Salivary Cortisol Mediates Effects of Poverty and Parenting on Executive Functions in Early Childhood

Clancy Blair
Douglas A. Granger
Michael Willoughby
Roger Mills-Koonce
Martha Cox
Mark T. Greenberg
Katie T. Kivlighan
Christine K. Fortunato
FLP Investigators

October, 2011
Salivary Cortisol Mediates Effects of Poverty and Parenting on Executive Functions in Early Childhood

Clancy Blair\textsuperscript{1,2}, Douglas A. Granger\textsuperscript{3}, Michael Willoughby\textsuperscript{4}, Roger Mills-Koonce\textsuperscript{5}, Martha Cox\textsuperscript{5}, Mark T. Greenberg\textsuperscript{1}, Katie T. Kivlighan\textsuperscript{3}, Christine K. Fortunato\textsuperscript{3}, and the FLP Investigators\textsuperscript{1,4,5}

\textsuperscript{1}Department of Human Development and Family Studies, 110 Henderson South, Pennsylvania State University, University Park PA 16802

\textsuperscript{2}Department of Applied Psychology, 239 Greene St, East Bldg 500, New York University, New York NY 10003

\textsuperscript{3}Department of Biobehavioral Health, 315 Health and Human Development East, Pennsylvania State University, University Park PA 16802

\textsuperscript{4}Frank Porter Graham Child Development Center, 521 S. Greensboro Street, CB 8185

\textsuperscript{5}Center for Developmental Science, 100 E. Franklin St., CB8115, University of North Carolina at Chapel Hill, Chapel Hill NC 27599

Address correspondence to:

Clancy Blair, PhD, 239 Greene Street, East Building, 500, New York, NY 10003

Phone 212-998-5853 clancy.blair@nyu.edu

Acknowledgements: We would like to thank the many families and research assistants that made this study possible. Support for this research was provided by the National Institute of Child Health and Human Development grants R01 HD51502 and P01 HD39667 with co-funding from the National Institute on Drug Abuse.
Abstract

In a predominantly low-income population-based longitudinal sample of 1,292 children followed from birth, higher level of salivary cortisol assessed at ages 7, 15, and 24 months was uniquely associated with lower executive function ability and to a lesser extent IQ at age 3 years. Measures of positive and negative aspects of parenting and household risk were also uniquely related to both executive functions and IQ. The effect of positive parenting on executive functions was partially mediated through cortisol. Typical or resting level of cortisol was increased in African American relative to White participants. In combination with positive and negative parenting and household risk, cortisol mediated effects of African American ethnicity, income-to-need, and maternal education on child cognitive ability.
Salivary Cortisol Mediates Effects of Poverty and Parenting on Executive Functions in Early Childhood

The effect of early experience on cognitive development (Ramey & Ramey, 1998) and on the development of the physiological response to stress (Gunnar & Quevedo, 2007; Meaney & Szyf, 2005) is well established. It is also well established that the physiological response to stress, as indicated by levels of neuroendocrine hormones, glucocorticoids and catecholamines, is related to distinct aspects of cognition, including declarative memory as well as executive functions (Arnsten, 2000; Diamond, Campbell, Park, Halonen, & Zoladz, 2007). No studies to our knowledge, however, have directly examined the extent to which the effects of early experience on stress physiology may mediate some of the well-known effects of early experience on cognitive development.

Relations of early experience to stress physiology and to executive functions are of particular interest in that executive functions are cognitive abilities associated with prefrontal cortex (PFC), including working memory, inhibitory control, and attention set shifting or flexibility, that enable the organization of information in goal-directed activities. Executive functions contribute substantially to the self-regulation of behavior (Carlson, Mandell, & Williams, 2004; Hughes & Ensor, 2007) and are central to early academic achievement (Blair & Razza, 2007; Diamond, Barnett, Thomas, & Munro, 2007). They are also a primary aspect of cognitive disability in a range of psychological disorders in children and adults (Zelazo & Muller, 2002).

