuli et al., 2010; but see also (Fernald & Gunnar, 2009). Meanwhile, factors that threaten the family and/or reflect poor family functioning generally operate better as predictors of child cortisol. Evans and Kim (Evans & English, 2002; Evans & Kim, 2007) found that the number of years children had lived in poverty predicted higher overnight urinary cortisol among a sample of 13 year olds. However, this poverty-cortisol link could not be explained by an index of psychosocial and physical-resource related risks, such as substandard housing, crowding, and exposure to violence. Flinn and England (Flinn, 1999; Flinn & England, 1997) did not find an effect of income on health outcomes, but did uncover a positive link

between higher cortisol levels during the day and family-related disruptions such as negative parenting practices (e.g., shaming).

Other studies have found that risk factors related to family functioning are more successful in predicting cortisol differences, usually among samples of low income children. In a rare longitudinal study, Chen, Cohen, and Miller (2009) found that 9 to 18 year olds from lower SES families showed greater increases in daily cortisol after two years compared to peers from higher SES families. This finding was partially mediated by the adolescent's level of perceived threat and family chaos. Specifically among homeless kindergarten-aged children, our group found that a cumulative risk score that included items related to a lack of physical resources did not relate to children's cortisol levels, but childhood adversity scores largely indexing threats to the child's family and family functioning were linked to higher morning cortisol levels, higher initial levels in a session of cognitive tasks, and a steeper decline across the session (Cutuli, Wiik, et al., 2010). In sum, among low income groups, factors related to family functioning, parenting, and the parent-child relationship likely play a larger role than poverty, per se, in influencing developing cortisol function. This includes family factors related to poverty (e.g., maternal stress) that may be mediated through family and parent level variables, consistent with a developmental-ecological perspective.

**Parenting, the parent-child relationship, and developing cortisol functioning.** Parenting in early life has implications for lasting differences in HPA-axis functioning. Extreme cases of dysfunctional caregiving have been linked to alterations in cortisol patterns. Childhood maltreatment represents a severe failure of the caregiving environment, and specific types of maltreatment have been linked to either increased or decreased levels of cortisol functioning. In

an early study, maltreated children were less responsive (lower cortisol levels) to stressful preschool days marked by high levels of conflict when compared to non-maltreated peers (Hart, Gunnar, & Cicchetti, 1995). Cicchetti and Rogosch (2001a) found that children who had been both physically and sexually abused showed higher levels of morning cortisol. In addition, psychopathological symptoms appeared to be relevant as maltreated children who showed clinical levels of internalizing symptoms also had high levels of cortisol across the day (Cicchetti & Rogosch, 2001b). Furthermore, children who had experienced either physical or sexual abuse prior to age 5 and high levels of depression symptoms showed higher levels of cortisol in the afternoon (Cicchetti, Rogosch, Gunnar, & Toth, 2010). Consistent with the view that developing systems are more vulnerable to perturbation than mature ones (Gunnar & Quevedo, 2007), this finding affirms relationships between extreme stress in the caregiving environment during the preschool period, developing cortisol functioning and related stress response systems.

Intervention work targeting parent sensitivity provides the most convincing evidence linking early caregiving and cortisol functioning. Fisher and colleagues (Fisher, Gunnar, Chamberlain, & Reid, 2000; Fisher, Stoolmiller, Gunnar, & Burraston, 2007) investigated a family-based intervention for foster parents with a 3 to 6 year old foster child. The intervention involved foster-parent training targeting the social-emotional and developmental needs of preschoolers in foster care, and has been found to reduce insecure attachment behaviors (Fisher & Kim, 2007). Foster families were randomized to either an intervention or a no-intervention control condition, and a third group of families was also included as controls that contained a non-maltreated child in the age range. Children placed in the intervention condition showed diurnal patterns of cortisol similar to the non-maltreated control children across 12

months of follow-up. Meanwhile, foster families that did not receive the intervention had foster children who showed flatter, more atypical cortisol levels. The authors speculate that this effect operates through foster parent behaviors that help protect the child's developing stress response systems starting with the highly stressful transition into foster care. Presumably, parents who are more sensitive to the child's developmental and social-emotional needs are also more likely to be able to buffer the negative impact of stress on developing physiological systems.

There are relatively few studies that investigate parenting quality and children's cortisol levels in less extreme populations. Nevertheless, parenting behavior has been linked to alterations in HPA axis functioning in children, especially with regard to cortisol levels. Essex, Klein, Cho, and Kalin (2002), for example, found that alterations in 4.5 year old children's diurnal cortisol levels vary as a function of prospectively measured exposure to maternal stress, an effect presumably mediated through the parent-child relationship. Further, cortisol levels at 4.5 years old predicted psychopathological symptoms in first grade based on self-, mother-, and teacher report. This suggests a link between parenting practices, child HPA-axis functioning, and later outcomes across contexts that are important to childhood competence, namely home and school.

Other studies have looked at parent-child interactions and their more immediate effect on child cortisol levels. In an exceptionally rare study of cortisol reactions among 5 year olds to a parent-child interaction session, Smeekens, Risksen-Walraven, and van Bakel (2007) collected cortisol before and 20 minutes after a ten-minute interaction task. Parents were instructed to ask children to recount and discuss past emotional events in the presence of an experimenter.

Parents were permitted to assist the child and help structure the child's performance. Videos of the session were coded for parenting and child behavior, yielding two broad factors of negative interactions and effective guidance. Teachers also completed a measure of child ego-control, reflecting the dynamic capacity to control impulses and emotions to meet the situational requirements. Neither parenting factor score was related to changes in child cortisol. However, an interaction term revealed that children who experienced negative parent-child interactions and were high on ego-control showed less of an increase in cortisol compared to children in negative interactions who had low ego-control. These findings echo others with younger children that show moderating effects of child temperament on cortisol reactivity in parent-child interactions (Nachimas, et al., 1996; Repetti, Taylor, & Seeman, 2002), affirming a multiple-levels approach.

Besides the deleterious effects of harsh, hostile, or negative parenting on cortisol levels, warm, sensitive, and supportive caregiving appears to also attune physiological systems related to HPA-axis functioning. A positive parent-child relationship and nurturing parenting behavior are linked with less activation of the HPA-axis when stressed, and a faster return to baseline levels. In work with younger children, stressful brief separations from parents resulted in higher levels of cortisol among children who showed disorganized patterns of attachment, an indicator of a suboptimal parent-child relationship early in life (Hertsgaard, Gunnar, Erickson, & Nachmias, 1995). The parent-child relationship is especially important to young children who are more temperamentally anxious: fearful/inhibited children with a secure attachment style showed a less pronounced response to experiencing stress compared to other fearful/inhibited children with insecure attachment patterns.

Animal models also assert the role of positive maternal behavior in developing HPA-axis functioning and related physiology. Gunnar and colleagues (Gunnar, Gonzalez, Goodlin, & Levine, 1981) found that infant rhesus monkeys with mothers who show more nurturing behaviors (e.g., more ventral contact) showed a more rapid decrease in cortisol levels after being returned from a prolonged separation. Numerous studies have linked positive maternal behavior in rodents to lasting differences in pups' stress reactivity. Pups that received greater amounts of maternal behaviors (arched-back nursing and tactile stimulation in the form of licking and grooming) show less fear and lower glucocorticoid responses to stress. This phenotype appears to be mediated, at least in part, by epigenetic processes denoted by differential rates of DNA-methylation of the GR promoter region that contribute to reduced GR density in the hippocampus (Hackman, Farah, & Meaney, 2010; Kaffman & Meaney, 2007; Meaney & Szyf, 2005).

Together, findings on parenting and the parent-child relationship support the importance of family-systems in the development of cortisol function, especially early in life, as postulated by a developmental-ecological perspective. Negative control and harsh, rejecting parenting behavior can exacerbate the processes of risk, while positive control and warm, sensitive caregiving can act as an external adaptive system that buffers the child's developing stress-response physiology from the negative effects of stress (Adam, Klimes-Dougan, & Gunnar, 2007; Gunnar & Quevedo, 2007).

**Predicting resilience: Key protective factors, processes, and adaptive systems.** A goal of this study is to link cortisol function to factors important for developmental competence among homeless children. Research into protective factors for homeless children has followed



from work on children who face adversity more generally. “Protective factors” refer to characteristics of a group or their situation that are related to some good outcome in the presence of risk or adversity (Luthar, Cicchetti, & Becker, 2000; Masten, et al., 2009). Two salient protective factors for children in the context of adversity are good cognitive functioning, especially skills related to self-regulation, and a close relationship with a competent adult, most often a parent (Luthar, 2006; Masten, 2001). Self-regulation and parent-child relationship quality are appealing to those interested in promoting resilience among homeless children. These constructs represent general protective factors linked to positive outcomes across multiple domains of functioning, and applied research has shown that they can be improved through intervention (Bierman, Nix, Greenberg, Blair, & Domitrovich, 2008; Diamond, Barnett, Thomas, & Munro, 2007; Luthar, et al., 2000; Masten, 2001).

Past work has established self-regulation skills and high quality parenting as important characteristics of resilient homeless children. Further work is needed that elaborates on how these naturally occurring protective factors have their effect and the processes that encourage or threaten their development. Numerous calls have gone out to better understand the processes and mechanisms of risk and protection with the goal of moving past correlations between broad factors and child outcomes (Curtis & Cicchetti, 2003; Masten, 2007; Rutter, 1987; Yates, et al., 2003). These efforts are valuable because they inform basic science related to successful adaptation and elucidate explanatory models of development in the contexts of risk and protection. This work holds the potential to reveal additional avenues for interventions while increasing the efficiency and effectiveness of existing approaches to prevention and other service provision.

***Parenting behavior and the parent-child relationship.*** Multiple perspectives on child development emphasize the role of parenting behavior and the parent-child relationship in the development of multiple child competencies, including the development of EF and self-regulation. Warm, supportive, involved, and sensitive caregiving in early life, including the preschool and early school years, has been linked to a diversity of positive developmental outcomes. Parents can encourage the internalization of child abilities and strategies important for developing social and cognitive competence. Parents can do this through recognizing the needs of the child and responding accordingly, and by effectively structuring new skills and situations. Research supports a link between the development of better executive self-control and more positive forms of parental control and warmth, sensitivity, and limit setting (Colman, Hardy, Albert, Raffaelli, & Crockett, 2006; Karreman, van Tuijl, van Aken, & Dekovic, 2008; Lengua, Honorado, & Bush, 2007). Meanwhile, negative forms of control, such as harsh, insensitive, rejecting and coercive parenting practices, are related to lower levels of effortful self-control (Colman, et al., 2006; Karreman, et al., 2008; Kochanska, Aksan, Prisco, & Adams, 2008; Kochanska & Knaack, 2003).

Competent parenting has been linked to more positive outcomes among low income and homeless youth, likely through any of a number of complex processes. Interviewer-rated parent-child closeness was related to better academic achievement and teacher-rated school behavior among a small group of African American school children staying in a Minneapolis emergency shelter (Miliotis, Sesma, & Masten, 1999). In a larger sample of formerly homeless families staying in an urban supportive-housing program, Gewirtz, DeGarmo, Plowman, August, and Realmuto (2009) asked parents to complete a 20-minute series of parent-child interaction

tasks analogous to those used in the current study. Interaction sessions were then coded for several dimensions of parenting combined as a latent construct: skill encouragement, positive involvement, problem solving, and inept coercive discipline (reversed). Parenting practices were positively related to parent-rated child adaptation in multiple domains.

***Self-Regulation and Executive Function.*** Cognitive functioning related to developing self-regulation is central to discussions of competence in the context of risk. Self-regulation is an expansive construct that has been pursued in multiple fields of inquiry, including educational science, social and cognitive psychology, neuroscience and neuropsychology, sociology, anthropology, and the developmental sciences more generally, to name just a few. Most broadly, self-regulation refers to an individual's "ability to monitor and modulate cognition, emotion and behavior, to accomplish one's goal and/or to adapt to the cognitive and social demands of specific situations" (p. 256; (Berger, Kofman, Livneh, & Henik, 2007). The processes of self-regulation simultaneously occur across, between and within individual levels of analysis, including genetic and molecular processes, physiological systems, family systems, school relationships and systems, and the child's broader psychosocial and cultural context (Blair & Diamond, 2008; Cicchetti & Tucker, 1994). As such, fruitful investigations of developing self-regulation can involve circumscribed aspects of the phenomenon while maintaining an appreciation of the complex influences from other levels of analysis that contribute to the portion under consideration. Explanatory models that incorporate influences at multiple levels of analysis hold the potential to more fully illuminate the processes of self-regulation.

A particular aspect of self-regulation that has come to the fore is cognitive control (Ochsner & Gross, 2005). Conceptually held apart from more automatic processes of emotional

reactivity, cognitive control is effortful and involves the volitional use of psychological resources to suppress or activate arousal as needed to meet the demands of a situation (Bush, Luu, & Posner, 2000; Posner & Rothbart, 1998). Executive functions (EF) have become of great interest to those looking to understand self-regulation. Executive functions are a group of metacognitive abilities that largely facilitate cognitive control and goal-directed behavior (Luria, 1973; Zelazo, Muller, Frye, & Marcovitch, 2003). EF appears to be a multidimensional construct and investigations of EF typically involve diverse cognitive processes related to planning, working memory capacity and the ability to update representations in working memory, executive attention and attentional shifting, and controlling impulsivity or inhibitory control of prepotent responses (Carlson & Wang, 2007; Liebermann, Giesbrecht, & Muller, 2007; Welsh, Pennington, & Groisser, 1991; Zelazo, et al., 2003). Overlap exists between conceptualizations of EF and higher cognitive functions generally considered (e.g., intelligence). Yet, studies of EF can represent efforts to identify both component and unique cognitive abilities that account for the effects of more superordinate models of general cognitive function or intelligence (Blair, 2006).

Effortful control appears to be particularly important in predicting resilience among children at risk. Effortful control refers to the ability to suppress a dominant response in order to perform a subdominant one (Rothbart, Rueda, & Posner, 2003). Effortful control relies heavily on a subset of EF skills, including executive attention and inhibitory control. Children who are more able to effortfully control their thoughts, feelings, and behavior show fewer adjustment problems in the context of risk compared to others with worse effortful control (Lengua, Bush, Long, Kovacs, & Trancik, 2008). Specifically with regard to children experiencing poverty and/or homelessness, effortful control and EF more generally are important for resilience. Interviewer-

rated self-regulation and executive function skills distinguished resilient from nonresilient children judged on multiple domains who had experienced extremely low income and/or homelessness (Buckner, et al., 2003; Buckner, Mezzacappa, & Beardslee, 2009). Blair and Razza (2007) found that math and literacy ability in low income kindergarteners were predicted by executive functions assessed in preschool, such as attention shifting, working memory, and the ability to inhibit one's behavior. Specifically in kindergarten-aged homeless children, Obradović (2010) used a protocol very similar to the current study to find that effortful control is a salient predictor of teacher-rated academic competence, peer competence, and low levels of externalizing and internalizing symptoms. These findings support the assertion that self-regulation and EF are important predictors of success in low income and homeless children.

***Non-independence of protective factors.*** Positive parenting and good child cognitive functioning are two protective factors that often co-occur. It is unlikely that their positive effects occur in isolation from other protective processes. Using a community sample of 2 to 4 year olds, Lengua, Honorado, and Bush (2007) demonstrated a link between psychosocial risk and lesser gains in children's effortful control skills 6 months later. This relationship was partially mediated by observed parenting behavior in a series of tasks: parents who provided more limit setting and scaffolding had children who made greater gains in effortful control, and differences in parenting partially accounted for the negative impact of risk.

Herbers, Cutuli, Laffavor, Vrieze, Leibel, Obradović, and Masten (2011) provided a rare demonstration of the links between parenting, EF and other cognitive abilities, and academic success among homeless kindergarten-aged children. Although limited by a largely cross-sectional design, these analyses confirmed that higher quality parenting predicted academic

success among homeless children in general, and better parenting was especially salient among homeless children who had experienced a higher level of risk (apart from the protective influences of EF and intellectual functioning). Furthermore, parenting appeared to exert its effect on academics indirectly through links with better EF and intellectual functioning, with the indirect path through EF showing the greatest magnitude. These findings assert that higher quality parenting is important for homeless children's academic success. Parenting seems to operate both indirectly through supporting developing EF skills and directly through protective effects for children at very high levels of risk. Parenting is a key adaptive system, both directly and through structuring and supporting other developing adaptive systems.

**(Some) physiological correlates of EF.** Given that EF is important for resilience, explanatory models of the development of EF that include factors at multiple levels of analysis may help understand how EF operates to protect competent development. They may reveal processes that threaten or support EF in the context of adversity. Executive functions and closely related higher cognitive skills have been linked to the structure and activity of neural networks. Most frequently, executive functions are thought of as abilities correlated with activity in the prefrontal cortex (Best & Miller, 2010), but they are associated with multiple regions of the prefrontal cortex (PFC), anterior cingulate cortex (ACC), and hippocampus. These areas show greater activation during tasks that require effortful regulation of attention and other higher-order cognitive processes among children (Blair, 2006; Blair, Zelazo, & Greenberg, 2005; Bush, et al., 2000; Casey et al., 1997; Lamm, Zelazo, & Lewis, 2006; Rothbart, Sheese, & Posner, 2007). Furthermore, they undergo a relatively protracted development, showing rapid morphological changes and differences in functional organization in early childhood through

adolescence with respect to tasks that require executive functions (Casey, Giedd, & Thomas, 2000; Casey, et al., 1997; Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005).

Evidence from social and cognitive neuroscience suggests a degree of overlap between the neuroanatomy associated with EF and that related to cognitive self-regulation of arousal more generally. The PFC, ACC, and hippocampus are incorporated into a corticolimbic circuit. This circuit involves reciprocal neural interconnections between these structures and the limbic system, which is broadly implicated in arousal, emotion, and some forms of stress reactivity. Activation of the amygdala and other components of the limbic system during negative emotional arousal and stress can have an inhibitory and disruptive effect of prefrontal activation and function. Meanwhile, paradigms that instruct participants to inhibit automatic arousal or to cognitively evaluate stimuli result in increased activation in the PFC and decreased amygdala activity during emotionally salient tasks (Beauregard, Levesque, & Bourgouin, 2001; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Ochsner & Gross, 2005). The ACC also appears to be central in the corticolimbic interface. The ACC functions to resolve conflicts in multiple neural systems, including PFC and limbic circuits, with separate portions involved with emotionally valenced and nonvalenced stimuli (Bush, Luu, & Posner, 2000). The hippocampus also has reciprocal influence with other aspects of the stress response system, including the hypothalamus which plays a central role in the initiation and cessation of the autonomic stress response. The hippocampus contributes to the regulation of the physiological stress response through acting on the hypothalamus. Conversely, suboptimal levels of cortisol, as a downstream endocrine product of hypothalamus-mediated stress response, can interfere with hippocampal function (Lupien & McEwen, 1997). In short, neurological structure and functioning suggests a

reciprocal relationship between arousal, stress response systems and systems implicated in higher cognition and cognitive control.

Cortisol levels may also be important for EF, given links between cortisol and neural functioning in systems that have been correlated with EF. Cortisol impacts other neurological structures in addition to the hypothalamus and pituitary. In the more immediate term, cortisol influences neurological functioning primarily through binding to two types of receptors. Mineralocorticoid receptors (MRs) show a high affinity for cortisol, with approximately 80 to 90% occupation at basal levels. MRs are present in areas of the brain related to modulation of the stress response, including high concentrations in the hippocampus and, to a moderate degree, in the amygdala and paraventricular nucleus of the hypothalamus (PVN). Meanwhile, glucocorticoid receptors (GRs) show a lesser affinity for cortisol, about 10% that of MRs, and tend to be largely occupied only when cortisol exceeds basal levels (e.g., in response to stress). GRs are diffusely expressed across the brain, with particularly dense concentrations in the hippocampus and PVN (Joels & Baram, 2009).

While an in-depth review of the molecular mechanisms of cortisol on neurological functioning are beyond the scope of this paper, cortisol has both genomic and nongenomic effects (through membrane-associated MRs and GRs) on neural systems that carry short and long term implications. Of particular note, MR binding in the relative absence of GR binding (e.g., when at typical basal levels of cortisol and the stress-response system is relatively inactive) has been associated with increased synaptic signal transduction and neuronal excitability. This may be thought of in terms of a 'proactive' regulation and maintenance of homeostasis when stress is not occurring (de Kloet, Oitzl, & Joels, 1999; Prager & Johnson, 2009). Based largely on



work with animal models, MR activation also appears to support behavioral reactivity to novel situations, encouraging investigation and goal-directed exploration (de Kloet, et al., 1999). GR binding appears to have a converse effect, with occupation associated with decreased excitability, decreased responsiveness to other neurological agents, and decreased transcription of a long list of proteins, including those related to synaptic structure and plasticity (de Kloet, et al., 1999; Prager & Johnson, 2009). Because GRs are present in many areas of the brain, these effects may influence functioning in a number of systems. Furthermore, GR binding has also been linked to the consolidation of memories, but this effect may be limited to memories that carry a negative emotional valence (Lupien, et al., 2009).

This past work has resulted in hypotheses about coordinated functioning of MR and GR mediated processes in information processing and learning. Work with humans and animal models have established that a moderate amount of cortisol is optimal for cognitive functioning and either low or high levels have a negative effect, resulting in an inverted-U relationship between cortisol and cognition (Lupien & McEwen, 1997). Moderate levels of cortisol, enough to permit MR but not much GR occupation, would encourage engagement and goal-directed exploration of novel situations. Anticipatory stress and uncertainty in the face of novel situations, however, would increase cortisol beyond basal levels. This would initiate GR-mediated processes as cortisol levels rise, resulting in a constraining of the stress response and, perhaps, encouragement of memory consolidation, all in relation to the amount of cortisol present and degree of GR binding. The end result would be a process of goal-directed engagement as MR receptors become saturated, (presumably slight) activation of the stress-response system, and the inhibition of this arousal coupled with increased consolidation as GR

receptors are increasingly occupied. As such, optimal information processing and learning might involve a moderate increase in cortisol followed by a return to basal levels (de Kloet, et al., 1999).

Many aspects of this hypothesis have yet to be expounded and reconciled with the complexities of MR/GR functioning (e.g., the effects of GR binding seems to be site-specific as it results in increased, not decreased, neuronal excitability in the amygdala; Joels & Baram, 2009). In addition, chronic stress has been linked to differences in neuroanatomy among structures and circuits implicated in executive functions. Children from chronically stressed groups (such as low-income or internationally adopted/post-institutionalized children) routinely underperform on neurocognitive tests that have been linked to the PFC, ACC, and broader executive networks, more so than on most other tasks that measure behaviors subserved by other neurological systems (Hackman & Farah, 2009; Noble, Norman, & Farah, 2005; Pollak et al., 2010). Electroencephalogram (EEG) studies support a link between low-income status and differences in children's and adolescents' prefrontal development and functioning (Hackman & Farah, 2009; Otero, 1997; Tomarken, Dichter, Garber, & Simien, 2004), and studies of event-related potential (ERPs) have linked low-income status to differences in neural activity on executive function tasks (D'Angiulli, Herdman, Stapells, & Hertzman, 2008; Hackman & Farah, 2009; Stevens, Lauinger, & Neville, 2009). Animal models also suggest that experiencing early life stress may result in neuronal death in the PFC and hippocampus/reduced hippocampal volume, reduced MR and GR density in the hippocampus, and differences in neuronal interconnectivity in an enduring way, and that these differences may be mediated by chronically elevated endogenous glucocorticoids (Arabadzisz et al., 2010; Erickson, Drevets, & Schulkin,

2003; Fenoglio, Brunson, & Baram, 2006; McEwen, 1999; Sapolsky, 2000). Findings in humans also support a link between certain types of stress in childhood and reduced hippocampal volumes, but this association may be limited to specific areas of the hippocampus (Wiedenmayer et al., 2006) and may be moderated by psychiatric outcomes (e.g., PTSD) and developmental changes over time (Carrion, Weems, & Reiss, 2007), making causal inferences more elusive. Nevertheless, chronically dysregulated levels of glucocorticoids may contribute to these differences (Carrion, et al., 2007; McEwen, 1999; Sapolsky, 2000), especially if occurring during preschool and early life when these areas are rapidly developing (Gunnar & Quevedo, 2007). Therefore, cortisol levels may play a role in EF in the short term through influencing neuronal signaling, as well as produce lasting differences in the neurophysiology associated with executive functions.

***Cortisol and EF in Preschool and the Early School Years.*** Several studies have investigated the links between cortisol and EF among preschoolers and children in the early school years. Blair and colleagues (Blair, Granger, et al., 2005; Willoughby, Vandergrift, Blair, & Granger, 2007) have reported findings on cortisol functioning among low income preschoolers during a session of cognitive tasks. Children who showed an ‘up-then-down’ pattern of cortisol during the session had better EF skills, better self-regulation, and better functioning at school. This approach emphasizes MR versus GR occupation effects on information-processing (see above), where optimal cognitive functioning involves a moderate increase of cortisol, resulting in an inverted-U type relationship between cortisol levels and cognitive functioning.

Similarly, Davis, Bruce, and Gunnar (2002) found that higher levels of cortisol during the day and during a testing session were each related to better performance on tasks involving

executive attention subserved by the ACC and other anterior attention networks among 6 year old children. However, there were no effects of performance on slope for cortisol trajectories during the session. Davis and colleagues assert that their participants' cortisol levels likely fall to the 'left of the inverted-U' that appears to define the relationship between cortisol and cognition, with the implication that higher cortisol would be facilitative in this group to a point.

In contrast to the above, work with young homeless children has revealed links between better performance on a series of executive function tasks and *lower* cortisol levels during the assessment session among 4 to 7 year olds (Wiik, et al., under review). This finding is consonant with the view that the cortisol-cognition relationship takes the form of an inverted-U. Since basal cortisol levels tend to be higher among individuals who are experiencing or have recently experienced stressful life events (Miller, Chen, & Zhou, 2007), children in emergency shelter may have higher cortisol levels that push them to the "right-of-center" when considering the inverted-u relationship, creating a negative relationship between cortisol and executive functions.

**Summary.**

Homelessness represents a context of risk for key developmental outcomes. This risk likely involves sociodemographic and other psychosocial factors associated with poverty generally (e.g., a lack of physical resources), as well as specific adversities that are more common among low income and homeless groups and that threaten family relationships and functioning. In addition, positive parenting behaviors and good executive function skills appear to buffer the negative effects of risk and adversity and allow many children to show resilience.

Experiencing stressful negative events and circumstances early in life has been linked to differences in cortisol. In the short term, individuals who have a recent stressful experience show relatively elevated cortisol levels that tend to decline over time. Chronic or repeated stressful circumstances have the potential to alter cortisol functioning in more lasting ways. This seems to be especially true for children who experience poor caregiving environments and other threats to family functioning. It remains unclear if and how the varieties of parenting behavior (e.g., positive parenting, negative control) impact developing cortisol functioning in the context of ongoing adversity, especially given the links between parenting and both EF and broader self-regulation.

Cortisol levels appear to influence and be influenced by children's executive functioning and related cognitive self-regulation skills. This reciprocal relationship likely operates through multiple processes, some of which are in an acute timeframe (e.g., increased excitability of neuronal signaling associated with MR occupation; decreased excitability associated with GR occupation; activation of inhibitory neural pathways to amygdala and HPA-axis from the PFC and ACC associated with EF) and some of which seem to reflect processes that are more chronic or slower to manifest (e.g., structural and functional differences in PFC and other neural networks/areas associated with both EF and other stress response systems thought to be influenced by repeated activation of neural pathways and/or chronic or repeated exposure to endogenous glucocorticoids). While multiple contributing processes are likely, past work has highlighted the facilitative effects of MR occupation on neuronal firing versus the dampening effects of GR occupation when cortisol is present at higher levels. This has resulted in an explanation that touts an 'inverted-U' relationship between cortisol and cognition/EF, such that

optimal levels of cortisol involve a moderate elevation past basal levels in most cases that would maximize MR occupation while minimizing GR occupation.

This project seeks to further explore possible links between EF, cortisol, parenting, and adversity among children staying in emergency shelter. These factors represent three levels of analysis that appear relevant to adaptation in the context of risk, especially with respect to competence in the school setting. I take a perspective that draws on ecological theory (Bronfenbrenner, 1979), developmental theory applied to the study of risk and resilience (e.g., Yates, et al., 2003)), and a multiple-levels-of-analysis approach that has been influenced by multiple frameworks and theories (e.g., behavioral epigenesis (Gottesman & Shields, 1982); experiential canalization (Gottlieb, 1991); developmental psychopathology (Cicchetti, 1993); developmental tasks/competence (Masten, Burt, & Coatsworth, 2006)).

#### **The Present Study: Aims and Hypotheses.**

This study examines how dynamic cortisol levels relate to adversity, parenting, and executive function in a sample of children living with their families in an emergency shelter during the summers of 2008 and 2009 who were entering kindergarten or first grade. The goal is to elucidate explanatory models of the processes of risk and resilience by incorporating relationships with cortisol, a component of physiological adaptive systems related to the stress response, self-regulation, and other functions. Children were separated from caregivers and completed a session of cognitive tasks that assessed executive functions and other abilities, followed by a session of parent-child interaction tasks with their primary caregivers. Saliva samples were collected throughout both sessions and assayed for cortisol concentrations. Parents reported on risk factors and stressful negative life events for each child. The study had

four primary aims related to further establishing the validity of salivary cortisol data in the ecologically-valid context of an emergency shelter and testing hypotheses regarding the links between cortisol, adversity, parenting, and executive function:

**Aim 1: Measurement of Salivary Cortisol in Shelter.** The shelter context provides a number of challenges to successful data collection and may raise concern about the quality of this cortisol data collected in an ecologically valid context and “outside of the laboratory.” Potential concerns include characteristics of the setting or population, such as increased potential for psychosocial conflict related to crowding, high rates of illness, high rates of asthma (Cutuli, Herbers, Rinaldi, Masten, & Oberg, 2010) and possibly higher rates of medications that contain glucocorticoids, the possibility of high rates of other medications that impact the functioning of the HPA-axis, missing data because of family or child refusal, missing data because families needed to end sessions early to adhere to shelter schedules, or low-quality samples because researchers do not have the same level of control as would be possible in a laboratory setting. Based on past success collecting this sort of data in shelter contexts, I expected low rates of these potential confounds (Cutuli, Wiik, et al., 2010). Descriptive statistics are provided to evaluate the success of cortisol assessments and the likely validity of these concerns.

**Aim 2: Relationship between EF and Cortisol.** I expected to replicate past work with low-income children linking cortisol function with EF skills. I expected that EF skills would be negatively related to cortisol intercept: children with better EF skills would show a lower level of cortisol at the start of the session. Furthermore, I expected no difference in change in cortisol (slope/trajectory effects) over the course of the session. This hypothesis follows from past work

with 4 to 7 year old children experiencing homelessness (Wiik, et al., under review). The current study involved a larger and more representative sample of homeless children with more data points that may yield more reliable effects.

This hypothesis is consonant with accounts of physiological functioning related to saturation of MR and mild occupation of GR receptors with cortisol in neural structures related to EF, one proposed influence driving the inverted-U relationship between cortisol and cognitive functioning. Other work with similarly aged children has found a positive association between cortisol levels and executive functions, where higher cortisol is linked to better cognitive functioning (Blair, Granger, et al., 2005; Davis, et al., 2002; Willoughby, et al., 2007). However, those studies involve groups of children who were not selected on the basis of experiencing acute or ongoing adversity. Individuals who experience recent or ongoing stressful life events (like homelessness) evidence elevated basal levels of cortisol (Miller, et al., 2007). As such, it is reasonable to believe that children experiencing homelessness are more likely to be right-of-center when considering the inverted-U relationship between cortisol and cognitive functioning, an area of the curve where a negative relationship would exist. As such, my hypothesis of a negative relationship between cortisol and executive functioning for these children is congruent with the inverted-U account as well as past literature finding a positive relationship among young children who were not experiencing ongoing stressful life events.

It should also be noted that Blair and colleagues (Blair, Granger, et al., 2005) demonstrated an elevation followed by a rapid decline among children who had better EF skills during a session of cognitive tasks. They conceptualized this pattern as a faster recovery from the stressful experience of meeting a new adult after having been in the more-familiar setting of



the child's preschool class, unaware that they were going to participate in the study that day. Due to methodological differences, I did not expect to see this increase because all of the participants in the current study were likely experiencing anticipatory stress that had increased cortisol levels prior to the first measure of cortisol (Gunnar, Talge, & Herrera, 2009): each child would have been told that they would be participating that day and would have been interacting with a novel adult for about 10 to 20 minutes prior to the first cortisol measure while their parent/guardian completed the consent process.

**Aim 3: Relationship between Cortisol, Adversity and Parenting Behavior.** The third aim involved relationships between children's adversity histories, current contexts and cortisol function during the session of cognitive tasks. In line with past work (Cutuli, Wiik, et al., 2010) and a developmental-ecological perspective, I expected that higher levels of cumulative adversity across the lifetime, reflected in the number of negative lifetime events, would predict higher initial levels of cortisol and greater declines throughout the session, beyond the effects of a cumulative risk indicator of low levels of physical resources. Then I tested whether lifetime events related to family functioning and family-level disruptions predicted these differences beyond lifetime events related to more direct threats to the individual child (e.g., near drowning, being in an auto accident). I predicted that the effects of lifetime events scores would be largely accounted for by family-level events.

I then tested for intercept and slope/trajectory effects of observed parenting under the hypothesis that children with parents who show more positive behaviors (sensitive, warm, and structured caregiving practices) would have more rapid declines in cortisol over the course of the session of cognitive tasks. Meanwhile, children whose parents show higher levels of

negative control (harsh, rejecting, hostile practices) would demonstrate higher initial levels of cortisol and declines that are less steep. It is important to note that parenting is multidimensional, with behaviors clustering into relatively independent constructs. When parenting is coded from observed behaviors, such as harshness/hostility and warmth/positive emotionality, uncorrelated or distinct dimensions have emerged repeatedly in past research (Alink et al., 2009; Calkins, Smith, Gill, & Johnson, 1998; Lengua, et al., 2007). It was neither assumed that positive parenting and negative control represent behaviors on a single continuum, nor that they would be negatively correlated in these data. I expected positive parenting and negative control to be largely unrelated. This is in line with a multidimensional view of parenting behavior.

**Aim 4: Child Cortisol during Parent-Child Interaction Tasks.** I explored a set of qualified hypotheses related to the children's cortisol responses to a session of parent-child interaction tasks. Consistent with work linking harsh and insensitive parenting with dysregulated cortisol, and work on the effects of positive parenting behaviors attuning developing physiological systems, I expected a more pronounced increase in cortisol for children who experienced less warm, sensitive, and structured caregiving, and for children who experienced more negative control. I also expected a moderating effect of child EF and positive parenting on cortisol trajectories. There should be little difference in child cortisol response in the context of warm, sensitive, and supportive caregiving. However, children with low EF who experienced lower positive parenting would have a more pronounced increase in cortisol during the session compared to children with high EF and parents who provide less positive control. This last hypothesis must be qualified by acknowledging that the parent-child interaction session almost

always immediately followed the cognitive-task session, and relationships between child cortisol levels and trajectories may include a residual association with EF skills as a function of the proximity of the two sessions.

### **Methods.**

This project was possible because of a close partnership between University researchers, local shelter providers, and area school districts. Data were collected in two waves during the summers of 2008 and 2009. During 2008, participants were recruited from two large emergency shelters for families. These two shelters serve the majority of sheltered families in Minneapolis. A third, smaller family shelter was added in the second wave. Approximately 60% of students identified as homeless or highly mobile in the local school district are identified while staying at one of these three shelters.

Eligible families had a child between the ages of 4 and 7 years who would be able to enter kindergarten or first grade during the following school year and had never previously attended first grade. This age and grade range was selected given the study's emphasis on school readiness and success in the early school years. Parents and children had to speak English proficiently to engage in the informed consent process and/or be able to complete the study tasks. Children were ineligible if they had previously been diagnosed with a developmental disorder, disability, or other impairment that would prevent them from completing the protocol. Children were also excluded if they had participated in this protocol during a previous year. Only one child was recruited per family. When there was more than one eligible child in a family, any child entering kindergarten was preferred, otherwise the target child was chosen randomly. Families were scheduled to participate after their third night in shelter to allow parents and

children some time to adjust to the shelter context. Approximately 72.25% of all families suspected to be eligible agreed to participate, resulting in an initial sample of 138 families. Additional rates of non-participation and exclusion are provided in Appendix A.

The original target sample size for the study was 140 and 140 families participated. In one case, however, we later discovered that the child was too young to attend kindergarten and in a second, the child was later determined to be entering 2<sup>nd</sup> rather than first grade. These two cases were excluded, resulting in a sample size of 138 children and their primary caregivers. Of these, 128 (92.75%) of primary caregivers were the child's biological mother; 5 (3.62%) the biological father; 2 (1.45%) the stepmother; 2 (1.45%) the stepfather; and 1 (0.72%) the child's grandmother. Herein I use the term 'parent' generally to refer to the child's primary caregiver, acknowledging this diversity in the children's family structures.

### **Procedures.**

Children and their parents completed two assessment sessions, usually on a single day. All data were collected in specially designated rooms in each emergency shelter. Almost all sessions occurred either in the morning (between 9 am to 12 pm) or afternoon (12 to 5 pm). Sessions were scheduled back-to-back, resulting in a 2-hour participation period for most families. The first assessment session followed an approximately 10 to 20 minute informed consent process during which the target child casually and calmly interacted with the experimenter in the presence of his or her parent and family. Following consent, the child and parent were separated and completed independent assessment sessions: Children were administered a number of developmentally appropriate cognitive tasks while parents were interviewed. This first session lasted between 60 and 90 minutes. Following the first assessment session, the child

and parent were reunited to complete a series of parent-child interaction tasks together for about an hour. Parents received \$40 in gift cards as an honorarium for their time and children received toys valued around \$10.

**Child Session of Cognitive Tasks.** In the first session, children were administered a series of cognitive tasks. Past work has shown that these tasks can serve as a context to examine cortisol trajectories related to differences in executive function skills in low-income and homeless preschoolers (Blair, Granger, et al., 2005; Cutuli, Wiik, et al., 2010; Willoughby, et al., 2007). Children completed the following tasks in the following order: Block Design and Matrix Reasoning subscales of the Wechsler Preschool and Primary Scale of Intelligence – Third Edition (WPPSI-3; (Wechsler, 2002), the Peabody Picture Vocabulary Test – Fourth Edition (PPVT-4; (Dunn & Dunn, 2007); Simon Says (SS; (Kochanska, Murray, & Coy, 1997); Dimensional Change Card Sort (DCCS; (Frye, Zelazo, & Palfai, 1995); Dinky Toys (Kochanska, et al., 1997); Peg tapping (Diamond & Taylor, 1996); the Computerized Pointing Stroop Task (Stroop; (Berger, Jones, Rothbart, & Posner, 2000), and Gift Delay (Kochanska, et al., 1997). All child tasks in this first session were completed one-on-one with a research assistant outside of the parent’s presence. Each research assistant was specially trained in psychological traumatology and development to be attuned to the child’s needs and reactions during the session.

**Child Executive Function.** Child executive functioning was represented by a composite of four executive function tasks that have shown good construct validity as an index of EF with children of the same age staying in homeless shelters (Obradović, 2007, 2010): SS, DCCS, Peg Tapping, and Stroop.

*Simon Says (SS)*. The Simon Says Task required children to follow the experimenter's instructions and imitate her behavior on each of 10 directions preceded by the phrase 'Simon Says' (activation trials) while inhibiting all behavior during 10 directions not preceded by 'Simon Says' (inhibition trials) (Strommen, 1973). All children received standardized instructions on the task followed by several practice trials. Then, activation and inhibition trials were presented in an intermixed and standardized order. This task was video recorded and later coded by trained research assistants blind to children's performance on other tasks. Interrater reliability was excellent (weighted  $K = 0.95$ ) based on 50 cases (36.2% of the sample). Scores were calculated as a percentage based on the child's overall performance on inhibition trials. To help ensure that the task largely indexed children's inhibitory control, executive attention and associated executive functions, scores were not computed for children who did not succeed in any inhibition practice or test trial, or did not earn at least a 50% accuracy score on activation trials. This minimized the likelihood that scores were unduly influenced by child misunderstanding or by overwhelming and consistent behavioral inhibition.