In humans the link between stress hormone levels and executive functions has been demonstrated in naturalistic studies with preschool children (Blair, Granger, & Razza, 2005; Davis, Bruce, & Gunnar, 2002) and in pharmacological manipulations with adults (Alexander,
Cortisol and Cognition

Hillier, Smith, Tivarus, & Beversdorf, 2007; Lupien, Gillin, & Hauger, 1999; Young, Sahakian, Robbins, & Cowen, 1999). The association between executive functions and stress physiology in part reflects the fact that stress hormone levels modulate synaptic activity in the neural circuitry of PFC that underlies executive functions (Arnsten & Li, 2005; Mizoguchi, Ishige, Takeda, Aburada, & Tabira, 2004). Although by no means the only brain area and cognitive ability affected by stress hormones (Diamond et al., 2007), PFC and executive functions are sensitive indicators of the effects of stress on development (Cerqueira, Mailliet, Almeida, Jay, & Sousa, 2007). Importantly, under conditions of ongoing or persistent stress, basal levels or set points of physiological stress response systems are altered either upward or downward, a phenomenon referred to as allostasis (McEwen & Wingfield, 2003). Stress physiology, including the hypothalamic-pituitary-adrenal (HPA) axis, is highly influenced by social interaction (Dickerson & Kemeny, 2004; Gunnar & Donzella, 2002) and conditions that are threatening, unpredictable, and lacking in support are associated with elevated levels of glucocorticoid hormones, namely cortisol. When stressful conditions are chronic or persistent, stress response systems are said to be under high allostatic load and adapt to the environment with over or under activation to an extent that impedes flexible regulation of stress physiology (McEwen, 1998, 2000), such as that associated with the self-regulation of behavior and executive functions (Ramos & Arnsten, 2007).

Poverty and child development. The environment of poverty is stressful for children and to date has been associated with increased levels of stress hormones (Evans, 2003). It is important to note that severe disruption of caregiving has been shown to result in under rather than over activation of stress response systems, as seen in altered diurnal variation in cortisol in children experiencing early caregiving adversity (Tarullo & Gunnar, 2006). In the far from
optimal but essentially adequate (i.e., not extreme adversity) conditions of poverty, however, increases in stress physiology are typically observed (Evans & English, 2002; Lupien, King, Meaney, & McEwen, 200) and likely represent a pathway through which poverty affects child development (Repetti, Taylor, & Seaman, 2004). Recent quasi-experimental evidence of the relation of poverty to stress physiology in a Mexican sample indicated lower cortisol levels in preschool children in homes in participating in a conditional cash transfer program relative to a matched comparison group (Fernald & Gunnar, 2009).

An important question for child development research concerns sources of stress in children’s lives. As noted above, the HPA axis is under strong social regulation in childhood and it may be that parenting acts as a primary pathway through which the conditions of poverty lead to increases in stress physiology in children. Research in animal models indicates early caregiving to be a primary influence on the development of the HPA axis component of the stress response system and on cognitive and behavioral flexibility associated with this system (Champagne et al., 2008; Liu, Diorio, Day, Francis, & Meaney, 200). Maternal behavior is known to adaptively shape offspring development to meet an expected environmental quality across a wide variety of species (Cameron, Champagne, Parent, Fish, Ozaki-Kuroda, & Meaney, 200). In humans, early sensitive parenting that appropriately scaffolds learning experiences and provides a nurturing and supportive environment is associated with a well regulated physiological response to stress (Gunnar & Quevedo, 2007) and with higher level of general cognitive (Lugo-Gil & Tamis-Lemonda, 2008) and emotion regulation ability (Sroufe, 2009).

Socioeconomic disadvantage is associated with lower parenting quality and more reactive and less effortfully regulated behavior in children (McLoyd, 200). The impact of stressful conditions of the low SES home environment on parenting is a primary mechanism
through which poverty affects child development (Brody, Murry, Kim, & Brown, 2002; Gershoff, Aber, Raver, & Lennon, 2007). Therefore, parenting likely mediates an association between poverty and elevated stress in young children. Such a mediating model for the association between poverty and the physiological response to stress in children does not preclude the possibility that high quality parenting might moderate effects of environmental stressors external to the parent-child relationship. As well, it does not indicate the mechanism or mechanisms through which caregiving is related to stress reactivity and regulation early in development (Tang, Akers, Reeb, Romeo, & McEwen, 2006). Early intervention programs have established that high quality care involving educational stimulation moderates effects of poverty on child development (Landry, Smith & Swank, 2006; Ramey & Ramey, 1998). However, given that environmental stressors and lower parenting quality tend to covary in families in poverty (Conger, Wallace, Sun, Simon, McLoyd, & Brody, 2002; Mistry, Biesanz, Taylor, Burchinal, & Cox, 2004) and parenting is a predominant influence on development in early childhood, it is likely that the typical or general process in the infant and toddler periods is one of mediation rather than moderation.

Positive and negative dimensions of parenting. When examining parenting quality as a potential mediator for the effects of poverty on child stress physiology and cognitive development, it is important to consider both positive and negative dimensions of the construct. Although highly related, positive and negative dimensions of parenting can be expected to have distinct effects on child development. A positive, responsive and emotionally supportive parent provides an interactive environment for young children to engage in reciprocal verbal and nonverbal exchanges that are stimulating and rewarding (Landry, Smith, & Swank, 2006; Tamis-LeMonda, Bornstein, & Baumwell, 2001). In contrast, negative and intrusive parenting focuses
Cortisol and Cognition

on the degree to which parents intrude on children’s interests and behaviors above and beyond the developmental or safety needs of the child and undermine autonomy and nascent attempts at self-regulation (Egeland, Pianta, & O’Brien, 1993; Ispa, Fine, Halgunseth, Harper, Robinson, Boyce, Brooks-Gunn, & Brady-Smith, 2004). It is likely that both positive and negative aspects of parenting uniquely influence the development of stress physiology and early cognitive ability.

*Parenting, stress physiology, and cognitive ability in children.* Although relations between early care and cognitive development are well established in children, relatively little is known about relations between stress physiology and cognitive ability in early childhood. The primary goal of this study is to examine the longitudinal relation of what can be considered a typical level for salivary cortisol measured across the child’s first two years to cognitive ability at age 3. Cortisol levels follow a pronounced circadian rhythm and are affected by various aspects of children’s experiences. However, by collecting saliva samples from children in the same way at the same time point in a carefully planned data collection, usually by the same data collector at generally near the same time of day over the child’s first two years and statistically controlling for time of day in our analysis we are able to estimate a reasonably stable component of each child’s level of cortisol. No prior studies of which we are aware have examined longitudinal relations between cortisol and cognition in early childhood. Given that cognitive ability is associated with both early parenting behavior and aspects of stress physiology, it may be that some of the effect of parenting on cognition is mediated through the effect of parenting on stress physiology. Therefore, a second goal of this study is to examine the relation of early parenting to both cortisol and cognitive ability, particularly executive functions but also intelligence, and to determine whether cortisol mediates some of the effect of parenting on cognitive ability. Although executive functions are largely distinct from general intelligence
(Blair, 2006), the executive function of working memory has been shown to be highly related to the general factor, or g, in a number of studies with adults (Friedman, Miyake, Corley, Young, Defries, & Hewitt, 2006). The relation between executive functions and intelligence is not well studied in children but available evidence indicates that the development of working memory ability underlies the development of the aspect of intelligence referred to as fluid intelligence (Fry & Hale, 1996; Kail, 2007), which is important for reasoning ability and the processing of novel information and is itself highly related to general intelligence (Carroll, 1993).

Finally, given well documented effects of poverty on child cognitive development (Bradley & Corwyn, 2001), a third goal of this analysis is to examine the extent to which the effects of poverty on child cognitive ability are mediated through positive and negative parenting and child cortisol. As outlined above, we expect that both positive and negative aspects of parenting quality are mediators of the effect of poverty, as indicated by income-to-need and maternal education, on child outcomes. As noted above, however, parenting, although predominant, is only one possible route through which poverty affects stress physiology and cognitive ability. Therefore, we include measures of household crowding or density (number of persons / number of rooms) and data collectors’ ratings of the safety and noise level of the home and the area around the home to determine the extent to which these characteristics of the home may account for relations among poverty, stress physiology, and child cognitive ability.