*Dimensional Change Card Sort (DCCS)*. The Dimensional Change Card Sort was presented as a tabletop task (Frye, et al., 1995; Zelazo, 2006). All cards depicted either a red rabbit or a blue boat. First, children were instructed in the rules of the "color game" and they completed 6 'pre-switch' trials where they were expected to match cards based on color: placing all cards showing a red rabbit in a bin labeled by a red boat and all blue boats in a bin labeled with a blue rabbit. Next, the experimenter created a switch in the rules by instructing the child in the 'shape game' wherein the child was to now match the cards based on shape: red rabbits cards go in the bin labeled with a blue rabbit, and blue boat cards into the red boat bin. This post-switch

condition contained six trials. Child scores were computed based on the number of correct post-switch trials. To help ensure that scores reflect the child's ability to switch, maintain, and follow rule-sets, children who failed the pre-switch trials ( $< 70\%$  correct) were not given a post-switch score.

*Peg Tapping.* In the Peg Tapping Task, the child was taught two rules for tapping a small wooden dowel after the experimenter for each trial. The child was instructed to tap two times on trials when the experimenter taps once, and one time on trials when the experimenter taps twice. Success on this task required children to inhibit the prepotent response of imitating the experimenter's actions, as well as the ability to remember and follow these rules. The experimenter presented 16 counterbalanced test trials. The children received scores based on the number of correct responses.

*Computerized Pointing Stroop.* This was a computer administered task (Berger, et al., 2000). Similar to the DCCS, this task required the children to be able to switch between, maintain, and demonstrate the ability to inhibit a prepotent response to follow two different sets of rules. First the children were trained in rules for both congruent trials and then incongruent trials ('the silly game'). In each congruent trial, two different animals were presented on the computer screen and an animal sound was played. Children were instructed to select the animal that made the sound that they heard. For incongruent trials, children were trained to select not the animal that matches the sound, but the other animal. All responses were made by children touching a touchscreen monitor, and experimenters also trained all children in recognizing the animals and their accompanying sounds prior to any test trials. After training the child on the rules of both conditions, the experimenter instructed the child that she

or he would first complete the congruent trials, reminded her or him of the rules, and then completed 16 congruent trials. Then the children were told that they would play 'the silly game,' they were reminded of the rules, and then presented with 16 incongruent trials. Children were given scores based on the number of correct incongruent trials.

I formed the EF composite by standardizing scores for each individual task through a z-transformation and then taking the average of all available scores. This produced a composite with an alpha = 0.71. Two tasks were not included in the composite, Dinky Toys and Gift Delay, because (1) these tasks differ in that they conceptually involve the activation of emotion and reward systems, (2) EF that involves emotion has been linked with distinct neural systems (Bush, et al., 2000), and (3) these particular tasks have shown a distinct pattern of association that contrasts with the four selected EF tasks (Obradović, 2007). Similarly, WPPSI-3 subscales and PPVT-3 scores were conceptualized as estimates of nonverbal and verbal intelligence, respectively, and not included in the EF composite. There is typically overlap between IQ and EF in both construct space and empirical findings. However, the focus of the current investigation is on EF as a component of cognitive control, and not cognitive functioning more generally. Furthermore cortisol has previously been linked to EF apart from more general indicators of cognitive functioning (Blair, Granger, et al., 2005).

**Parent Interview.** While children were completing the session of cognitive tasks, parents were interviewed by a second research assistant also trained to be sensitive to the context of homelessness and psychological sequelae of psychosocial trauma. Parents reported on child behavior and health, family characteristics, cumulative socioeconomic risk, negative lifetime event histories, and characteristics of the child's situation that may act as potential



confounds of cortisol function. All measures were administered as an interview to circumvent many problems related to poor literacy. Parents completed the following measures relevant to the current study:

***Cumulative socioeconomic risk.*** Parents reported on the presence or absence of five demographic or otherwise poverty-related risks in the physical environment: parent unemployed, parent with less than a high school education, having lived in an unsafe neighborhood at last residence, not able to pay rent at last residence, and experienced substandard or unsafe housing at last residence. Each child was assigned a cumulative socioeconomic risk score by summing the number of risk factors present (Evans, 2004; Sameroff, Gutman, & Peck, 2003; Sameroff, Seifer, Zax, & Barocas, 1987). These particular risk factors were selected to index the family's level of risk with respect to physical resources and human capital, with an attempt to avoid risk factors related to disruptions and disturbances in family functioning. This allowed for a comparison of cumulative socioeconomic risk versus negative lifetime events (see below).

***Negative lifetime events.*** Lifetime event scores were sums of the responses on the Lifetime Life Events Questionnaire (Masten, et al., 1993) completed by the parent. This questionnaire elicits the number of stressful events that the child experienced during his or her lifetime, including events and situations that threaten the child directly as well as those experienced by the child that threaten a parent, a family member, or the integrity of the family. A sum-total score was computed that includes the overall number of events endorsed. Two component sum scores were computed to reflect the number of experienced events that threaten the family/family functioning (e.g., witnessing violence happening to a parent) and to

reflect the number of experienced events that were direct threats to the child (e.g., nearly drowned), respectively. Total negative lifetime event scores and scores for family-related events were each truncated at about the 95% mark to reduce the influence of a few outliers who experienced an even more disproportionate number of negative events. This corresponded to an artificial maximum of 8 being set for total lifetime event scores, and 4 for family-related events.

***Potential confounds of child cortisol.*** Parents completed a brief checklist that asked about daily factors that may have influenced a child's cortisol levels, namely whether the child was sick, used an inhaler medication in the past 24 hours, was using other medication, was particularly stressed or unusually active that day, or had argued with a parent or sibling. The following potential confounds were also recorded when saliva samples were collected and assayed: whether the child rinsed his/her mouth recently, more than one sample needed to be collected at one time to ensure sufficient volume, the child was showing strong affect (e.g., crying), and whether the sample was discolored, contained debris, or was low in volume.

**Parent-Child Interaction Session.** Following the independent parent interview and child cognitive sessions, the child and parent were reunited and completed a series of eight interaction tasks together, including free play, clean-up, two problem-solving discussions, a safety plan discussion and three game tasks (Labyrinth, Guessing Game, and Tangoes). Tasks were presented in the same order for all parent-child dyads. All tasks were video recorded and coded later for content.

These tasks were adapted from the *Family Interaction Tasks* for use with homeless and formerly homeless families (Gewirtz, et al., 2009). These tasks were designed to observe

parenting behavior in a variety of situations ranging from unstructured to highly structured. The tasks were presented in the same order listed below:

**Free Play.** The child and parent were placed in a room that contained two shelves of toys. Prior to being reunited with the child, the research assistant explained to the parent that during the first task s/he was to enforce a rule that the child was not to play with the top shelf of toys, and only the bottom shelf was permitted. The top shelf contained more attractive toys. No other instructions or structure was provided from the research assistant to the dyad other than the statement, "Take a break and talk or play with some of the toys." The parent and child were then left alone for five minutes.

**Clean-up.** Following Free Play, the research assistant reentered the room to give the parent a magazine to read. Prior to the parent-child interaction session, the parent had been informed that at this point s/he would instruct the child to clean up the toys. There was also a small note attached to the front of the magazine reminding parents to have their children clean-up. The parent and child were again left alone for five minutes.

**Discuss an issue 1 and 2.** Next the parent and child were instructed to discuss an issue for five minutes that the parent had previously identified as relevant to the dyad with the goal of coming to a solution. Common issues involved trouble with child compliance in a number of situations, like cleaning up, arguing with siblings, or following other family rules and avoiding bad behavior. After five minutes, the research assistant instructed the parent and child to discuss a different issue previously identified by the child with the goal of coming to a solution. Common issues were similar to those selected by parents and included cleaning up toys, fighting with brothers and sisters, and being allowed more time to do favorite activities. Parents had

identified two potential issues for discussion to prevent the same issue from being discussed twice. This second discussion also lasted five minutes. Both discussions occurred outside the presence of the experimenter.

***Labyrinth.*** Following the discussions, the research assistant presented the dyad with the first game task. For three minutes the dyad first worked together and then in competition to move marbles into holes on a wooden board.

***Safety Plan.*** The sixth task involved a discussion of a safety issue relevant to the child that had been previously identified by the parent. Common issues involved strangers, bullies, and getting lost. This discussion lasted five minutes and the research assistant was not present.

***Guessing Game.*** The seventh task was another game in which the parent and child took turns trying to get the other to guess pictures on a series of cards. This game lasted six minutes.

***Tangoes.*** The final task was also a game wherein the dyad attempted to use geometric shape pieces from the commercial game *Tangoes* to recreate specific shapes shown on cards. The parent was instructed to assist the child. This task lasted four minutes.

Coders reviewed the video recordings. All coders were familiar with this methodology and experienced working with homeless and low income families. The coders were blind to child performance on cognitive tasks and all information collected from parents. 130 participants completed at least half of the interaction tasks and were coded. This was a microcoding scheme in which each second of parent behavior was classified into one of four mutually exclusive categories: 'Positive control,' 'Non-directive responsiveness,' 'Disengaged/distracted,' and 'Negative control.' Specifically, 'Positive control' occurred when parents provided constructive strategies to regulate the child's behavior or affect while maintaining positive or neutral affect.

Some examples of positive control are giving instructions or giving requests, providing explanations, teaching, redirecting the child to on-task behavior, distracting the child from upsetting stimuli, and comforting the child in a role-appropriate way. 'Non-directive responsiveness' refers to involved, responsive parenting that is also nondirective and is done with a positive or neutral affect, such as monitoring the child's behavior, recognizing on-task behavior, and briefly repeating or responding child verbalizations in the context of acknowledgment. 'Negative control' refers to behaviors intended to control the child in a harsh, punitive, insensitive, or intrusive way. Many different behaviors compose the 'Negative control' category, such as yelling or shouting, threats, criticism, shaming, insensitive invalidation of the child's point of view, interrupting, insults, negative questions, teasing or taunting, aggressive play, physical aggression, or harsh physical contact/discipline. 'Disengaged/Distracted' is not included in the current analyses, but involves parent behaviors that are neither directive nor focused on the child and task, such as being distracted by something else (e.g., cell phone), looking away or 'zoning out'/dissociating, attending to the experimenter or objects in the room, or pursuing a topic that is entirely parent-focused.

The metric of these codes is the number of times the parent initiates each type of behavior, divided by the total duration of the interaction tasks to control for sessions that varied in length. A subset of 40 (30.77%) interaction sessions were coded by a second trained coder to produce indicators of good reliability (observer accuracy > 0.90 for all four parent codes corrected for observed base rates (Grove, Andreasen, McDonald-Scott, Keller, & Shapiro, 1981). Other analyses of these data have demonstrated the relationship between the total duration of parent control (combined Positive and Negative) and child executive functioning, and between

Non-directive responsiveness and later child outcomes in school (Herbers, 2011). The current analyses are interested in positive parenting and negative control. As such, 'Positive control' and 'Non-directive responsiveness' were averaged to produce an indication of parent warmth, structure, support, limit setting, and responsiveness. The 'Negative control' classification was used to reflect negative control.

**Child Cortisol.** Child cortisol was assessed through saliva. Children provided a maximum of 8 saliva samples during the session of cognitive tasks and during the parent-child interaction. The first sample was collected immediately following the informed consent process and subsequent samples followed at approximately 20 minute intervals. Twenty minute intervals were chosen because HPA axis activation eventuates with salivary cortisol secretion between 5 and 20 minutes later (Kirschbaum & Hellhammer, 2000). A maximum of four samples were collected during the session of cognitive tasks and four during the interaction session.

Saliva was obtained by having the child dip a 1.5" cotton dental roll in about 0.025 g of cherry flavored Kool-Aid™ mix before placing the cotton roll into her or his mouth. The use of this small amount of Kool-Aid™ mix increases salivation and the appeal of the task, but does not meaningfully affect the cortisol assay or the rank-ordering of cortisol values (Talge, Donzella, Kryzer, Gierens, & Gunnar, 2005). The child held the cotton roll in her/his mouth until it became saturated. A needleless syringe was used to express the saliva into a 1.5 ml Eppendorf Safe-Lock microtube, which was then frozen at -20C until assaying.

Samples were assayed in duplicate for cortisol concentration with a time-resolved fluorescence immunoassay (DELFIA). Duplicates were highly correlated ( $r = 0.997$ ). Mean intraassay coefficient of variation was 5.1% (SD: 4.27%) for the samples in this study. The

intraassay coefficient of variation for this assay was between 4.0% and 6.7%, while the interassay coefficient of variation was between 7.1% and 9.0%.

**Missing Data.** The realities of collecting data in emergency shelters with children and families actively in crisis resulted in a degree of missing data. Reasons for missing data varied, most frequently because of child refusal or high levels of distress, and families needing to end early for another appointment or to attend meals that were only offered during certain times in one of the shelters. Sixteen cortisol values were not considered plausible and not included in analyses because they exceeded 3 standard deviations above the mean for the entire sample. The plausibility of cortisol values was also considered in relation to each individual's mean cortisol levels. Plausible cortisol values were those within 300% of the next highest value for that child or within 3 standard deviations from the child's mean level of cortisol. No additional values were identified as implausible in this way. Data are assumed to be missing at random (MAR; (Schafer & Graham, 2002)). The analytic approach estimated missing values on the dependant variable using maximum likelihood estimation (see below), and these cases were included in analyses so long as they had all independent variables and at least one relevant cortisol observation. However, cases with missing data on any independent variable were excluded from that block of analyses. This resulted in different sample sizes for blocks of models: EF and cortisol during the session of cognitive tasks (n = 133); parenting and cortisol during the session of cognitive tasks (n = 126); and parenting, EF, and cortisol during the parent-child interaction session (n = 127). Additional details regarding rates of missingness are presented in Appendix B.

**Analytic Approach.**

Analyses proceeded in multiple steps. First, I analyzed descriptive data to uncover systematic differences in cortisol levels by year. Differences of  $|3|$  or more standard deviation units (Cohen's  $d$ ) were considered meaningful (Cohen, 1988). There were no meaningful differences in cortisol between the two major sites. Mean-levels of cortisol were higher in 2009 compared to 2008. As such, year of data collection was covaried in all analyses involving cortisol. Means are reported in Appendix C. Between-year differences should also be noted for EF, age, and negative parenting. However, controlling for year does not appear to impact the predictive power of those variables on cortisol. See Appendix D.

Second, I calculated correlations between cortisol values and each potential confound thought to influence cortisol (e.g., child taking medication). None of these factors had a sizeable association with cortisol (all  $r$ 's  $< |0.25|$ ). None of these factors appear to have a confounding effect on cortisol and, therefore, none will be included as covariates in analyses. See Appendix E. Due to established age and sex differences regarding cortisol function, both of these variables were covaried as statistical controls in all models with respect to intercept and slope/trajectory. Since cortisol declines throughout the day, and time of day affects reactivity, the time of the session was controlled with respect to intercept and slope/trajectory for all analyses. Correlation coefficients for variables used in analyses are provided in Table 1.

I tested hypotheses using a latent growth modeling/linear mixed modeling (LMM) approach appropriate for repeated measures data correlated within individuals (Fitzmaurice, Laird, & Ware, 2004). Linear mixed modeling allows for the testing of effects on initial level (intercept) and differences in change over time (slope or trajectory). Change over time can be estimated in different ways, such as linear change, higher-order polynomial change (e.g.,



quadratic trends), and log-transformation of the time metric to produce a trajectory that can estimate linear and simple non-linear rates of change (Long & Ryoo, 2010). Furthermore, linear mixed modeling accommodates missing data on the dependent variable through maximum likelihood estimation. In addition to fixed effects, each LMM also included random effects for intercept and slope/trajectory based on a priori expectations.

I expected a quadratic rate of change to be appropriate for the data, given the patterns found by Blair and colleagues (Blair, Granger, et al., 2005), among others. Nevertheless, I compared models using three different estimations of change over time: a quadratic trend, linear slope, and a log-transformation of the time metric. This means that the selection of the function of the rate of change metric was data driven (among these three possibilities). For the time metric, samples were divided into one of nine 10-minute intervals based on when they were collected in the session of cognitive tasks, or one of eight 10-minute intervals for the parent-child interaction session. Consistent with other work, the distributions of cortisol values were skewed within most intervals and required a log<sub>10</sub> transformation to approximate normality and better meet the assumptions of linear mixed modeling.

I evaluated meaningful contributions to model fit ('significant results') by comparing model fit via the Akaike's information criterion (AIC) value of models and the related weight of evidence with and without each fixed effect of interest. P-values/Traditional null hypothesis testing were not used in interpretation, given the non-consensus that surrounds reliable means of estimating degrees of freedom for multilevel models (Baayen, Davidson, & Bates, 2008), in addition to longstanding thought on the shortcomings of traditional null-hypothesis testing based on p-values (Anderson, 2008; Kullback & Leibler, 1951; Meehl, 1997). I calculated the

weight of evidence to reflect the relative goodness of fit between models. The weight of evidence is a probability (scaled 0 to 1) that denotes the fit of any one model relative to the entire set of models considered in any analysis. The sum of all weights for a set of models equals 1. The weight of evidence is computed by the following equation, where  $W_k$  is the weight for the  $k$ th model,  $L$  is the total number of models considered, and  $\Delta_k = AIC_k - AIC_{min}$ , with  $AIC_{min}$  being the minimum AIC in the set:

$$W_k = \frac{\exp(-.5 \cdot \Delta_k)}{\sum_l^L \exp(-.5 \cdot \Delta_l)} \quad (1)$$

In addition to weights, changes in AIC were considered when determining meaningful contributions to model fit. The model with the lowest AIC is the best fitting. Consistent with Burnham and Anderson (2004), models with a change in AIC of less than 2 were considered equivalent. When this occurred for the best fitting model, the more parsimonious model was adopted as the best fitting. It should be noted that relative model fit is less clear when the difference in AIC is between 2 and 10, while there are clear differences in model fit when there is a change in AIC changes of 10 or more (Burnham & Anderson, 2004).

## Results

Results are presented in four sections, organized by study aims. Analyses testing hypotheses are described, along with follow-up analyses to clarify results or explore additional models in a post-hoc fashion. Additional information is provided in appendices, where relevant.

### **Aim 1: Measurement of Salivary Cortisol in Shelter.**

Descriptive statistics are provided to evaluate the feasibility and validity of salivary cortisol data collected from young children staying in the ecologically valid context of an emergency

homeless shelter for families. Only 16 (1.45%) saliva samples produced cortisol concentration values that were deemed implausible. Appendix B provides further rates of missing data for cortisol values. Only three children failed to contribute any cortisol data to analyses. These were because of child refusal (2 children) or all implausible values obtained (1 child). Two additional children did not provide any cortisol data for the parent-child interaction session. Fewer samples were observed for the parent-child interaction session (which always occurred after the session of cognitive tasks), but high rates of observed data were obtained for both sessions: 91.76% and 87.86%, respectively.

Appendix E lists endorsement rates for potential confounds of cortisol function. Most confounds related to individual participants were endorsed by < 20% of participants. The exception was the number of participants who were in an argument with a parent or sibling earlier in the day (57%). Cortisol values were not meaningfully different comparing those who endorsed these confounds versus those who did not (maximum  $r$ 's for each cortisol sample < 0.20). At the level of individual samples, very few were identified by researchers as having some sort of concerning characteristic, and the presence of most any concern seemed unrelated to cortisol values (maximum  $r$ 's < 0.20). Samples that contained debris were the exception, as these samples had slightly higher levels of cortisol (maximum  $r$ 's = 0.24).