Furthermore, most if not all prior studies of poverty and stress physiology have been conducted with white low-income samples. We addressed this aspect of the literature by examining the relation of poverty to cortisol and child development in a sample of African American as well as white participants in two geographically distinct regions of high poverty in the U.S. Prior analyses conducted with longitudinal samples have demonstrated that risk
processes in the context of poverty work similarly in African American and white families (Conger et al., 2002; Raver, Gershoff, & Aber, 2007). Given the overrepresentation of African American families in deep and persistent poverty in the U.S. (McLoyd, 1998), however, we expect that African American ethnicity in this sample will serve as a marker for a number of unmeasured aspects of risk associated with socioeconomic disadvantage and social inequality.

In sum, we examine longitudinal relations of cortisol and early parenting measured in infancy and toddlerhood to cognitive ability at age 3 years and the extent to which relations among these constructs account for relations of poverty indicators to child outcomes. A prior analysis of the sample reported on here demonstrated that high level of positive but not negative parenting in infancy is associated with lower basal levels of cortisol and greater cortisol reactivity to emotional arousal at age 7 and 15 months (Blair et al., 2008). In that prior analysis we also found that typical or resting level of cortisol was increased for African American children, likely reflecting conditions of increased risk associated with deep poverty in African American families in this sample. This report expands on these findings to include measurement of cortisol at a subsequent time point, at child age 24 months. The focus in this report, however, is on typical level for cortisol adjusted for time of day of saliva collection over the child’s first two years rather than cortisol reactivity at each time point. We expected that positive parenting would be associated with lower level of cortisol, adjusted for time of day of saliva collection, and negative parenting would be associated with higher level of cortisol. Further, we expected that cortisol level would account for significant variance in both executive functions and IQ and partially mediate effects of positive and negative parenting on both aspects of cognitive ability. Additionally, given established relations between cortisol and executive functions specifically, we expected that the relation of cortisol to executive functions would be greater than that for IQ.
Finally, we expected that effects of parenting and cortisol levels on executive functions and IQ would mediate effects of poverty indicators on child cognitive ability, including income-to-need, maternal education, and household risk and in this sample African American ethnicity.

Method

Participants

Recruitment. Complex sampling procedures were used to recruit a representative sample of 1,292 families in two regions of the U.S. at the time that mothers gave birth to a child. Low-income families in both regions and African American families in one region were over-sampled. African-American families were not over-sampled in the second region as the target communities were 95+% Caucasian. Further details on the sampling plan and recruitment procedures are available in Burchinal, Vernon-Feagans, Cox and the FLP Investigators (2008b). Based on the mothers’ ethnic status, the sample was 58% Caucasian and 42% African American and 66.6% of the sample had an income-to-need ratio less than 200% of poverty. Most (51.9%) of the mothers were not married at the time the study began and the majority (88.8%) of single mothers had never been married.

Procedures

Families were seen in home visits at child ages of approximately 7, 15, 24, and 36 months. At all time points except 15 months, families were seen in two separate visits. All home visits for data collection were two or more hours in duration. During visits for data collection conducted at 7, 15, and 24 months, mothers completed questionnaires concerning family demographics, income, and child temperament, and engaged in a free play interaction (at 7 and 15 months) and an interactive puzzle completion task (at 24 months) with their child that was recorded with digital video for 10 minutes (Cox, Paley, Burchinal, & Payne, 1999; NICHD
ECCRN, 1999). During the free play interaction mothers were given a standard set of toys and instructed to play with the child as they normally would if they had a little free time during the day. During the puzzle completion task, children were presented with 3 consecutive board puzzles that increased in difficulty. Mothers were instructed to interact and help their children with the puzzles as they saw necessary.

Near the conclusion of the home visit for data collection at 7, 15, and 24 months (usually the second visit at 7 months, usually the first visit at 24 months), at which time the data collectors had been in the home for at least one hour, children were presented with emotion challenge tasks designed to elicit emotional responding, including a mask presentation, barrier task, and arm restraint at 7 months, and a toy removal and mask presentation at 15 and 24 months. All procedures have been previously validated (Stifter & Braungart, 1995). To assess basal levels of cortisol and cortisol response to the emotion arousal, unstimulated whole saliva was collected using either cotton or hydrocellulose absorbent material and expressing sample into 2 ml cryogenic storage vials using a needleless syringe (cotton) or by centrifugation (hydrocellulose). Two prior studies have indicated no differences in cortisol concentrations associated with the two collection techniques (Granger, Kivlighan, Fortunato, Harmon, Hibel, Schwartz & Whembolua, 2007; Harmon, Granger, Hibel & Rumyantseva, 2007). Saliva was collected at baseline prior to the administration of the emotion challenge procedures and at 20 and 40 minutes post peak emotional arousal following exposure to the procedures.

For this analysis, only the baseline cortisol measures adjusted for time of day of collection were used. Although the 20- and 40-minute post peak arousal measures of cortisol are available, we focus on baseline levels in order to most directly address hypotheses concerning processes of allostatic load and variation in typical level or set point for cortisol over the child’s
first two years. Furthermore, whereas the emotion arousal procedures resulted in emotional and cortisol reactivity in the sample as a whole at 7 and 15 months of age, the procedures did not produce substantial emotional arousal or increase in cortisol in the sample at 24 months.

The characteristics of the sample, repeated interview schedule, length of each interview protocol (2-4 hours), and age of the infants required that in-home assessments were scheduled when families were available. Therefore, time of the day of the interview and saliva collection varied. Mean time of day of saliva sample collection was 13:04 hours (SD = 2.88) at age 7 months, 13:45 hours (SD = 2.94) at 15 months, and was 13:33 hours (SD = 3.20) at 24 months. Time of day of sample collection was moderately correlated between time points, $r_{7,15} = .23$, $r_{15,24} = .21$. Collection of saliva always occurred near the end of the home visit for data collection. After collection, samples were immediately placed on ice, transported to interviewers homes and frozen (−20 °C). They were stored frozen until batched and shipped on dry-ice overnight to the Behavioral Endocrinology Laboratory at Penn State. Samples were then stored frozen at −80 °C until assay. On the day of testing, samples were brought to room temperature, centrifuged at 3,000 RPM for 15 minutes, and the clear top-phase of the sample was pipetted into appropriate test wells by robot (Genesis, Tecan).

At approximately 36 months of age, children were administered tasks to assess executive functions and IQ. Children were seated across from the experimenter at a convenient location in the home. All tasks were administered in a standard order. The executive function tasks were administered at the conclusion of an assessment session in which children also completed a series of tasks with the mother that included a picture book reading task, an empathy task, and a puzzle task. Cumulatively, these tasks took about one hour to complete.
Measures

Executive function was assessed with three tasks modeled on tasks previously used successfully with young children. These included a span-type working memory (WM) task, an item selection attentional flexibility (AF) or set shifting task, and a spatial conflict inhibitory control (IC) task. Children were also administered a go no-go task in which they were asked to selectively withhold responding to a specific stimulus and a Stroop like task which required the inhibition of a prepotent response. Rates of completion on these latter tasks were too low to warrant inclusion.

In the working memory task, children must hold in mind two pieces of information simultaneously and activate one while overcoming interference from the other. In the task, children are presented with a line drawing of an animal figure above which is a color dot. Both the animal and color dot are located within the outline of a house. After establishing that the child knows both colors and animals in a pretest phase, the examiner asks the child to name the animal and then to name the color. The examiner then flips a page containing only the outline of the house to cover the page with the animal and the color dot. The examiner then asks the child which animal was/is/lives in the house. The task requires children to perform the operation of naming and holding in mind two pieces of information simultaneously and to activate the animal name while overcoming interference occurring from naming the color.