### **Aim 2: Relationship between EF and Cortisol.**

The following analyses tested the overall hypothesis that there would be an association between cortisol and EF. Nine models were compared to determine (1) the best estimation of the cortisol pattern of change in the session of cognitive tasks (linear, quadratic, or logarithmic), and (2) evaluate a relationship of EF with initial cortisol levels (intercept) and with rate of

change (slope/trajectory). Relevant model fit statistics are provided in Table 2. A log-transformation of the time metric produced a relatively poor fit for the data and will be excluded from further groups of models that consider cortisol during the session of cognitive tasks. The best fitting model involved a quadratic trend to denote change in cortisol, and contained a fixed effect for EF on intercept ( $t = -2.34$ ) but not on trajectory (weight = 0.61). This model is adopted and coefficients are provided in Table 3. The adopted model had somewhat better fit than the model with a fixed effect of EF on quadratic trajectory ( $\Delta AIC = 3.2$ ; weight = 0.13) and one without any fixed effects of EF ( $\Delta AIC = 3.3$ ; weight = 0.12). These two alternative models should be considered in future work. The adopted model suggests that children with higher levels of EF have lower levels of cortisol at the start of the session of cognitive tasks, and this relationship persists over the course of the session. See Figure 1.

### **Aim 3: Relationship between Cortisol, Adversity and Parenting Behavior.**

The third set of analyses tested hypotheses on family risk and parenting (the child's psychosocial context) in relation to cortisol during the session of cognitive tasks. Analyses evaluated the relative contributions of negative lifetime life events, controlling for the effects of age, gender, time of session, year data was collected, and cumulative resource-related risk on both intercept and trajectory. Given that a logarithmic transformation of the time metric produced demonstrably poor model fit while testing the first hypothesis, models that include a log transformation were not considered in this set of models.

First, I compared six models to evaluate the possible relationships between total negative lifetime event scores (LTE), initial session cortisol, and cortisol slope/trajectory over the course of the session (no LTE effects, LTE effect for intercept only, LTE effects for intercept and

trajectory). Model fit statistics and weights are provided in Table 4. The absolute best fitting model contained a quadratic trend and no effect of negative lifetime event scores on neither intercept nor on trajectory over time (weight = 0.42). The second best-fitting model contained an effect of LTE on intercept levels of cortisol, but not quadratic trend (weight = 0.27;  $\Delta AIC = 0.9$ ). Since these two models have equivalent fit, the more parsimonious model is adopted. In this case, the model without any effects of negative lifetime events is the more parsimonious model. The rejected model with considerable weight should continue to be considered in future work.

Second, negative lifetime event scores were separated into lifetime events that directly threatened the child and those that denoted family-level threats and disruptions. See Table 5 for items and endorsement rates. As before, age, sex, time of session, and resource-related cumulative risk were covaried as statistical controls. Ten models were compared. Model fit statistics and weights are provided in Table 6. The best-fitting and most parsimonious model involved only covariates with change estimated as a quadratic trend (weight = 0.31). Neither lifetime event scores (individual nor family-level threats) improved model fit to a large degree when effects were included for intercept, or intercept and trajectory/slope. Two alternative, less parsimonious models fit about as well, had considerable weights, and should receive consideration in future work. These were (1) the model that contained an intercept effect for direct events (weight = 0.18;  $\Delta AIC = 1.1$ ), and (2) the model that contained an intercept effect for family-related events (weight = 0.17;  $\Delta AIC = 1.1$ ).

Third, relationships were tested between positive parenting, parents' use of negative control, and cortisol levels during the session of cognitive tasks (intercept and slope/trajectory).

Because of missing parenting data (predictors in the current set of analyses), models are based on data from 126 participants ( $n$ ) contributing 493 observed cortisol values ( $n^*$ ). Ten models were compared to evaluate relative contributions of positive parenting and negative control on initial levels of cognitive session cortisol, and on slope/trajectory with time modeled as linear slope and as a quadratic trajectory in separate models. Model fit statistics and weights are provided in Table 7. Seven of the models have equivalent model fit with the best fitting model, including the model that contained only control variables with time modeled linearly ( $\Delta AIC = 0.2$ ; weight = 0.14) and the models that contained only control variables with time modeled as a quadratic term ( $\Delta AIC = 0.8$ ; weight = 0.11). As such, neither positive parenting nor negative control improved model fit for either cortisol intercept, or intercept and change in cortisol over the session of cognitive tasks (trajectory/slope).

Less parsimonious models with equivalent fit and sizeable weights should be considered in future work. These included separate models with: no intercept or trajectory effects of either positive parenting or negative control and change modeled quadratically (weight = 0.11;  $\Delta AIC = 0.8$ ); an effect of negative control on initial cortisol levels only, with change modeled linearly (weight = 0.15;  $\Delta AIC = 0.1$ ) or quadratically (weight = 0.16;  $\Delta AIC = 0.0$ ); an effect of negative control on intercept and linear slope (weight = 0.10;  $\Delta AIC = 0.8$ ); an effect of positive parenting on initial cortisol levels with change modeled linearly (weight = 0.06;  $\Delta AIC = 1.9$ ); and effects of positive parenting on intercept and slope with change modeled linearly (weight = 0.16;  $\Delta AIC = 0.0$ ).

**Aim 4: Child Cortisol during Parent-Child Interaction Tasks.**

The fourth aim was to examine the child's cortisol response during the session of interaction tasks with the parent. It was hypothesized that children who experienced higher levels of negative control, and children who experienced lower levels of positive parenting (less warm, sensitive, structured parenting with poor limits), would show a greater cortisol response to the parent-child session tasks (slope/trajectory effects). Twenty-one models were compared that involved intercept effects or intercept and slope/trajectory effects for positive parenting, negative control, and both, with time modeled linearly, as a log function, or as a quadratic trend. Model fit statistics and weights are provided in Table 8. Four models had equivalent fit with the best fitting model, but the best fitting model was also among the most parsimonious among the four. The best fitting model reflected change over time as a log-function and included only an intercept effect for negative control (weight = 0.30). Model coefficients are provided in Table 9 for the adopted model. Higher rates of negative control were related to higher cortisol levels throughout the parent-child interaction session ( $t = 2.38$ ). See Figure 2.

In addition, Table 8 indicates that the best fitting quadratic model has considerably worse model fit (weight = 0.02) when compared to the comparable log-function ( $\Delta AIC = 5.6$ ) and linear models ( $\Delta AIC = 4.3$ ). As such, quadratic transformations of time will not be included in further analyses of parent-child interaction session cortisol data.

Additional exploratory aims were also evaluated with respect to child cortisol during the parent-child interaction session. Thirty-four models were compared to evaluate the relationship between cortisol and EF, positive parenting, negative control, and their interaction, with time fit as a linear slope or a log-transformation of time. The models, fit statistics, and weights are provided in Table 10. Eight models had equivalent fit with the absolute best fitting model. Three

of these were equally parsimonious models. Two contained a log-transformation of time; one with an intercept (and not trajectory) effect for negative control only ( $\Delta\text{AIC} = 1.3$ ; weight = 0.06), and the other with just an intercept effect for EF ( $\Delta\text{AIC} = 0.8$ ; weight = 0.08). The third model estimated slope linearly and contained just an intercept effect for EF ( $\Delta\text{AIC} = 1.7$ ; weight = 0.05). These models provided better fit for the data than the respective comparison models that contained only covariate controls. It is also worth noting that models containing just intercept effects for both EF and negative control had equivalent model fit among the best fitting models, whether change was modeled linearly ( $\Delta\text{AIC} = 1.1$ ; weight = 0.07) or via a log-transformation of time ( $\Delta\text{AIC} = 0.0$ ; weight = 0.12). Meanwhile, both models that contained a product term between negative control and EF were also in this equivalent group (log-transformed time:  $\Delta\text{AIC} = 0.0$ ; weight = 0.11; linear time:  $\Delta\text{AIC} = 1.2$ ; weight = 0.06). In sum: (1) models have better fit if they contain either an effect of EF or negative control on initial cortisol levels in the parent-child interaction session, and (2) adding additional effects of EF or negative control (or their product) does not change model fit to a meaningful degree. The shared variance between EF and negative control accounts for a proportion of the effect on cortisol during the parent-child interaction session, but not enough to suggest moderation. Coefficients for the three adopted models are provided in Table 11. See Figures 2, 3, and 4.

### **Discussion**

The current study adds incremental evidence that cortisol functioning is related to psychological, family-level, and other contextual factors with implications for developmental failure or resilience among homeless children. The approach tested for links between cortisol and adversity history, parenting behavior, and executive functions; important predictors of



failure or competence for children who experience a high level of risk. Results indicated that in the testing session when children were separated from their parents to assess cognitive functioning, there was an association between lower levels of cortisol and higher levels of executive functioning. This finding is consistent with an ‘inverted-U’ relationship between cortisol and cognitive abilities, if one assumes that cortisol levels are elevated among these children living in an emergency shelter. In the inverted-U account, optimal cognitive functioning occurs at moderate elevations of cortisol. The current results add support to the “right-side” of the inverted-U relationship, as the context of ongoing homelessness likely contributes to higher levels of cortisol and a negative relationship with EF. Contrary to expectations, when children were separated from their parents for testing, neither negative lifetime event scores nor positive parenting or negative control were associated with cortisol. However, lower levels of EF and higher levels of negative control-type parenting were about equally related to higher levels of cortisol when children were interacting with their parents. This study also affirmed that salivary cortisol can be measured in a valid and reliable way among children staying in emergency shelter. Results are discussed in relation to the aims of this study.

**Aim 1: Measurement of Salivary Cortisol in Shelter.**

Results from this study further corroborated the feasibility of collecting valid and reliable cortisol data among young children in the ecologically important context of an emergency shelter for families (Cutuli, Wiik, et al., 2010; Wiik, et al., under review). Generally, cortisol values were correlated with each other at a moderate to very high degree (see Table 1), and the magnitude of the correlations were generally higher for samples that were closer to each other in time relative to those that were not as close. This speaks to reliability. In line with past work,

cortisol data conformed to expected diurnal patterns where levels are highest in the morning and decline throughout the day. This speaks to validity. The decline is evident in the negative correlation between the initial cortisol sample and the time day the session occurred. Although, this relationship does not apply to subsequent cortisol samples, perhaps due to increased variability in the time these samples were collected relative to the start of the session, or due to differences in cortisol response over the course of the session.

Children in shelter did not show an inordinate amount of potential confounds to cortisol levels. We asked about a number of factors that are thought to influence cortisol and potentially interfere with the ability to detect effects related to the constructs of interest. Most of these potential confounds received low endorsement rates (as reported by parents or observed by research assistants handling samples), and were modestly related to cortisol levels, at worst (see Appendix E). An exception was the number of children who had been in an argument earlier in the day that they participated in the study (57.1% of children), but this was only weakly related to cortisol ( $r = -0.18$ ). This high rate of endorsement may reflect any of a number of processes related to shelter-life (e.g., cramped living conditions; feelings of irritability or distress related to other factors, etc.), but it did not seem to have large consequences for cortisol levels at the time that samples were collected. A relatively small number of samples (18) contained debris, usually because the child had been eating recently and food had contaminated the saliva sample. These samples had a slightly higher cortisol value than others without debris, but the relationship still appears modest ( $r$ 's in the 0.2 range). This slight difference may reflect interference of debris in the assaying process. Nevertheless, the low endorsement rates of confounds, and the low

impact that these factors had on cortisol levels, supports the feasibility of collecting valid and reliable data in the shelter context.

Finally, given the age of the children, I expected and found that anticipation and the initial separation from caregivers would activate the HPA-axis as a component of the stress response. Consistent with this, the initial cortisol levels were higher and then declined across the course of the session of cognitive tasks, on average and generally taking the form of a quadratic function. This is an effect that has been demonstrated by other work with low-income, kindergarten-aged children (Blair, Granger, et al., 2005; Cutuli, Wiik, et al., 2010). It further supports the validity of measurement and approach in this context and population.

#### **Aim 2: Relationship between Executive Functions and Cortisol.**

Higher initial levels of cortisol were linked to lower levels of executive functions during the session of cognitive tasks. There was no effect on change in cortisol over the course of the session. This is a replication of past work with similarly-aged homeless children (Wiik, et al., under review). Prima facie and qualified by a number of assumptions, this finding is consistent with one popular, proposed means of influence by which cortisol has an effect on cognitive functioning through the ratio of mineralocorticoid versus glucocorticoid receptor occupation in key neural structures. MR versus GR occupation appears to modulate the stability of neuronal signaling (e.g., (de Kloet, et al., 1999)). Because MRs have greater affinity than GRs, the relationship between cortisol and cognitive functioning is thought to follow an inverted-U shaped curve with optimal cortisol concentrations slightly elevated past 'typical' diurnal levels, enough to fully occupy MRs while still minimizing GR occupation (with likely exceptions for memory encoding; (Prager & Johnson, 2009)). An effect of EF on change in cortisol levels was

neither expected nor found. This may indicate that recovery from the initial elevation may be subject to different factors than the increase in cortisol, which has support in the architecture of the HPA axis (Hackman, et al., 2010; Prager & Johnson, 2009; Sapolsky, et al., 2000).

Furthermore, sessions were largely standardized with respect to which measures were administered (children completed the same tasks), but the nature of some measures caused them to vary based on child performance (e.g., children who did better on the PPVT-4 completed more items than children who did worse). These differences may have artifactually altered changes in cortisol over the course of the session as children with higher levels of cognitive functioning may have experienced more items that might have caused them to feel more frustrated or bored than if they only experienced the items encountered by peers with lower levels of cognitive functioning (see Appendix F). This may have occluded trajectory effects of EF on cortisol.

Other studies have found the opposite association where higher levels or increases in cortisol were linked to better executive function abilities in lower-risk groups of preschool and kindergarten aged children (Blair, Granger, et al., 2005; Davis, et al., 2002). However, past analyses of data from similarly aged homeless children found the negative association that is demonstrated in the current study (Wiik, et al., under review). The apparent inconsistency can be resolved by recognizing that children in emergency homeless shelters tend to be experiencing a significant amount of recent or ongoing life stress (Gewirtz, et al., 2008; Rog & Buckner, 2007; Samuels, et al., 2010), and major life stress tends to increase basal levels and stress-induced cortisol before gradually declining over time (Gunnar & Vazquez, 2006; Miller, et al., 2007). If children staying in homeless shelters have already-elevated levels of cortisol due to

recent life stress, they are more likely to (1) go past optimal MR versus GR occupation with smaller elevations of cortisol, or (2) they may already be past optimal levels without any further elevation, and/or (3) they may be more likely to show greater elevations for longer periods of time in response to new stress. As such, this group may be 'on the right' of the inverted-U relationship between cortisol and cognitive functioning such that higher levels of cortisol are related to lower levels of cognitive functions like EF. Meanwhile, lower-risk groups who are less likely to have experienced recent stressful life events are more likely to be below optimal cortisol levels, and a moderate elevation in cortisol would move them closer to the optimal level. This account helps explain why studies with lower-risk children show a positive association between cortisol and executive functions, while the current study with children actively experiencing homelessness shows a negative association.

These analyses make an important contribution by affirming a negative association between cortisol and EF among a group of children who are actively experiencing high life stress and, therefore, are more likely to have already-elevated levels of cortisol. Past work has affirmed a positive relationship between EF and cortisol among lower-risk groups of preschoolers, consistent with the "left side" of an inverted-U relationship between cortisol and cognition. However, observing the "right side" of the inverted-U requires the inclusion of young children at higher levels of risk, which has been largely absent from the literature (Blair, 2010). These findings provide qualified evidence in support of the inverted-U relationship between EF and cortisol. Additional work is necessary to support the assumption that children show higher levels of cortisol while staying in emergency shelter.

Despite this explanation, the results across studies remain somewhat difficult to reconcile because the mean session cortisol levels in the Davis (2002) study of typically developing children were about 1 standard deviation *above* those in the Wiik study with homeless children (under review), even though the Davis samples were taken in the late afternoon when mean levels are expected to be lower and the Wiik samples in the morning or early afternoon. The mean cortisol levels in the Wiik study and Blair study (of children experiencing poverty more generally) (2005) appear to be more similar. Some of this variability may be due to differences in saliva collection and cortisol assay methodology, as no single, standardized procedure exists, let alone representative norms for child salivary cortisol. Nevertheless, if Davis' sample was left-of-center on the inverted-U, resulting in a 'higher-cortisol, better-EF' explanation, then the Wiik sample also should be left of center given the mean values of the cortisol concentrations, and higher cortisol should be linked to better EF as well. Because this is not the case, additional investigation is needed to understand the complexities of the relationship between EF and cortisol function, as well as the processes that impact developing physiological and psychological systems in kindergarten-aged and homeless children. These future efforts should be open to interpretations that appeal to receptor-occupation explanations, but other mechanisms related to differences in neurological structures and functions, as well as processes at other levels of analysis, must be considered.

A second possible explanation for the current findings appeals to neurodevelopmental effects of chronic or repeated exposure to high levels of cortisol. Children's cortisol response to anticipating the cognitive assessment session and being separated from their caregiver may indicate their typical cortisol response to stress in their day-to-day life in shelter (Wiik, et al.,

under review). In other words, higher cortisol levels at the start of the session may signal exposure to cumulatively higher levels of endogenous cortisol. Given the age of these children, these exposures are occurring during a period of rapid neurological development for structures such as the PFC and ACC that have been linked with executive functions (Blair, 2006; Casey, et al., 2000; Rueda, et al., 2005), and neural systems appear to be more vulnerable during periods in which they are developing (Gunnar & Quevedo, 2007). Chronically elevated endogenous glucocorticoids appear to play a role in mediating the effects of early life stress on neurological differences in animal models, including analogous structures that have been correlated with EF in humans: neuronal death in the PFC and hippocampus/reduced hippocampal volume, reduced MR and GR density in the hippocampus, and enduring differences in neuronal interconnectivity (Arabadzisz, et al., 2010; Erickson, et al., 2003; McEwen, 1999; Sapolsky, 2000). This explanation also coincides with work on allostatic load, whereby repeated or chronic activation of biological homeostatic processes carry negative repercussions for the organism's continued ability to maintain homeostasis (McEwen, 1998). In sum, chronic exposure to elevated cortisol may have neurotoxic effects on structures that appear to be important for EF.

The previous two explanations imply a unidirectional relationship where cortisol impedes neural processes and structures related to executive functions and, thereby, executive functions themselves. Yet, correlational data cannot adequately support (nor preclude) a unidirectional relationship whereby cortisol influences cognition, nor is such a position supported by the broader literature (Gunnar & Quevedo, 2007; Gunnar & Vazquez, 2006; Joels & Baram, 2009). A third possible explanation recognizes that other regulatory systems play roles in modulating the child's physiological stress response broadly, and cortisol levels specifically.

These regulatory systems can be internal (e.g., cognitive control and other psychological coping mechanisms; non-cognitive physiological self-regulation systems) or external (e.g., the experimenter, parent, or other adult that acts to help regulate the child); they can be calming and help inhibit the stress response, or stress-inducing and exacerbate the response. It may be that other regulatory systems modulate the impact of situations or experiences when it comes to stress/perceived stress on the individual, resulting in an attenuated (or amplified) cortisol response. Given their role in cognitive self-regulation processes, better executive function skills may buffer children's stress and arousal in response to separation from their caregiver. It may be the EF and cognitive control that negatively influences cortisol levels, and not the other way around. Children with better EF may have lower levels of cortisol as a product of psychological self-regulatory processes inhibiting HPA axis activation and/or other aspects of the stress response (Wiik, et al., under review).

The three possible explanations proposed above are not mutually exclusive, nor is it apparent that any exerts primacy over the others. Because of the correlational nature of this study and other reasons, it remains unclear if (1) cortisol levels acutely facilitate or interfere with neuronal processes related to EF, (2) chronic exposure to higher levels of cortisol influences neurodevelopment over the first five (or so) years of life, (3) EF operates as a psychological regulator of cortisol function and the physiological stress response, or (4) any or all of the above (Wiik, et al., under review). It appears most likely that relationships between EF, cognitive functioning, and the physiological stress response are multiply determined by factors across different levels of analysis, comprising an ongoing and complex interplay between physiology,



psychology, families, and broader contexts (Gunnar & Quevedo, 2007; Gunnar & Vazquez, 2006; Lupien, et al., 2009; Miller, et al., 2007; Sapolsky, et al., 2000).