The Item Selection task is modeled on the Flexible Item Selection Task developed by Jacques and Zelazo (2001). In the version of the task developed for flipbook administration, children are first presented with a page on which there are two line drawn items that are identical in terms of shape, size or color. The examiner draws the child’s attention to the dimension along which the items are identical, stating “See, here are two pictures. These pictures are the same,
they are both (cats, blue, big, etc.)”. The examiner then flips a page which presents the same two items again, to the right of which is a dashed vertical line and a picture of a third item. The new third item is similar to one of the first two items along a second dimension that is different from the similarity of the first two items (e.g., if the first two items were identical in shape, the third item would be identical to one of the first two items in either size or color.) When presenting the new, third item to the child the examiner states to the child, “See, here is a new picture. The new picture is the same as one of these two pictures. Show me which of these two pictures is the same as this new picture?” Percent correct responding on 14 trials was used for analysis. This task is preceded by a pretest in which children demonstrate knowledge of color, shape, and size.

The Spatial Conflict task is a Simon task similar to that used by Gerardi-Caulton (2000) and Diamond et al. (2007) in which children alternate same-side and opposite side responding to line drawings of a toy car and a toy boat. A picture of the car is placed in front of the child on the right and a picture of the boat is placed in front of the child on the left. The examiner then flips pages on which are printed pictures of the car or the boat in either the same side or the opposite side position. Children are presented with 16 same side trials and then are presented with 16 intermixed same side and opposite side trials. Percent correct responding on opposite side trials was used for analysis. This task is preceded by a pretest phase in which children respond to centrally presented line drawings (a sun and a star) to left (sun) and right (star) targets.

As is standard for executive function measures with children (Zelazo, 2006), for all tasks children were required to successfully complete pretest trials in which they clearly demonstrated knowledge of the rules for the task and the ability to successfully complete the pretest trials as instructed. Children were also required to complete 75% of test trials in a given task in order to receive a score for that task. Of 1,105 children administered the executive function tasks, 764
successfully completed the working memory span task, 795 successfully completed the attention flexibility task, and 866 successfully completed the spatial conflict inhibitory control task. For the go no-go and the Stroop-like tasks, only 465 and 497 children met criteria for completion. All tasks were scored as percent correct responding: WM, $M=.27$, $SD=.25$ for 11 items administered as one 1-item, two 2-item, and two 3-item trials; AF, $M=.52$, $SD=.24$ on 15 dimensional shift trials; IC, $M=.66$, $SD=.26$ on 12 opposite side responding trials. Scores were moderately correlated ($r = .22 - .32$, $p < .001$).

**Intelligence.** The receptive verbal ability and block design subscales of the Wechsler Preschool and Primary Scales of Intelligence (WPPSI; Wechsler, 2002) were used to assess child intelligence at age 36 months.

**Income-to-need** was calculated as the estimated total household income divided by the federal poverty threshold for 2005 adjusted for number of persons in the home. Income-to-need was highly correlated across time points ($r = .80$, $p < .0001$) and averaged at child ages 7, 15, and 24 months to create a single indicator.

**Salivary cortisol.** All samples were assayed for salivary cortisol using a highly-sensitive enzyme immunoassay US FDA 510k cleared for use as an in vitro diagnostic measure of adrenal function (Salimetrics, State College, PA). The test used 25 µl of saliva (for singlet determinations), had a range of sensitivity from .007 to 1.8 µg/dl, and average intra-and inter-assay coefficients of variation of less than 10% and 15%. All samples were assayed in duplicate. The criterion for repeat testing was variation between duplicates greater than 20%, and the average of the duplicates was used in all analyses. The cortisol distributions were subject to log transformation to correct positive skew. Outliers greater than 3 standard deviations from the mean were treated as missing (n = 15, 16, and 17 at 7, 15, and 24 months.) Time of day of saliva
collection was significantly related to cortisol level at each time point, \( r = -.25, -.19, -.32, \) all \( p < .01 \) at 7, 15, and 24 months. We also examined child temperature, time since eating, time since sleeping, and use of medications (e.g., acetaminophen) as influences on child cortisol levels at 7 and 24 months (data not available at 15 months.) Small significant relations of time since eating and time since sleeping with cortisol at 7 months were accounted for by adjustment for time of day of saliva collection.