**The “mechanism” problem across multiple levels of analysis.** Even if one knew with certainty that cortisol binding to MR versus GR facilitated or disrupted the stability of neuronal signaling in brain systems that are related to EF like the PFC and ACC, or that chronic exposure to endogenous cortisol altered those brain structures over time, neither would constitute a mechanism that would explain correlations between cortisol and EF. Rather, it would be a mechanism that explains how different levels of cortisol influence PFC or ACC structure or functioning. It is quite a different thing to equate neuronal firing or activation in various brain systems (or in any biological systems) with EF, as executive functions refer to a hypothetical construct that is psychological in nature (MacCorquodale & Meehl, 1948; Miller, 2010). By definition, EF refers to something different than the activation of biological systems, although EF may influence biology and vice versa. Assertions that equate psychological variables with biology appear inappropriate given the current state of a largely nascent and correlational literature relating the two, and doing so represents a form of naïve reductionism that carries the potential to distract from efforts aimed at understanding the phenomenology of central constructs (e.g., EF at the psychological level) and how constructs at different levels of analysis relate to each other (Lilienfeld, 2007; MacCorquodale & Meehl, 1948; Miller, 2010). Theory and findings that speak to the nature of these relationships must continue to advance beyond asserting the mere existence of such relationships (correlations), or be silent if the issue is beyond epistemological or communicative limits.

**Aim 3: Adversity and cortisol function.**

The current results failed to replicate past findings that linked adversity to cortisol function. The number of stressful life events experienced by children did not predict differences in the initial level of cortisol nor change over the course of the session. This is contrary to the hypothesis that a greater number of stressful lifetime life events would be related to higher initial levels of cortisol and a steeper decline over the course of the session of cognitive tasks. This was not the case. I also hypothesized that family-related adversity would have a stronger effect on initial child cortisol and change than life events that more directly threatened the child. This hypothesis failed to find support.

Together, these results suggest that stressful, negative lifetime events have, at best, a small and potentially negligible effect on child cortisol levels. Neither family-level nor more direct negative life events seem to have a more pronounced effect on cortisol function. This contrasts with past work showing a positive relationship between negative lifetime life event scores and initial cortisol levels and a negative relationship with change in cortisol over the duration of a session of cognitive tasks (Cutuli, Wiik, et al., 2010). It also contrasts with the broader literature linking early adversity to differences in cortisol functioning, especially adversities linked to family-level stress (Chen, et al., 2009; Flinn & England, 1997; Gunnar & Vazquez, 2006).

There are a number of possibilities that may address differences between past findings and the current results. Objections related to the fact that this was not an attempt at a literal replication of the Cutuli et al. (2010) study are not particularly persuasive (see Appendix G). More likely, the current analyses may not attend to enough of the relevant factors involved in the links between stressful negative life events and differences in cortisol function, such as the

developmental timing of events, the chronicity or repeated nature of events, psychological sequelae, and other risk factors (e.g., for depression) (Bevans, et al., 2008; Cicchetti, et al., 2010; Fernald, et al., 2008; Gunnar & Vazquez, 2006; Heim, et al., 2000; Lupien, et al., 2009).

Researchers have been trying to better elucidate the connections between adversity and cortisol function. One line of thought relies heavily on the idea of allostatic load (McEwen, 1998). Experiences that activate stress-response systems disturb homeostasis as the organism mobilizes resources and suppresses functions unrelated to stress-responding in favor of those more likely to aid in dealing with threat. Once the threat has passed, homeostasis must be regained: stress-systems must be returned to relative abeyance and other, suppressed functions must be restored. Accounts based on allostatic load assert that each disruption and return to homeostasis comes at a cost to the organism, a process of physiological wear-and-tear that reduces the likelihood of successful regulation in the future.

Following the idea of allostatic load, researchers have postulated and/or found that chronic or repeated negative life events or circumstances are much more likely to lead to lasting dysregulation of stress-response systems in general, and cortisol / HPA axis function specifically (Bevans, et al., 2008; Gustafsson, et al., 2010; Heim, et al., 2000). As these experiences accumulate over time, chronic elevations in cortisol can lead to down-regulation of the HPA axis and a reduction in cortisol levels, termed hypocortisolism (Pervanidou, 2008). This results in another inverted-U relationship, this time between the number of adverse events and cortisol levels (Fries, et al., 2005; Gustafsson, et al., 2010). The analyses in the current study looked for a linear association between the number of negative lifetime events and initial cortisol levels (at intercept), a hypothesis based on past work specifically with young homeless children. It is also

reasonable to hypothesize that cortisol levels at intercept would have a quadratic (inverted-U) relationship with negative life events which may be obscured by the linear mixed-modeling approach in the current study which tested for linear intercept effects.<sup>1</sup>

A second implication of allostatic load is that the nature of the cortisol dysregulation changes over time and may still be unfolding among these children. As allostatic load builds, the HPA axis may down-regulate to the point of hypocortisolism, but only after repeated or chronic activation of stress-response systems and accompanying elevations of cortisol. However, if the dysregulation is tied to the accumulating degree of life-stress *over time*, the children in the current study may be at different points in that process depending on when those life events started to occur, reoccurred, and how long it has been since. Meanwhile, certain kinds of adversities seem to impact cortisol functioning differently, such as sexual abuse, or one versus multiple forms of childhood abuse (Cicchetti & Rogosch, 2001a; Gunnar & Vazquez, 2006), and other factors such as parent behavior appear to impact the perception of stress following negative life events (Scheeringa & Zeanah, 2001). Furthermore, developing systems are more vulnerable to perturbation (e.g., from stress) than those that are mature or more fully developed, which adds additional complexity to predicting the impact of adversity (Gunnar & Quevedo, 2007). Greater attention to when adversity occurred and the chronicity or repeated nature of adversity may add precision that uncovers effects that are obscured in the current analyses.<sup>2</sup>

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<sup>1</sup> There is no such relationship (coefficients based on exploratory post-hoc analyses not reported).

<sup>2</sup> Although, unpublished analyses involving a different sample of 38 young homeless children using a similar protocol in 2007 failed to show an effect on cortisol when carefully accounting for the age in which negative lifetime events began and the chronicity of events.

Other neglected intricacies become apparent when considering the conceptual framework and proposed (but unmeasured) mechanisms through which people seem to think negative life events lead to differences in cortisol function. Namely, accounts that invoke the idea of allostatic load often appeal to chronic or repeated elevations in cortisol levels over time. However, as noted in previous sections, it seems very unlikely that experiencing the same event has the same impact on different individuals' cortisol functioning. Other factors influence the development of cortisol function beyond the characteristics of adversity, factors that may be internal (e.g., cognitive self-regulation/appraisals, the degree of autonomic response, receptor densities, methylated regions of DNA / modified histones, genotype, etc) or external (parenting behavior, family relationships, experiences with peers, at school, etc). For example, children with markers of vulnerability for internalizing symptoms (e.g., manifest internalizing symptoms; having a depressed parent) are particularly vulnerable to dysregulated cortisol levels in response to adversity (Cicchetti & Rogosch, 2001b; Cicchetti, et al., 2010; Essex, et al., 2002; Fernald, et al., 2008). Genetically informed studies suggest an influence of genotype on HPA axis functioning and vulnerability to stress (Alexander et al., 2009; Gotlib, Joorman, Minor, & Hallmayer, 2008), and recent work with animal models suggest a contribution through differential rates of gene regulation on GR expression in neural tissue based on maternal behavior generally, or maternal behavior following stressful experience early in life (Hackman, et al., 2010; Kaffman & Meaney, 2007; Meaney & Szyf, 2005). Links between negative life events and cortisol function would become clearer with greater attention to the potential contributing and/or moderating effects of other internal and external regulatory systems, and their potential to buffer the negative impact of stress on developing systems.

**Parenting behavior and child cortisol during the session of cognitive tasks.** As a small step towards incorporating additional levels of analysis, the current study explored the impact of parenting behavior in shelter as a possible contributor to differences in cortisol function. First, I predicted that positive and negative parenting behaviors during a session of interaction tasks would constitute distinct constructs, especially if they were indexed in a way that did not artifactually inflate their relationship (e.g., ratios of time engaged in positive versus negative parenting). The multidimensional nature of parenting behavior was apparent, given the low correlation between positive parenting and negative control. Any effects related to high levels of positive parenting do not appear to be merely the absence of negative control, and effects of negative control do not appear to be the mere absence of positive parent behavior.

Observed parenting was not related to children's cortisol function during the session of cognitive tasks. This is contrary to hypotheses that predicted trajectory effects for positive parenting and both intercept and trajectory effects for negative control. These rejected hypotheses were supported by past work showing effects of positive parenting on cortisol levels in response to stress among preschoolers and among animal models that suggest normalizing effects of high-quality maternal care on HPA-axis development following stress (Gunnar, 2000; Gunnar, et al., 1981; Hackman, et al., 2010; Hertsgaard, et al., 1995; Kaffman & Meaney, 2007; Nachimas, et al., 1996; Suchecki, Rosenfeld, & Levine, 1993). In the literature, negative effects of trauma and adversity at the behavioral level appear to impact children through parenting (Scheeringa & Zeanah, 2001). Also, positive parenting has been shown to support the development of internalized self-regulation skills, including better executive functions and cognitive skills more generally in a past sample of similarly aged homeless children (Herbers, et

al., 2011) as well as the current sample<sup>3</sup> (Herbers, 2011). Meanwhile, negative control has been linked to lower levels of EF and cognitive functioning (Herbers, 2011). While it seems that parenting behavior is important for child outcomes at the psychological level, and psychological constructs like executive functioning are linked to cortisol, parenting (positive or negative) was not meaningfully related to child cortisol outside of the parent's presence.

Several considerations might help explain this lack of relationship between parenting behavior and child cortisol response to the session of cognitive tasks. First, child cortisol functioning might be disrupted in less predictable ways immediately following recent stressful life events related to the move to shelter, and the role of parenting behavior in successful adaptation (more typical cortisol responses) may unfold over longer timeframes following the disruption. Findings by Fisher and colleagues (Fisher, et al., 2007) support this possibility: maltreated preschoolers had similar diurnal cortisol patterns during the first several months after entering foster care, but children in families that received a parenting intervention showed increased normalization of cortisol function over the course of a year. Analogously, almost all families staying in emergency shelter have experienced at least one recent major stressful life. It may be that parenting behavior over longer time periods would show stronger relationships with child cortisol functioning. Longitudinal work is required to test this possibility in families who experience homelessness.

Parenting behavior prior to the episode of homelessness is another worthy consideration, as previous experience and competence influence later development. This is a central tenant in theories of developmental risk and resilience (Masten et al., 2005; Yates, et al.,

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<sup>3</sup> This effect is largely driven by the 'Non-directive responsiveness' component of positive parenting, and not 'Positive control.'

2003). Furthermore, early caregiving has been linked to later cortisol function (Gunnar & Quevedo, 2007; Gunnar & Vazquez, 2006). The current study only observed parenting behavior in shelter and is unable to confidently speak to parenting behavior prior to the homeless experience. Observed parenting in shelter may not reflect a history of parenting more generally. Parent behavior in shelter is oft constrained, disrupted, or otherwise influenced by the characteristics of the shelter or situation itself (Bassuk & Rubin, 1987; Boxil & Beaty, 1990; Hausman & Hammen, 1993; Koblinsky, Morgan, & Anderson, 1997). For example, crowding and shared resources require many families to change typical routines, approaches, and strategies to accommodate. Parents may also be affected by recent stressful life events that may impair their ability to function at typical levels. Many families are unable to fully partake in some forms of social support due to shelter rules that prevent contact with certain individuals (e.g., unrelated father-figures; (Koblinsky, et al., 1997). In addition, parents frequently report that they feel constrained in their typical behaviors by shelter rules and “public parenting.” This includes parents who feel like they are “being watched” by shelter staff and other service providers who are mandated to report suspected child maltreatment to county services (Bassuk & Rubin, 1987; Boxil & Beaty, 1990). Shelters also may provide supports and resources that assist parents in engaging in more positive parenting behaviors. Therefore, it is unclear if observed parenting behaviors are different in shelter, and if parenting before the homeless experience is related to cortisol function during the shelter stay. These questions can start to be answered with additional longitudinal work that is prospective in nature (and extremely difficult to do), or follows families out of shelter after they have been re-housed.

**Aim 4: Child cortisol functioning during the session of parent-child interaction tasks.**



The fourth aim of this study explored the relationship between different forms of parenting behavior as an external regulatory system and child cortisol function during a set of parent-child interaction tasks. Children who went on to experience higher rates of negative control by parents *started* the session of interaction tasks with higher levels of cortisol. Negative control was related to higher cortisol at intercept, but not change in cortisol over the parent-child interaction tasks. Positive parenting behaviors did not predict differences in cortisol trajectories or initial levels. This is contrary to the hypothesis that positive parenting would have a main effect predicting a faster decline in child cortisol over the course of the session as a source of external regulation that helps attune child self-regulatory systems (e.g., HPA-axis functioning; EF and cognitive control).

Past work has shown that both internal (e.g., EF) and external (e.g., positive parenting and negative control) regulatory systems need to be considered in relationships with cortisol functioning (Smeekens, et al., 2007). When this was done in the current study, EF and negative control were related to child cortisol. EF was negatively related to initial cortisol levels while higher levels of negative control were related to higher initial levels. However, these effects do not seem to be independent, but moderation did not occur either. This means that children's level of EF and later-experienced negative control statistically account for about the same amount of variance in initial cortisol levels during the interaction task session, and their degree of overlap ( $r = -0.32$ ) did not predict beyond the individual effects. No meaningful effects were found for positive parenting, or for change in cortisol.

Positive parenting failed to emerge as a source of external regulation with respect to child cortisol. However, it should be noted that the parent-child interaction tasks share few

characteristics of validated stressor paradigms (Gunnar, et al., 2009). Furthermore, mean cortisol trajectories suggest little, if any, activation of the HPA-axis during the interaction tasks, and only mild decline over the course of the session (e.g., see Figures 2, 3, and 4). It seems unlikely that the interaction tasks could be viewed as an activating stressor, producing fewer (or, perhaps, different) opportunities for positive parenting to regulate child cortisol. Positive parenting might serve as an external regulatory system in situations with greater demands that are more stressful for the child, an area to be explored in future research.

Along the same lines, negative control was related to differences in initial child cortisol levels, but not differences in activation or change in cortisol over the course of the second session when they were interacting with their parent. This might suggest that children experience anticipatory stress at the prospect of rejoining a caregiver who engages in somewhat more harsh, hostile, or insensitive forms of control. This seems less likely because there was no trajectory effect of negative control on cortisol in the first session (of cognitive tasks): children would have begun to show an elevation at the end of the first session in anticipation of rejoining their parent for the second session. A more parsimonious account involves the realization that intercept effects (without trajectory effects) reflect a mean difference in cortisol across all timepoints in the session. Children who experienced more negative parenting tended to have higher levels of cortisol over the session as a whole. This relationship was not present in the session of cognitive tasks, perhaps because of differences in stress responding related to the challenge of separation.

These findings must be qualified by the fact that the session of parent-child interaction tasks almost always followed the child session of cognitive tasks. The differences seen in the

interaction task session may be attributed to differences seen in the session of cognitive tasks, at least in part. Because children with higher levels of EF had lower levels of cortisol during the first session (of cognitive tasks) and there was no difference in change in cortisol over the course of that session, there should still be a positive relationship between EF and cortisol at the start of the second session as a function of the effect found in the first session. However, negative control was not related to cortisol differences during the first session of cognitive tasks. It remains unclear how much the results are influenced by the order of the sessions.

**Limitations.**

The current study had a number of limitations that restricted conclusions. First, the study had a cross-sectional, correlational design that lacked a matched control group. These limitations prevented stronger conclusions about the direction of effects between cortisol and EF and between cortisol and parenting. They also leave open questions about how the experience of homelessness and living in shelter alter child and parent functioning. This includes concerns about possible distortions in parent reporting of past events, as well as observed child EF and parenting behavior. Longitudinal designs can help alleviate these limitations if they include repeated measures of key variables, include a carefully matched control group of more stably-housed families, and are either prospective in nature or follow families out of shelter through a period of relative housing stability. Well-designed intervention research will further increase understanding by addressing proposed factors that contribute to maladaptive or suboptimal processes, thereby providing a manipulation to more strongly test theories of cause and process. Additional work is sorely needed that better accounts for the complex and changing factors that impact successful adaptation in the context of homelessness.

A number of other limitations deserve mention. This study involved homeless children in families from three shelters in a single large, urban area. This threatens the generalizability of the findings with respect to homeless families who do not stay in emergency shelter, families in more rural areas, and families in other states or countries that may have different policies and contexts. Similarly, the data were collected over the span of two subsequent summers, with cohort effects evident in key variables. Others have noted the likelihood of cohort effects among the homeless population, especially considering how this group might be impacted by differences in local and national economic trends (Buckner, 2008). In addition, several participants lacked data on key variables (besides cortisol observations) for a variety of reasons and were not included in analyses. Although, missingness appeared to conform to patterns consistent with Missing at Random criteria (Schafer & Graham, 2002). Cohort effects and missing data further threaten generalizability.

Finally, the analyses abandoned a frequentist approach to null hypothesis testing (e.g., p-values compared to a beta of 0.8 and alpha of 0.05) in favor of model comparisons and evaluation of effect sizes (an information-theoretic approach). This was done for good reason (e.g., see (Baayen, et al., 2008; Burnham & Anderson, 2004; Kullback & Leibler, 1951; Long, in press), and unveiled some meaningful effects while large amounts of variance continue to go unaccounted for. The information-theoretic approach is not without limitations, including a lack of consensus on which information criteria is best in different situations, what constitutes meaningful improvement when comparing model fit statistics, and how to best address missing data on the independent variables (Anderson, 2008; Burnham & Anderson, 2004).

Advancements in quantitative methodology hold the potential to improve the quality of inferences.

### **Conclusion and Implications.**

The current study elaborated on models of risk and resilience for young homeless children by incorporating measures of dynamic cortisol levels. This approach is grounded in developmental-ecological theory applied to a resilience framework while acknowledging factors at multiple levels of analysis. This study affirms that high-quality measurement of salivary cortisol is possible in the 'real world' context of an emergency shelter. While great care and sensitivity is required, it is important to include children and families experiencing active disruption and ongoing adaptation in research.

Study findings link cortisol levels with executive functions and negative parenting behaviors, factors that have been implicated in studies of resilient children. These links affirm that factors and systems at multiple levels of analysis are interconnected. Appreciating the interface of these systems furthers understanding of the processes of successful adaptation among children who experience homelessness and high levels of risk.

The current results contribute to a developing and nascent body of work by describing associations between cortisol levels and executive functions. However, the nature and mechanism of this relationship remains unclear: it may be that cortisol influences EF or EF influences cortisol in the short term; an effect where endogenous cortisol alters systems important for EF over the longer term; or a more complex relationship where additional factors at multiple levels of analysis contribute. Testing hypotheses in future work that incorporates additional factors across multiple levels of analysis (including physiological variables as well as

contextual ones) will further explicate the processes of risk and resilience for at-risk children. Incorporating measures of salivary cortisol into intervention research will also help understand the directionality (or multidirectionality) and mechanisms that contribute to the association of EF and cortisol.

Executive functions represent an area of particular interest to stakeholders in the wellbeing of children who experience poverty and homelessness because (1) children with poor EF abilities are overrepresented among low income groups (Hackman & Farah, 2009), (2) poor EF predicts poor academic achievement and mental health problems (Blair, 2002; Blair & Razza, 2007; Obradović, 2010), and (3) an albeit limited literature suggests that EF and related cognitive abilities can be improved through psychosocial interventions during early life and preschool (Bierman, et al., 2008; Diamond, et al., 2007; Rueda, et al., 2005; Thorell, Lindqvist, Nutley, Bohlin, & Klingberg, 2009), middle childhood (Holmes, Gathercole, & Dunning, 2009), and across the lifespan (Tang & Posner, 2009). The current findings hold open the possibility that interventions targeting improvements in child EF also will positively affect cortisol function, with likely downstream effects on multiple internal adaptive systems that are linked to cortisol in the short and long terms.

Interventions targeting negative parenting behaviors likely will encourage positive adaptation with respect to both EF and cortisol function for young, high-risk children. While family-level stressors and positive parenting were not related to cortisol in the present study, children who experienced more harsh, hostile, or negative interactions with their parent had higher cortisol levels when they were together, as did children who had worse EF. Meanwhile, links between lower levels of negative parenting behavior, child EF, and positive adaptation are

reported elsewhere for homeless children (Herbers, 2011; Herbers, et al., 2011). Interventions that support parents and reduce negative parenting have the potential to interdict maladaptive processes evident in poor child EF and dysregulated cortisol function.

In closing, homelessness represents a context of risk for poor developmental outcomes, especially for young children. Nevertheless, many homeless children show good developmental outcomes. The processes of risk and resilience that contribute to this variability involve multiple adaptive systems that are impacted by factors across levels of analysis, such as cortisol and physiology, executive function and other aspects of psychological functioning, and parenting behavior and the family context. Explaining how resilience occurs requires that we understand the processes of positive adaptation and the complex ways that that factors across levels of the individual and her context promote or impede adaptive systems that lead to developmental competence. This is certainly a daunting task, given what is known about the complexity of these adaptive systems in the context of development, and what remains unknown about how factors impact each other within and between levels of analysis over time. Nevertheless, our best models of risk and resilience provide the blueprint for policy, intervention, and social understanding for children and families who experience homelessness. The better these models become, the better we will be able to help those at the highest levels of risk.

Table 1. Correlation coefficients between variables used in primary analyses.

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
<i>Cognitive Session</i>												
1. Cortisol Sample 1	--											
2. Cortisol Sample 2	0.83	--										
3. Cortisol Sample 3	0.57	0.77	--									
4. Cortisol Sample 4	0.58	0.80	0.82	--								
<i>Parent-Child Tasks</i>												
5. Cortisol Sample 5	0.29	0.44	0.36	0.56	--							
6. Cortisol Sample 6	0.34	0.40	0.68	0.51	0.47	--						
7. Cortisol Sample 7	0.44	0.49	0.49	0.62	0.77	0.61	--					
8. Cortisol Sample 8	0.46	0.57	0.69	0.69	0.59	0.71	0.64	--				
9. Year of Data Collection	0.15	0.20	0.21	0.26	0.13	0.09	0.13	0.14	--			
10. Child Age	0.02	0.09	0.12	0.16	-0.04	-0.00	-0.01	0.04	0.18	--		
11. Child Sex	0.06	-0.07	-0.17	-0.10	-0.08	-0.14	-0.02	-0.03	0.03	0.01	--	
12. Time of Session	-0.35	-0.27	-0.08	-0.13	-0.06	-0.01	-0.04	-0.13	0.09	0.08	0.07	--
13. Cumulative Risk	0.07	-0.13	0.10	0.06	0.02	-0.11	-0.12	0.02	-0.01	0.05	-0.00	-0.00
14. Lifetime Events Total	0.05	0.05	0.17	0.15	0.09	0.12	0.05	0.03	0.04	0.07	0.01	-0.01
15. Family Lifetime Events	0.01	0.02	0.16	0.11	0.07	0.03	0.05	0.02	0.08	0.03	0.02	0.09
16. Direct Lifetime Events	0.01	0.01	0.08	0.17	0.09	0.13	0.03	0.02	0.01	0.14	-0.07	-0.06
17. Negative Control	0.09	0.03	0.03	-0.01	0.05	0.16	-0.00	0.12	-0.23	-0.08	0.06	-0.04
18. Positive Parenting	0.07	0.02	0.03	-0.02	-0.06	0.09	-0.08	0.05	0.06	-0.09	0.20	0.06
19. Executive Function	-0.12	-0.07	-0.03	-0.02	-0.09	-0.02	-0.10	-0.13	0.18	0.46	-0.07	0.04

Note: Coefficients are based on available data with a maximum N of 133 used in primary analyses. Cortisol Samples are listed by number for simplicity. Samples 1 to 4 refer to the Cognitive Task Session. Samples 5 to 8 refer to the Parent-Child Interaction Task Session.



	13.	14.	15.	16.	17.	18.	19.
13. Cumulative Risk	--						
14. Lifetime Events Total	0.19	--					
15. Family Lifetime Events	0.17	0.84	--				
16. Direct Lifetime Events	0.14	0.65	0.27	--			
17. Negative Control	-0.04	-0.04	-0.02	-0.09	--		
18. Positive Parenting	0.02	-0.01	-0.04	-0.10	0.11	--	
19. Executive Function	-0.08	0.23	0.23	0.06	-0.32	0.00	--

Note: Coefficients are based on available data with a maximum N of 133 used in primary analyses.

Table 2. Models and fit evaluating fixed effects of executive function on cortisol during the cognitive task session.

EF Fixed Effect	Curve	AIC	$\Delta$ AIC	Weight
None	Linear	-466.5	6.0	0.03
None	Log	-421.8	50.7	< 0.001
None	Quadratic	-469.2	3.3	0.12
Intercept	Linear	-468.4	4.1	0.08
Intercept	Log	-423.6	48.9	< 0.001
<b>Intercept<sup>a</sup></b>	<b>Quadratic</b>	<b>-472.5</b>	<b>0.0</b>	<b>0.61</b>
Intercept, Trajectory	Linear	-466.5	6.0	0.03
Intercept, Trajectory	Log	-421.8	50.7	< 0.001
Intercept, Trajectory	Quadratic	-469.3	3.2	0.13

Note: Adopted model is in bold.

<sup>a</sup> denotes equivalent models  $\leq 2.0 \Delta$ AIC of the best fitting model

Table 3. Adopted model coefficients evaluating fixed effects of executive function on cortisol during the cognitive task session.

	<b><u>Fixed Effects</u></b>					
	<u>Intercept</u>	<u>t</u>	<u>Linear Slope</u>	<u>t</u>	<u>Quadratic Trajectory</u>	<u>t</u>
Executive Function	-5.75 (2.46)	-2.34	--	--	--	--
Year of data collection (2008 vs. 2009)	12.68 (4.77)	2.66	-0.46 (1.27)	-0.37	0.06 (0.15)	0.39
Child age in months	0.40 (0.35)	1.14	0.09 (0.09)	1.02	0.01 (0.01)	-0.74
Child sex (Female vs. Male)	2.35 (4.57)	0.51	-2.74 (1.22)	-2.25	0.26 (0.15)	1.77
Time of session start	-0.10 (0.02)	-5.03	0.01 (0.00)	2.58	-0.00 (0.00)	-0.98
Unconditional Effects	-101.6 (24.06)	-4.22	-9.81 (6.18)	-1.59	0.41 (0.77)	0.54
	<b><u>Variance Components</u></b>					
<i>Intercept (Std.Dev)</i>	6.09 (24.69)					
<i>Linear Slope (Std.Dev)</i>	0.18 (4.25)					
<i>Quadratic Slope (Std.Dev)</i>	0.00 (0.36)					
<i>Intercept, Quadratic Slope Covar.</i>	-0.22					
$\sigma^2$	0.66 (8.11)					
	<b><u>Model Fit</u></b>					
Akaike Information Criterion	-472.5					

Note: Unstandardized coefficients expressed in terms of  $x * 100$ . T-scores are expressed in their natural metric. Child sex and Year of data collection were entered as dummy-coded dichotomous variables. All other variables were continuous.

Table 4. Models and fit evaluating fixed effects of negative life events on cortisol during the session of cognitive tasks.

LTE Fixed Effect	Curve	AIC	$\Delta$ AIC	Weight
None	Linear	-462.8	2.2	0.14
<b>None<sup>a</sup></b>	<b>Quadratic</b>	<b>-465.0</b>	<b>0.0</b>	<b>0.42</b>
Intercept	Linear	-461.8	3.2	0.08
Intercept <sup>a</sup>	Quadratic	-464.1	0.9	0.27
Intercept, Trajectory	Linear	-460.0	5.0	0.04
Intercept, Trajectory	Quadratic	-460.5	4.5	0.04

Note: Adopted model is in bold.

<sup>a</sup> denotes equivalent models  $\leq 2.0 \Delta$ AIC of the best fitting model

Table 5. Negative life event endorsement rates and categorization.

<u>Has this child...</u>	<u>n</u>	<u>%</u>	<u>Family/Individual Threat</u>
Lived in a home with fights or severe relationship problems between parents or adults taking care of him/her	47	35%	Family
Had a parent who was in prison	44	33%	Family
Experienced the divorce or permanent separation of his/her parents	39	29%	Family
Been separated from his/her parents for more than 2 weeks	39	29%	Unclear
Been hospitalized	32	24%	Individual
Seen violence happening to other people	32	24%	Individual
Seen a parent injured by another person	28	21%	Family
Lived with a parent who had a mental illness	26	20%	Family
Other (unlisted) major life event	20	15%	Unclear
Lived with a parent who had a serious alcohol or drug problem	17	13%	Family
Been in a serious accident (car, bike, boat) or nearly drowned	14	11%	Individual
Lived with a parent who had a serious physical illness	12	9%	Family
Been attacked by an animal	10	8%	Individual
Witnessed a serious accident involving a car, plane, or boat	9	7%	Individual
Been in a house fire	9	7%	Individual
Ever lived in a foster home	7	5%	Family
Been the victim of physical violence (For example, your child was seriously injured by another person or raped)	7	5%	Individual
Experienced a natural disaster such as a flood, hurricane, or tornado	7	5%	Individual
Experienced death of a parent	3	2%	Family
Experienced death of a brother or sister	3	2%	Unclear
Been kidnapped	2	2%	Individual
Experienced any other severe threat to his/her life or safety	2	2%	Unclear

Table 6. Models and fit evaluating fixed effects of family and direct negative lifetime events on cortisol during the session of cognitive tasks.

LTE Fixed Effect	Curve	AIC	$\Delta$ AIC	Weight
None	Linear	-462.8	2.2	0.10
<b>None<sup>a</sup></b>	<b>Quadratic</b>	<b>-465.0</b>	<b>0.0</b>	<b>0.31</b>
LTE-Direct: Intercept	Linear	-461.2	3.8	0.05
LTE- Direct: Intercept <sup>a</sup>	Quadratic	-463.9	1.1	0.18
LTE-Family: Intercept	Linear	-461.6	3.4	0.05
LTE-Family: Intercept <sup>a</sup>	Quadratic	-463.9	1.1	0.17
LTE- Direct: Intercept, Trajectory	Linear	-459.5	5.5	0.02
LTE- Direct: Intercept, Trajectory	Quadratic	-462.0	3.0	0.07
LTE-Family: Intercept, Trajectory	Linear	-459.9	5.1	0.02
LTE-Family: Intercept, Trajectory	Quadratic	-460.4	4.6	0.03

Note: Adopted model is in bold.

<sup>a</sup> denotes equivalent models  $\leq 2.0 \Delta$ AIC of the best fitting model

Table 7. Models and fit evaluating fixed effects of negative control and positive parenting on cortisol during the session of cognitive tasks.

Models	Curve	AIC	$\Delta$ AIC	Weight
<b>None<sup>a</sup></b>	<b>Linear</b>	-459.8	<b>0.2</b>	<b>0.14</b>
None <sup>a</sup>	Quadratic	-459.2	0.8	0.11
Neg. Control: Intercept <sup>a</sup>	Linear	-459.9	0.1	0.15
Neg. Control: Intercept <sup>a</sup>	Quadratic	-460.0	0.0	0.16
Pos. Parenting: Intercept <sup>a</sup>	Linear	-458.1	1.9	0.06
Pos. Parenting: Intercept	Quadratic	-457.0	3.0	0.04
Neg. Control: Intercept, Trajectory <sup>a</sup>	Linear	-459.2	0.8	0.10
Neg. Control: Intercept, Trajectory	Quadratic	-457.7	2.3	0.05
Pos Parenting: Intercept, Trajectory <sup>a</sup>	Linear	-460.0	0.0	0.16
Pos Parenting: Intercept, Trajectory	Quadratic	-457.4	2.6	0.04

Note: Adopted model is in bold.

<sup>a</sup> denotes equivalent models  $\leq 2 \Delta$ AIC of the best fitting model

Table 8. Models and fit evaluating fixed effects of negative control and positive parenting on cortisol during parent-child interaction tasks.

Parenting Fixed Effects Models	Curve	AIC	$\Delta$ AIC	Weight
None	Linear	-280.6	4.7	0.03
None	Quadratic	-274.0	11.3	< 0.01
None	Log	-281.8	3.5	0.05
Neg. Control: Intercept <sup>a</sup>	Linear	-284.0	1.3	0.16
Neg. Control: Intercept	Quadratic	-279.7	5.6	0.02
<b>Neg. Control: Intercept <sup>a</sup></b>	<b>Log</b>	<b>-285.3</b>	<b>0.0</b>	<b>0.30</b>
Pos. Parenting: Intercept	Linear	-278.7	6.6	0.01
Pos. Parenting: Intercept	Quadratic	-272.1	13.2	< 0.001
Pos. Parenting: Intercept	Log	-279.8	5.5	0.02
Both: Intercept	Linear	-282.0	3.3	0.06
Both: Intercept	Quadratic	-277.5	7.8	0.01
Both: Intercept <sup>a</sup>	Log	-283.3	2.0	0.11
Neg. Control: Intercept, Trajectory	Linear	-282.1	3.2	0.06
Neg. Control: Intercept, Trajectory	Quadratic	-277.0	8.3	< 0.01
Neg. Control: Intercept, Trajectory <sup>a</sup>	Log	-283.4	1.9	0.11
Pos Parenting: Intercept, Trajectory	Linear	-277.6	7.7	< 0.01
Pos Parenting: Intercept, Trajectory	Quadratic	-269.4	15.9	< 0.001
Pos Parenting: Intercept, Trajectory	Log	-279.2	6.1	0.01
Both: Intercept, Trajectory	Linear	-278.9	6.4	0.01
Both: Intercept, Trajectory	Quadratic	-272.1	13.2	< 0.001
Both: Intercept, Trajectory	Log	-280.6	4.7	0.03

Note: Adopted model is in bold.

<sup>a</sup> denotes equivalent models  $\leq 2.0 \Delta$ AIC of the best fitting model



Table 9. Adopted model coefficients evaluating fixed effects of negative control and positive parenting on cortisol during parent-child interaction tasks.

	<b><u>Fixed Effects</u></b>			
	<u>Intercept</u>	<u>t</u>	<u>Log Trajectory</u>	<u>t</u>
Negative Control	342.1 (143.8)	2.38	--	--
Year of data collection (2008 vs. 2009)	14.79 (4.75)	3.12	-1.93 (1.61)	1.20
Child age in months	-0.38 (0.32)	-1.19	0.25 (0.11)	2.27
Child sex (Female vs. Male)	-6.88 (4.45)	-1.54	2.09 (1.54)	1.36
Time of session start	-0.03 (0.09)	-1.58	0.00 (0.01)	0.34
Unconditional Effects	-84.88 (22.67)	-3.74	-19.16 (7.67)	-2.50
	<b><u>Variance Components</u></b>			
<i>Intercept (Std.Dev)</i>		4.72 (21.73)		
<i>Log Trajectory (Std.Dev)</i>		0.02 (1.46)		
<i>Intercept, Log Trajectory Covar.</i>		0.91		
$\sigma^2$		1.46 (12.10)		
	<b><u>Model Fit</u></b>			
Akaike Information Criterion		-285.3		

Note: Unstandardized coefficients expressed in terms of  $x * 100$ . T-scores and AIC are expressed in their given metrics. Child sex and Year of data collection were entered as dummy-coded dichotomous variables. All other variables were continuous.

Table 10. Models and fit evaluating fixed effects of parenting and EF on cortisol during parent-child interaction tasks.

Parenting and EF Fixed Effects Models	Curve	AIC	$\Delta$ AIC	Weight
None	Linear	-280.6	6.0	< 0.01
None	Log	-281.8	4.8	< 0.01
<b>EF: Intercept<sup>a</sup></b>	<b>Linear</b>	<b>-284.9</b>	<b>1.7</b>	<b>0.05</b>
<b>EF: Intercept<sup>a</sup></b>	<b>Log</b>	<b>-285.8</b>	<b>0.8</b>	<b>0.08</b>
Neg. Control: Intercept	Linear	-284.0	2.6	0.03
<b>Neg. Control: Intercept<sup>a</sup></b>	<b>Log</b>	<b>-285.3</b>	<b>1.3</b>	<b>0.06</b>
Pos. Parenting: Intercept	Linear	-278.7	7.9	< 0.01
Pos. Parenting: Intercept	Log	-279.8	6.8	< 0.01
Neg. & Pos. Parenting: Intercept	Linear	-282.0	4.6	0.01
Neg. & Pos. Parenting: Intercept	Log	-283.3	3.3	0.02
Neg. Control & EF: Intercept <sup>a</sup>	Linear	-285.5	1.1	0.07
Neg. Control & EF: Intercept <sup>a</sup>	Log	-286.6	0.0	0.11
Pos. Parenting & EF: Intercept	Linear	-283.1	3.5	0.02
Pos. Parenting & EF: Intercept	Log	-284.0	2.6	0.03
Neg. Control * EF: Intercept <sup>a</sup>	Linear	-285.4	1.2	0.06
Neg. Control * EF: Intercept <sup>a</sup>	Log	-286.6	0.0	0.11
Pos. Parenting * EF: Intercept	Linear	-281.1	5.5	< 0.01
Pos. Parenting * EF: Intercept	Log	-282.0	4.6	0.01
EF: Intercept, Trajectory	Linear	-284.1	2.5	0.03
EF: Intercept, Trajectory <sup>a</sup>	Log	-285.2	1.4	0.06
Neg. Control: Intercept, Trajectory	Linear	-282.1	4.5	0.01
Neg. Control: Intercept, Trajectory	Log	-283.3	3.3	0.02
Pos. Parenting: Intercept, Trajectory	Linear	-277.6	9.0	< 0.01
Pos. Parenting: Intercept, Trajectory	Log	-279.1	7.5	< 0.01
Neg. & Pos. Parenting: Intercept, Trajectory	Linear	-278.9	7.7	< 0.01
Neg. & Pos. Parenting: Intercept, Trajectory	Log	-280.6	6.0	< 0.01
Neg. Control & EF: Intercept, Trajectory	Linear	-282.8	3.8	0.02
Neg. Control & EF: Intercept, Trajectory	Log	-284.0	2.6	0.03
Pos. Parenting & EF: Intercept, Trajectory	Linear	-281.3	5.3	< 0.01
Pos. Parenting & EF: Intercept, Trajectory	Log	-282.8	3.8	0.02
Neg. Control * EF: Intercept, Trajectory	Linear	-283.1	3.5	0.02
Neg. Control * EF: Intercept, Trajectory	Log	-283.9	2.7	0.03
Pos. Parenting * EF: Intercept, Trajectory	Linear	-283.7	2.9	0.03
Pos. Parenting * EF: Intercept, Trajectory	Log	-284.5	2.1	0.04

Note: Adopted models are in bold.

<sup>a</sup> denotes equivalent models  $\leq 2.0$  dAIC of the best fitting model

Table 11. Coefficients of adopted models evaluating fixed effects of parenting and EF on cortisol during parent-child interaction tasks.

	<b>Model 1</b>		<b>Model 2</b>		<b>Model 3</b>	
<u>Intercept Effects</u>	<u>Estimate (SE)</u>	<u>t</u>	<u>Estimate (SE)</u>	<u>t</u>	<u>Estimate (SE)</u>	<u>t</u>
Negative Control	342.1 (143.8)	2.38	--	--	--	--
Executive Function	--	--	-7.62 (3.04)	-2.51	-7.85 (3.07)	-2.56
Year of data collection (2008 vs. 2009)	14.79 (4.75)	3.12	13.71 (4.70)	2.92	13.79 (4.75)	2.90
Child age in months	-0.38 (0.32)	-1.19	-0.04 (0.02)	-0.12	0.00 (0.36)	0.01
Child sex (Female vs. Male)	-6.88 (4.45)	-1.54	-7.16 (4.49)	-1.60	-7.04 (4.53)	-1.55
Time of session start	-0.03 (0.09)	-1.58	-0.03 (0.02)	-1.66	-0.03 (0.02)	-1.54
Unconditional Effect	-84.88 (22.67)	-3.74	100.1 (24.70)	-4.05	-103.9 (24.91)	-4.17
<u>Trajectory Effects</u>	<u>Estimates: Log Traj.</u>		<u>Estimates: Log Traj.</u>		<u>Estimates: Linear Slope</u>	
Negative Control	--	--	--	--	--	--
Executive Function	--	--	--	--	--	--
Year of data collection (2008 vs. 2009)	-1.93 (1.61)	1.20	-1.97 (1.61)	-1.23	-0.76 (0.54)	-1.41
Child age in months	0.25 (0.11)	2.27	0.25 (0.11)	2.29	0.08 (0.04)	2.26
Child sex (Female vs. Male)	2.09 (1.54)	1.36	2.13 (1.54)	1.38	0.77 (0.52)	1.48
Time of session start	0.00 (0.01)	0.34	0.00 (0.01)	0.33	0.00 (0.00)	0.10
Unconditional Effect	-19.16 (7.67)	-2.50	-19.35 (7.67)	-2.52	-6.32 (2.59)	-2.44
<u>Variance Components</u>						
<i>Intercept (Std.Dev)</i>	4.72 (21.73)		4.80 (21.91)		5.17 (22.74)	
<i>Trajectory (Std.Dev)</i>	0.02 (1.46)		0.02 (1.44)		0.01 (0.77)	
<i>Intercept, Trajectory Covar.</i>	0.91		0.76		0.18	
$\sigma^2$	1.46 (12.10)		1.46 (12.10)		1.44 (0.12)	
<u>Model Fit</u>						
Akaike Information Criterion	-285.3		-285.9		-285.0	

Note: Unstandardized coefficients expressed in terms of  $x * 100$ . T-scores and AIC are expressed in their given metrics.

Child sex and Year of data collection were entered as dummy-coded dichotomous variables. All other variables were continuous.

Figure 1. Cortisol trajectories for lowest and highest EF groups during the session of cognitive tasks. Lines represent the highest and lowest thirds on EF composite scores. Lines are quadratic estimations of observed cortisol means. Shaded areas represent 95% confidence intervals.

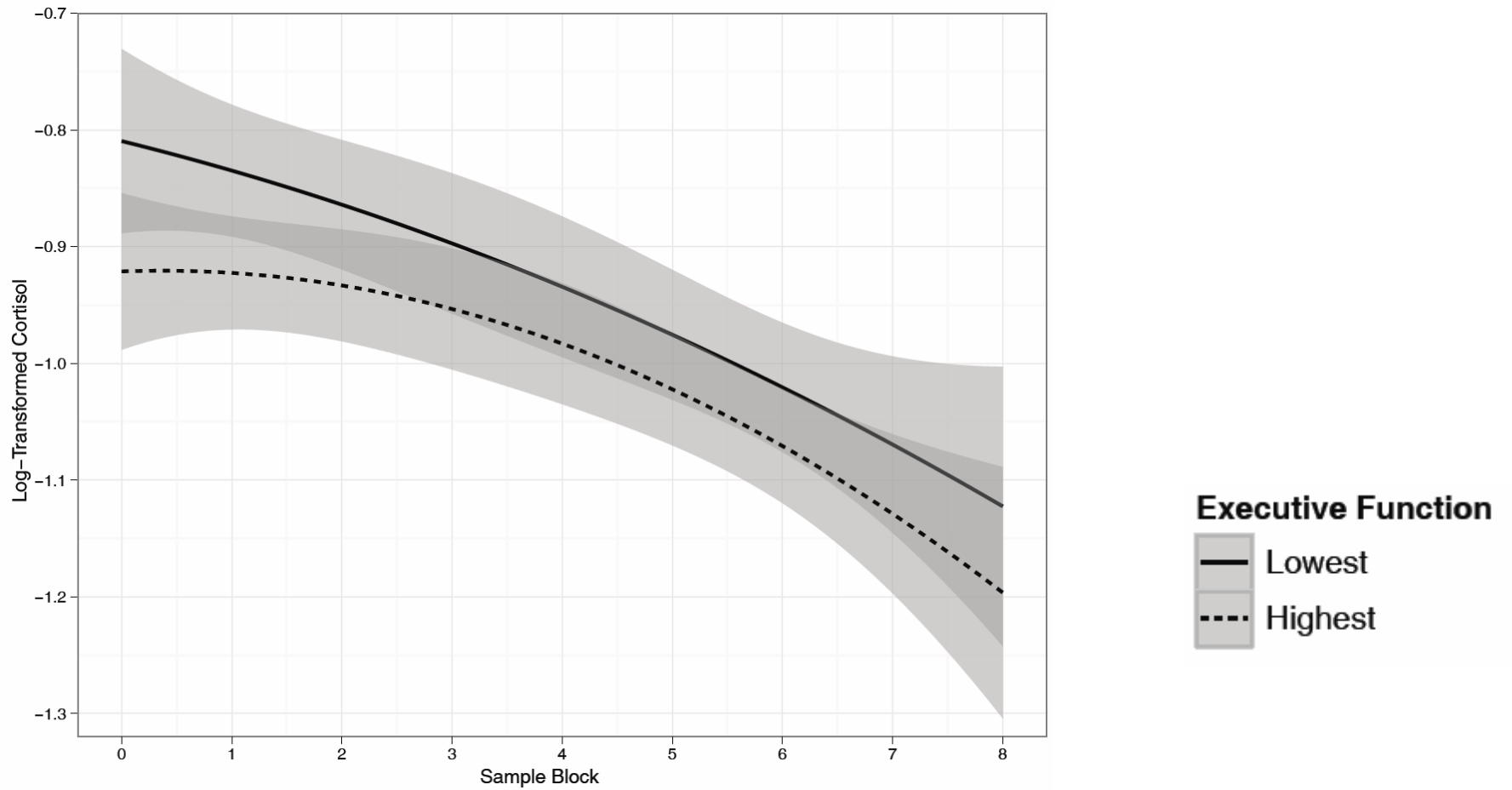


Figure 2. Cortisol trajectories for children with low and high levels of parental negative control. Lines represent groups created by a median split of negative control scores. Lines are linear estimations of observed cortisol means during the session of parent-child tasks. Shaded areas represent 95% confidence intervals.

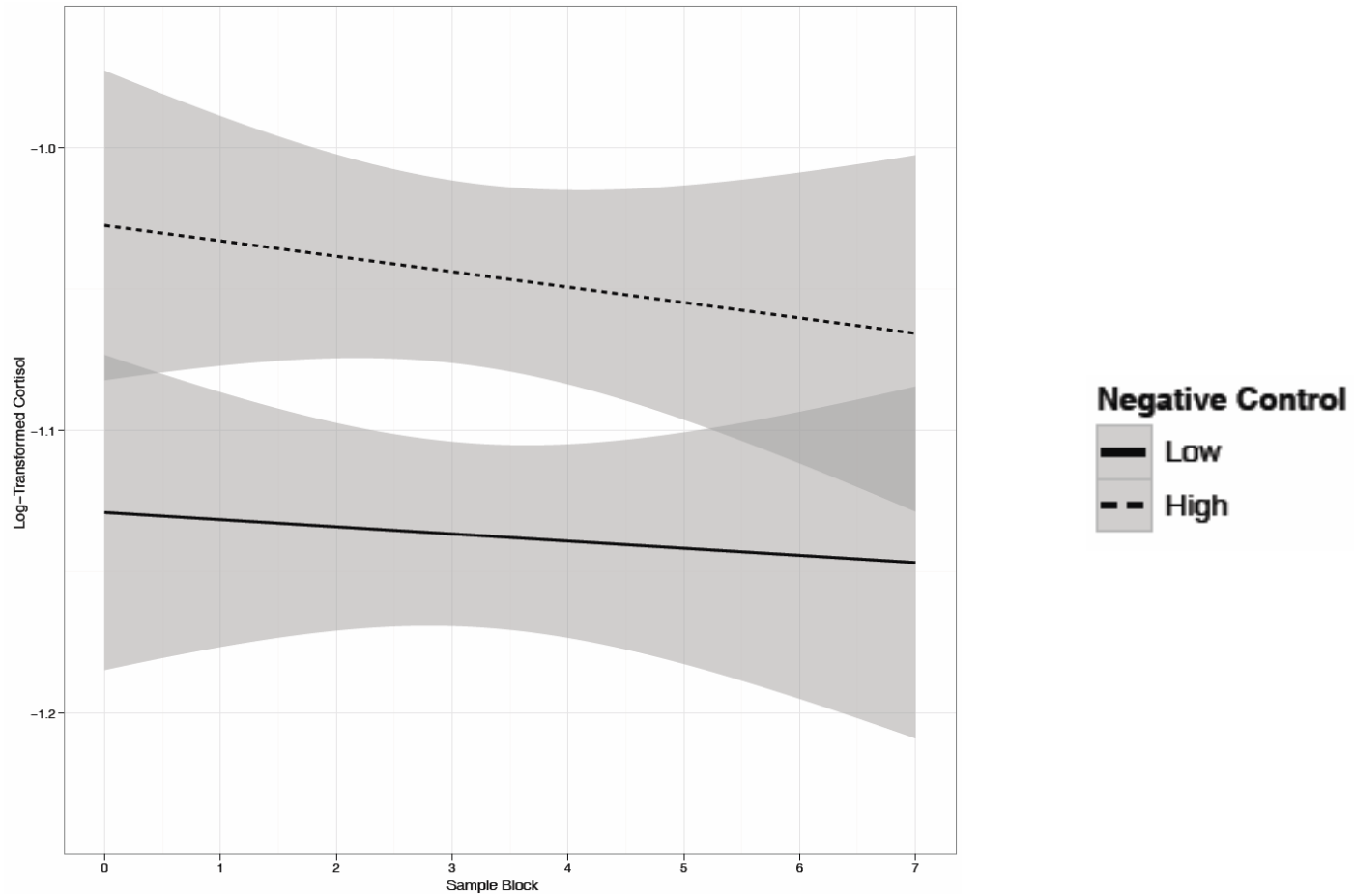


Figure 3. Cortisol trajectories for children with low and high levels of executive functioning. Lines represent groups created by a median split of composite EF scores. Lines are linear estimations of observed cortisol means during the session of parent-child tasks. Shaded areas represent 95% confidence intervals.

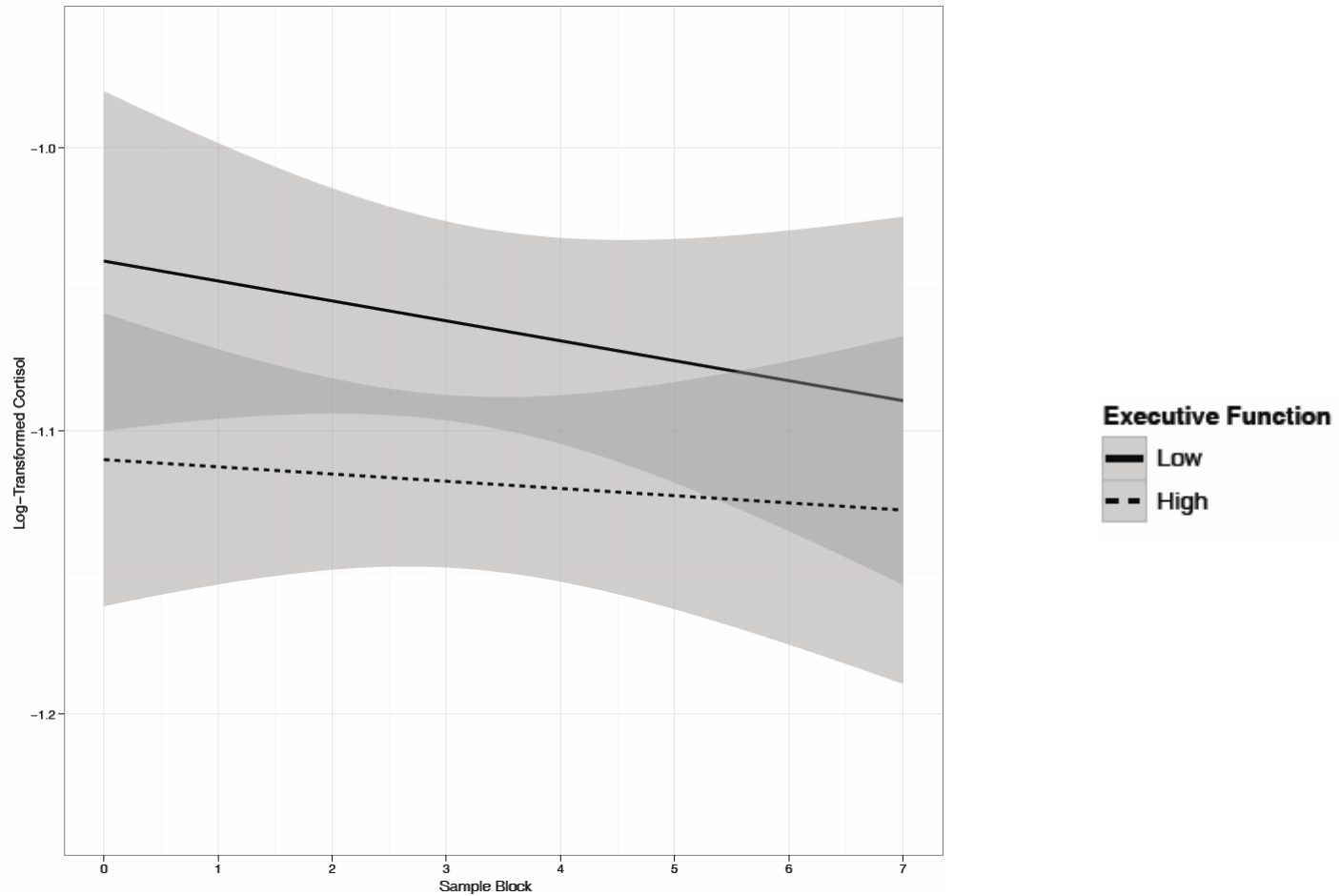
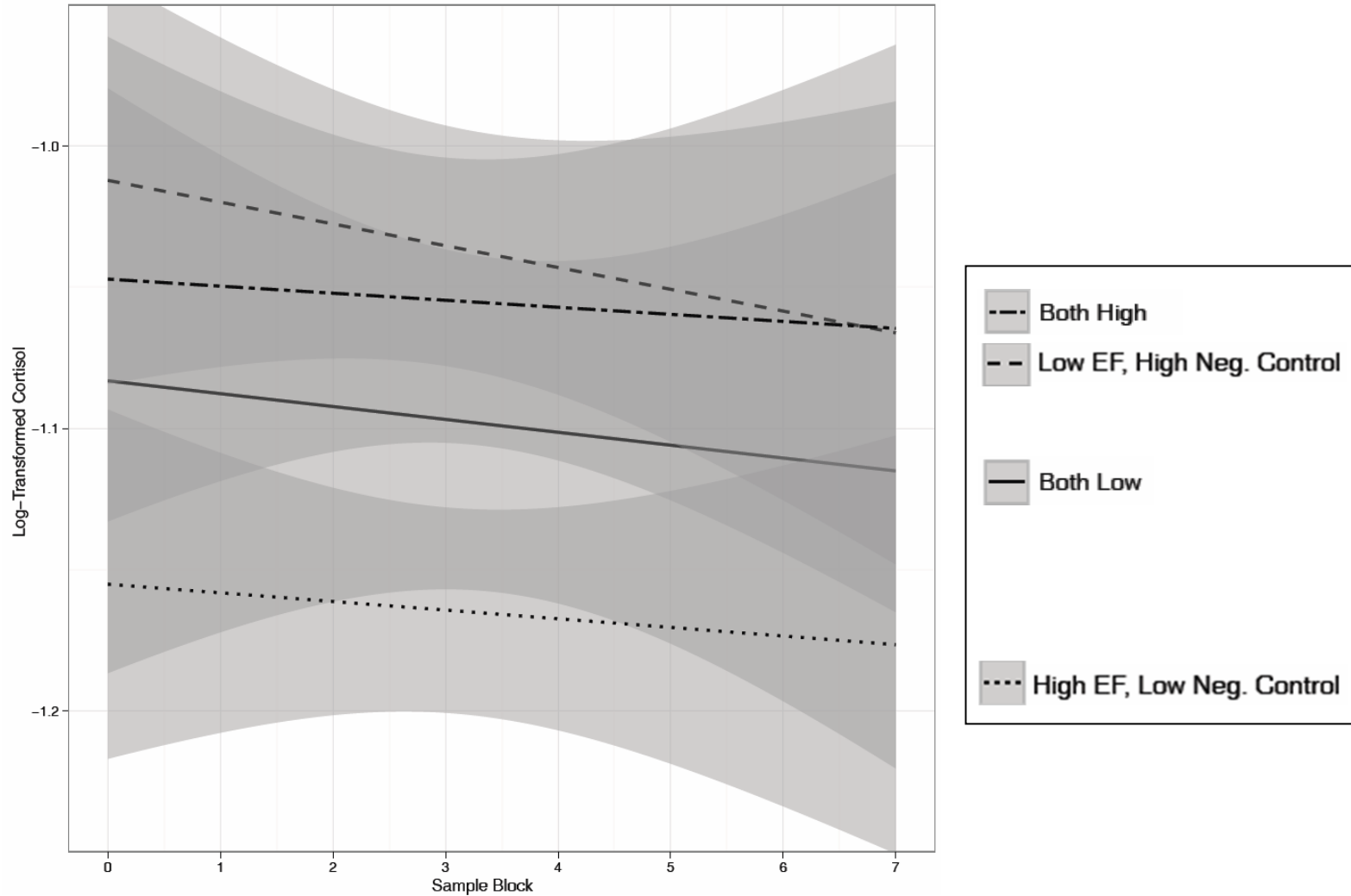


Figure 4. Cortisol trajectories for children separated into four groups based on two median splits: high EF and high negative control, high EF and low negative control, low EF and high negative control, and low EF and low negative control. Lines are linear estimations of observed cortisol means during the session of parent-child tasks. Shaded areas represent 95% confidence intervals.



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## **Appendix A. Additional information on non-participating and excluded families.**

All families meeting inclusion/exclusion criteria and staying in shelter during the study periods were invited to participate. Forty-six families with a child in the target age range were excluded from participation: 31 families were excluded because they did not stay in shelter beyond 3 nights during the data collection period; 13 families because of limited English proficiency; and 2 families because the child had a diagnosed developmental delay that would prevent her or him from participating in the protocol. There were 53 additional families who were not enrolled in the study but were believed to be eligible based on shelter records. Thirteen of these families declined to participate after learning of the study, including 3 families following the consent process. The other 40 families were a combination of those who expressed interest in the study but never participated (e.g., were not able to schedule a time, or failed to keep scheduled appointments due to other obligations) and families with whom we did not have face-to-face contact because they were not aware of the study or did not respond to fliers, invitations, telephone calls to their housing unit, or information tables in shelter common areas.

## **Appendix B. Rates of observed and missing data.**

Overall, 1,013 out of a possible 1,104 (91.76%) cortisol values were observed and available for analyses. A higher proportion of samples were available for the session of cognitive tasks (528 of 552; 95.65%) than for the parent interaction session (485 of 552; 87.86%). Missing cortisol data (the dependent variables) were judged to be missing at random (coefficients not reported). Data that are missing at random or completely at random do not interfere with the maximum likelihood estimation of the linear mixed models. However, cases were excluded if there is any missing data on any of the independent variables, considered separately for analyses relevant to each hypothesis. No child was missing data for age, sex, time of session, negative lifetime events, or cumulative risk. For the first hypothesis, 2 participants were missing all EF scores and 3 did not provide any cortisol data. This results in a final sample of 133 participants providing 516 (93.49% of possible) cortisol values to inform analyses for hypotheses involving cortisol during the session of cognitive tasks. Nine participants lacked parenting scores, resulting in a smaller sample ( $n = 126$ ) considered for testing hypotheses that involve parenting behavior, with 493 cognitive-assessment session cortisol values (89.31%) available for this subset. Finally, nine participants were missing data on at least one independent variable relevant to the testing of the third hypothesis: 7 missing parenting, 1 missing EF, and 1 missing both parenting and EF. Two additional children did not provide any cortisol data during the parent-child interaction session and were excluded from those analyses. A total of 127 individuals representing 473 (85.89% of possible) cortisol values were available for those analyses. The number of observed cortisol values by session collection time block is provided below.



Session Time Blocks (in minutes)	Cognitive Task Session			Parent-Child Task Session		
	Count	Mean	Std.Dev.	Count	Mean	Std.Dev.
0	132	0.18	0.13	126	0.10	0.09
0.1 - 9.9	0			0		
10 - 19.9	61	0.16	0.11	50	0.10	0.10
20 - 29.9	67	0.13	0.09	85	0.10	0.08
30 - 39.9	60	0.14	0.12	58	0.09	0.08
40 - 49.9	63	0.13	0.10	74	0.11	0.10
50 - 59.9	60	0.11	0.07	56	0.09	0.06
60 - 69.9	56	0.11	0.08	34	0.09	0.06
70 - 79.9	23	0.08	0.04	0		

**Appendix C. Means for key variables by year, site, and total.**

	Breakdown by year						Cohen's d
	2008			2009			
	Mean	Std.Dev.	n	Mean	Std.Dev.	n	
Age in Months	67.80	6.79	50	70.41	7.13	83	<b>-0.37</b>
Nights in shelter	37.58	60.30	50	30.04	33.48	83	0.17
Lifetime Events	2.90	2.25	50	3.07	2.09	83	-0.08
- Direct threats	0.90	1.04	50	0.93	0.92	83	-0.03
- Family threats	1.44	1.36	50	1.65	1.32	83	-0.16
Cumulative Risk	2.06	1.04	50	2.05	1.04	83	0.01
Executive Function	-0.21	0.89	50	0.08	0.70	83	<b>-0.37</b>
Negative control	0.03	0.02	46	0.02	0.01	80	<b>0.45</b>
Positive Parenting	0.07	0.01	46	0.07	0.01	80	-0.09
Cognitive task session							
- Cortisol sample 1	0.16	0.11	50	0.20	0.14	80	<b>-0.31</b>
- Cortisol sample 2	0.12	0.07	49	0.16	0.11	80	<b>-0.41</b>
- Cortisol sample 3	0.10	0.05	49	0.15	0.13	80	<b>-0.45</b>
- Cortisol sample 4	0.08	0.03	48	0.12	0.10	80	<b>-0.43</b>
Parent-child task session							
- Cortisol sample 5	0.09	0.05	45	0.11	0.11	76	<b>-0.30</b>
- Cortisol sample 6	0.09	0.07	46	0.11	0.10	76	-0.19
- Cortisol sample 7	0.08	0.07	45	0.11	0.09	75	-0.28
- Cortisol sample 8	0.08	0.04	37	0.10	0.09	73	<b>-0.32</b>

Note: Statistics based on cases used in primary analyses. Cortisol samples are divided by collection number for simplicity sake.

	Breakdown by Site									
	Site 1			Site 2			Cohen's d	Site 3		
	Mean	SD	n	Mean	SD	n			Mean	SD
Age in Months	68.85	6.96	71	70.17	7.29	59	-0.19	68.67	7.02	3
Nights in shelter	30.08	52.02	71	37.12	37.03	59	-0.15	15.33	14.05	3
Lifetime Events	2.89	1.88	71	3.15	2.48	59	-0.12	3.00	1.00	3
- Direct threats	0.80	0.89	71	1.07	1.05	59	-0.28	0.67	0.58	3
- Family threats	1.63	1.34	71	1.47	1.36	59	0.12	2.00	0.00	3
Cumulative Risk	2.17	0.94	71	1.95	1.11	59	0.22	1.33	1.53	3
Executive Function	-0.24	0.85	71	0.23	0.65	59	<b>-0.61</b>	0.03	0.42	3
Negative control	0.02	0.02	69	0.02	0.01	54	<b>0.42</b>	0.02	0.00	3
Positive Parenting	0.06	0.14	69	0.07	0.01	54	-0.03	0.07	0.02	3
Cognitive task session										
- Cortisol sample 1	0.19	0.14	69	0.17	0.12	58	0.15	0.16	0.13	3
- Cortisol sample 2	0.15	0.10	70	0.14	0.10	56	0.15	0.13	0.11	3
- Cortisol sample 3	0.14	0.12	70	0.13	0.11	56	0.06	0.10	0.06	3
- Cortisol sample 4	0.10	0.07	69	0.10	0.07	56	-0.01	0.08	0.03	3
Parent-child task session										
- Cortisol sample 5	0.10	0.09	67	0.11	0.10	51	-0.06	0.06	0.03	3
- Cortisol sample 6	0.09	0.08	66	0.11	0.10	53	-0.19	0.07	0.04	3
- Cortisol sample 7	0.09	0.07	66	0.11	0.10	51	-0.15	0.08	0.05	3
- Cortisol sample 8	0.10	0.09	61	0.09	0.06	46	0.11	0.08	0.05	3

Note: Statistics based on cases used in primary analyses. Cortisol samples are presented by collection number for simplicity.

	Mean	Total SD	N
Age in Months	69.43	7.09	133
Nights in shelter	32.87	45.38	133
Lifetime Events	3.01	2.15	133
- Direct threats	0.92	0.96	133
- Family threats	1.57	1.33	133
Cumulative Risk	2.05	1.03	133
Executive Function	-0.27	0.79	133
Negative control	0.02	0.02	126
Positive Parenting	0.07	0.01	126
Cognitive task session			
- Cortisol sample 1	0.18	0.13	130
- Cortisol sample 2	0.14	0.10	129
- Cortisol sample 3	0.13	0.11	129
- Cortisol sample 4	0.10	0.07	128
Parent-child task session			
- Cortisol sample 1	0.10	0.09	121
- Cortisol sample 2	0.10	0.09	122
- Cortisol sample 3	0.10	0.08	120
- Cortisol sample 4	0.10	0.07	110

Note: Statistics based on cases used in primary analyses. Cortisol samples are divided by collection number for simplicity sake.

#### **Appendix D. The impact of controlling for year on the relationship between EF, Negative Parenting, and cortisol.**

Cohort (year) differences emerged for a number of variables, including cortisol levels, child age, executive function, and negative control. While efforts were taken to minimize methodological differences between years (e.g., assuring that cortisol was collected using the same batch of cotton both years, and the same antibody was used in assaying), unintentional or uncontrollable differences may have emerged from one year to the next (e.g., conditions associated with shipping all of the samples in 2008 versus 2009). Statistically controlling for year of data collection on cortisol levels and slopes/trajectories served to account for differences that are likely artifacts of method.

However, controlling for year effects on cortisol introduces the risk of reducing true associations between other variables and cortisol. This is especially likely for those variables that differ by year, namely executive function and negative control. I computed a series of regressions to explore the likelihood that controlling for year of data collection occludes relationships between EF and cortisol levels, and between Negative Control and cortisol levels. I provide standardized beta's with and without year considered predicting each cortisol sample (grouped by sample number for simplicity and to increase stability through a higher number of observations). Controlling for year does not seem to meaningfully alter the magnitude of the associations, but seems to strengthen them (not weaken), if anything. (Note: I do not provide similar coefficients for child age, given its use as a control/nuisance variable in primary analyses)

Cortisol Sample Cognitive Session	Standardized Betas			
	Executive Function		Negative Control	
	Without Year	With Year	Without Year	With Year
1	-0.10	-0.14	0.09	0.15
2	-0.05	-0.09	0.04	0.10
3	-0.04	-0.08	0.04	0.09
4	-0.04	-0.10	0.04	0.10
<i>Parent-Child Session</i>				
1	-0.10	-0.14	0.13	0.17
2	-0.11	-0.13	0.21	0.24
3	-0.08	-0.12	0.06	0.10
4	-0.11	-0.13	0.17	0.20

**Appendix E. Potential confounds for cortisol level.**

<u>Potential confounds related to individuals</u>	<u>n</u>	<u>Max r</u>
Any medication	20	0.12
- Not endorsed	110	
Inhaler medication	13	0.14
- Not endorsed	120	
Sick today	7	-0.11
- Not endorsed	126	
Unusually active today	26	0.10
- Not endorsed	106	
In an argument today	76	-0.18
- Not endorsed	57	

Max r = Largest magnitude correlation coefficient between variable and each cortisol sample (either session)

<u>Potential confounds related to samples</u>	<u>Number of Samples</u>	<u>r's</u>	
		<u>Cognitive Session</u>	<u>Parent-Child session</u>
Child rinsed mouth recently	3	0.01	--
More than one collection needed for sample	18	0.00	0.03
Child showing strong affect (e.g., crying)	16	-0.05	-0.01
Sample contains debris	18	0.22	0.24
Sample discolored	64	0.11	-0.06
Sample low in volume	67	0.02	0.03



## **Appendix F. Performance IQ, Verbal ability, and EF.**

It is unclear if differences in cortisol levels would be related to EF abilities specifically, or if the same relationship would hold for cognitive abilities more generally. There exists considerable debate as to whether executive functions constitute a set of distinct cognitive abilities, a cluster of abilities that largely overlap with fluid intelligence or performance/nonverbal IQ; or abilities that are generally indistinguishable from general intelligence: *g* (Best & Miller, 2010; Blair, 2006; Burgess, Braver, & Gray, 2006; Demetriou, 2006; Garlick & Sejnowski, 2006; Garon, Bryson, & Smith, 2008; Heitz et al., 2006). Adding to the difficulty are findings that cognitive abilities, like different EF components or performance versus crystallized/verbal IQ, are less differentiated in preschoolers and become more distinct with age (Best & Miller, 2010; Garon, et al., 2008).

The current analyses (for Aim 2) investigated links between executive function and cortisol, following from past work that has found relationships between these factors among preschool-aged children (Blair, Granger, et al., 2005; Davis, et al., 2002; Wiik, et al., under review). These studies and the broader literature proffer an inverted-U explanation whereby a moderate level of cortisol optimally facilitates neuronal (and cognitive) functioning through maximal occupation of MR receptors without a sizeable occupation of GR receptors, in addition to other possible mechanisms.

An alternative approach to Aim 2 would be to test for distinct associations between cortisol and performance IQ estimate (representing a component of fluid intelligence), verbal ability estimate, and EF. Given the overlap between the construct of fluid intelligence, I would expect performance IQ and EF to account for a similar component of differences in cortisol. Meanwhile, crystallized abilities (like verbal ability) often show a high correlation with performance IQ or fluid intelligence, likely because the factors that encourage good cognitive

functioning generally also tend to encourage the development of good crystallized abilities (Blair, 2002, 2006). It has already been shown in the current dataset that reactive parenting (either positive control or negative control reversed) statistically predicts better child EF but not IQ, and nondirective responsiveness statistically predicts both better EF and better IQ. This suggests that EF seems to be differentiated from IQ (verbal and performance estimates), at least when it comes to some forms of parenting (Herbers, 2011). It is unclear if verbal ability would make a unique contribution to the prediction of cortisol differences, and is treated as an exploratory consideration.

In addition to the EF tasks, children also completed the Block Design and Matrix Reasoning subscales from the Wechsler Preschool and Primary Scales of Intelligence, 3<sup>rd</sup> edition (WPPSI-3) which were averaged to produce an estimate of performance IQ. In addition, each child completed the Peabody Picture Vocabulary Test, 4<sup>th</sup> edition (PPVT). The PPVT is a test of receptive vocabulary and serves as an indication of the children’s verbal functioning.

Correlations (Pearson’s *r*) between EF, the Performance IQ estimate, and Verbal functioning are provided below:

	EF	Perf. IQ	Verbal
EF	--		
Perf. IQ.	0.46	--	
Verbal	0.38	0.38	--

Note: Starting with the 133 children that informed analyses of Aim 2, an additional 3 children were missing Performance IQ scores (*n* = 130), and 3 more children were missing Verbal scores (*n* = 127).

The highest correlation is between EF and Performance IQ (*r* = 0.46) which is somewhat consistent with the view that these two constructs are related to a more general factor of fluid intelligence. Correlations with Verbal functioning were also considerable (*r*’s = 0.38).

First I compared 14 models to test for meaningful separate effects of EF, performance IQ (Perf. IQ), and verbal ability (PPVT) on cortisol intercept and on trajectory over the course of the session of cognitive tasks, controlling for age, sex, and time of session relative to the child waking up:

Fixed Effects	Curve	$\Delta$ AIC	Weight
None	Linear	7.1	< 0.01
None	Quadratic	3.1	0.06
EF: Intercept	Linear	5.5	0.02
<b>EF: Intercept<sup>a</sup></b>	<b>Quadratic</b>	<b>0.2</b>	<b>0.27</b>
Perf. IQ: Intercept	Linear	6.7	0.01
Perf. IQ: Intercept	Quadratic	2.5	0.09
PPVT: Intercept	Linear	7.7	< 0.01
PPVT: Intercept	Quadratic	4.3	0.04
EF: Intercept, Trajectory	Linear	7.5	< 0.01
EF: Intercept, Trajectory	Quadratic	3.6	0.05
Perf. IQ: Intercept, Trajectory	Linear	7.5	< 0.01
Perf. IQ: Intercept, Trajectory	Quadratic	5.4	0.02
<b>PPVT: Intercept, Trajectory<sup>a</sup></b>	<b>Linear</b>	<b>1.7</b>	<b>0.13</b>
PPVT: Intercept, Trajectory <sup>a</sup>	Quadratic	0.0	0.30

Note: Adopted models are in bold.

<sup>a</sup> denotes equivalent models  $\leq 2.0$   $\Delta$ AIC of the best fitting model

The above table suggests three plausible models. First, there appears to be an effect of verbal ability on change in cortisol over the course of the session (fixed effect for the term for the linear model:  $t = -2.87$ ). Higher PPVT scores correspond to a more rapid decline in cortisol over the course of the session. An equally plausible model contains the fixed effect for EF expounded in the main analyses of this study: children with higher levels of cortisol at the start of the session had lower EF scores.

Comparing fixed effects for EF and for Performance IQ on intercept, both models with just an intercept effect for EF provided a better model fit than models that contained just an intercept effect for Performance IQ (relative  $\Delta AIC's > 2$ ). This provides evidence that EF is a better predictor of cortisol than performance IQ as estimated in the current study.

A more telling analysis would be to compare the relative contribution of EF to the prediction of cortisol beyond verbal functioning or performance IQ. I compared nineteen models: the 14 in the above comparison, plus models with fixed effects for PPVT and EF on intercept (quadratic time), with fixed effects for PPVT and EF on intercept and trajectory (quadratic time), with fixed effects for Performance IQ and EF on intercept (quadratic time), with fixed effects for Performance IQ and EF on intercept and trajectory (quadratic time), and with fixed effects for PPVT and EF on intercept and slope (linear time). Results below:

Fixed Effects	Curve	$\Delta$ AIC	Weight
None	Linear	8.0	< 0.01
None	Quadratic	4.0	0.03
EF: Intercept	Linear	6.4	0.01
<b>EF: Intercept<sup>a</sup></b>	<b>Quadratic</b>	<b>1.1</b>	<b>0.14</b>
Perf. IQ: Intercept	Linear	7.6	< 0.01
Perf. IQ: Intercept	Quadratic	3.4	0.05
PPVT: Intercept	Linear	8.6	< 0.01
PPVT: Intercept	Quadratic	5.2	0.02
EF: Intercept, Trajectory	Linear	8.4	< 0.01
EF: Intercept, Trajectory	Quadratic	4.5	0.03
Perf. IQ: Intercept, Trajectory	Linear	8.4	< 0.01
Perf. IQ: Intercept, Trajectory	Quadratic	6.3	0.01
PPVT: Intercept, Trajectory	Linear	2.6	0.07
<b>PPVT: Intercept, Trajectory<sup>a</sup></b>	<b>Quadratic</b>	<b>0.9</b>	<b>0.16</b>
Perf. IQ & EF: Intercept	Quadratic	2.7	0.07
Perf. IQ & EF: Intercept, Trajectory	Quadratic	8.4	< 0.01
PPVT & EF: Intercept	Quadratic	3.2	0.05
<b>PPVT &amp; EF: Intercept, Trajectory<sup>a</sup></b>	<b>Quadratic</b>	<b>0.0</b>	<b>0.26</b>
PPVT & EF: Intercept, Trajectory	Linear	2.4	0.08

<sup>a</sup> denotes equivalent models  $\leq 2.0 \Delta$  AIC of the best fitting model

Three models had equivalent fit: intercept-only fixed effect for EF with quadratic change (as above), verbal effects on quadratic change, and EF and verbal effect on quadratic change. I then reran these analyses to include a 20<sup>th</sup> model that contained an intercept effect of EF and a trajectory (quadratic) effect of verbal ability. This 20<sup>th</sup> model proved to be the best fitting, although not meaningfully different from the model with PPVT and EF effects on trajectory listed above ( $\Delta$  AIC = 1.1; weight = 0.31). Together these findings suggest that there are relatively separate and meaningful effects of EF on intercept, and of verbal ability on trajectory.

The intercept effect of EF on cortisol appears to be distinct from any effect of performance IQ estimate.

These results are the product of mostly post-hoc analyses and consideration. As such, they should be interpreted with caution.

**Appendix G. Why the current study is not a literal replication of Cutuli et al. (2010), and why most of the methodological differences do not seem to matter very much.**

A number of methodological differences must be acknowledged with respect to past work with similarly aged homeless children (Cutuli, Wiik, et al., 2010). Statistical methodology was different between the current study and past work. A model comparison approach and AIC values likely provided a better indication of meaningful effects than the reliance on p-values and the contentious estimation of degrees of freedom in longitudinal mixed modeling with missing data (see (Baayen, et al., 2008; Long, in press)). Nevertheless, a comparison of the t-values between the Cutuli et al. (2010) analysis and the current findings shows that the size of the intercept fixed effect in the previous work is at least twice that in the current findings ( $t = 2.25$  in the previous study versus  $t = 1.06$  in the current analyses). Also, the current study used a more precise estimation of time during the session as well as 4 instead of 3 saliva samples, both of which should help produce more robust estimations. It is unlikely that the failure to replicate can be contributed to differences in statistical methodology alone.

A second consideration acknowledges potential differences in the sheltered population between the Cutuli et al. (2010) study (data collected in 2006) and the current analyses (data collected in 2008 and 2009). The size and characteristics of 'homeless' populations may change in many ways as factors at other levels of analysis shift from year to year (Buckner, 2008). For example, the housing crisis that began in the summer of 2007 and intensified in 2008, along with the resulting national economic recession, likely contributed to increased rates of family homelessness that have existed since. While core characteristics of the population are unlikely to completely abate (e.g., low-income status; high rates of other adversities), it may be that aspects of the population and/or the homeless experience have changed to a degree that have



influenced results. There is evidence of a small decline in cumulative risk scores between the previous and current samples (Cohen's  $d = 0.31$ ). Additional items were added to the Lifetime Life Events Questionnaire for the current data collection to better index lifetime events that directly threaten the child. Comparing just the items used in the Cutuli et al. (2010) study, there is essentially no difference in the number of lifetime events across studies (Past study mean: 2.77; SD = 2.19; Current study mean: 2.67; SD = 2.18; Cohen's  $d = 0.04$ ). Other differences in the homeless population between studies are still possible, but there do not seem to be differences in lifetime event scores, and only a small difference in cumulative risk scores (which have never produced a large effect when predicting cortisol).

Other methodological differences should be acknowledged, such as the inclusion of three family shelters in the current study versus just one in the past work. The sample collected in 2006 was based entirely on families staying at an emergency shelter that largely served county-supported clients with very few turned away for reasons other than the availability of space. A sizeable percentage (44%) of families in the current analyses were staying at a different large shelter for families which is privately owned and operated. Including this additional site probably produces a more representative sample of homeless kindergarten and first graders, but may help account for differences between the past and current studies. Comparing differences between the first site (shared by both studies) and this second site using data from the current study, there was less negative control among families and higher levels of EF among children at the second site. However, there were no differences in a range of other variables listed in Appendix C.

Finally, a measure of verbal functioning changed between studies (WPPSI-vocabulary in the previous study; PPVT-4 in the current study). This may have influenced differences in cortisol

patterns, especially slope as performance PPVT-4 can be shown to influence changes in cortisol decline during the session (see Appendix F). No similar analysis is available for the previous study. However, the characteristics of the PPVT-4 that likely contributed to most of this difference (e.g., frustration- or boredom-causing testing to failure; see (Gunnar, et al., 2009)) are also features of the WPPSI-vocabulary.