*Parenting.* Mother-child interactions in the free play at 7 and 15 months and in the structured interaction at 24 months were coded to assess levels of mothers’ sensitivity, detachment, intrusiveness, positive regard, negative regard, and animation in interacting with the child. Ratings for each code were made on a 1-5 scale at 7 and 15 months and a 1-7 scale at 24 months, with one being *not at all characteristic* and five (or seven) being *highly characteristic.* Factor analyses conducted with an oblique rotation (i.e., Promax) at each time point indicated distinct positive and negative dimensions of parenting. Maternal positive parenting included five maternal characteristics: sensitivity, detachment (reverse-scored), positive regard (e.g., positive feelings expressed toward child), animation (level of energy), and stimulation for development (appropriate level of scaffolding of activities with child). Maternal negative parenting included two maternal characteristics: intrusiveness and negative regard (level of harsh, negative feelings expressed toward child). Inter-rater reliability was determined by calculating the intra-class correlation (ICC) for ratings made by two coders to approximately 30% of the tapes randomly drawn at the infant and toddler assessments. ICCs were .85 - .91 for positive parenting and .72 - .86 across 7, 15, and 24 month assessments.

*Household risk characteristics.* Information on the number of persons residing in the home was obtained from the primary caregiver in response to a structured questionnaire.
Information on the number of rooms in the home and safety and noise level of the home and neighborhood were obtained from data collector ratings completed at the conclusion of data collection in the home at 7 and 24 months. Density was calculated by dividing the number of persons in the home by the number of rooms. Safety and noise level ratings were combined to create an overall rating ranging from 1 (very unsafe/very noisy) to 4 (very safe/quiet).

Data analysis. Total sample size recruited at study entry was 1,292 with 1,204 children seen at age 7 months, 1,169 at 15 months, 1,144 at 24 months, and 1,123 at 36 months. To assess possible differential attrition in the sample at each time point we examined a number of variables for which we had complete information collected at child age of approximately 2 months including state of residence, race, sex, child age at the 2 month follow-up, an income screen, total number of household members, number of children in the household, and primary caregiver age, education, marital status, and employment. Few variables indicated differences between families who were present and those who were missing at each time point. At the 7 month assessment, missing participants were more likely to have been older at the 2 month follow-up, to have resided in North Carolina, and to have a primary caregiver who was employed. At 15 months, no variables differentiated participants who were missing from those who were present. At 24 months, missing participants were more likely have been older at the 2 month follow-up, to have resided in North Carolina, and to have a primary caregiver who was employed. At 36 months, missing participants were more likely to be male. To avoid bias in estimates associated with listwise deletion we used full information maximum likelihood estimation for all analyses. Structural equation models were estimated using Mplus 5.1 and tests of mediation were conducted using MacKinnon’s (2008) conceptualization of mediation in which indirect effects
involve Sobel tests in order to evaluate the statistical significance of the product of coefficients linking the focal to the outcome variable through the mediating variable.

Results

Preliminary analysis

Table 1 presents means and standard deviations and Table 2 correlation among the variables in the analysis. The measure of executive function in the tables is the mean percent correct responding on the operation span, spatial conflict, and dimensional set shifting tasks. IQ is the full scale estimate derived from the WPPSI Block Design and Vocabulary subtests. These are presented for descriptive purposes here and are examined as latent variables below. Cortisol measures at each time point are log transformed and adjusted for time of day of saliva sample collection. The table indicates that both executive function and IQ at 36 months have small negative correlations with cortisol at 7, 15, and 24 months. The measures of parenting at child ages 7, 15, and 24 months, particularly positive parenting, have small correlations with cortisol at most time points. Positive and negative parenting at each time point are moderately correlated with executive function and IQ at child age 36 months. Maternal education, family income, and the household risk variables (density and safety) are moderately correlated with parenting and with the cognitive measures. Executive function and IQ are moderately correlated.

Notable in the table is the high level of risk associated with African American ethnicity in this sample. On every indicator examined, African American children and families fare worse than do White participants. Levels of cortisol are significantly higher at each time point and child executive function and IQ significantly lower. Ratings of positive parenting are significantly lower and negative parenting significantly higher. Income-to-need and maternal education are
significantly lower for African Americans and African American families’ homes are significantly more crowded and rated as less safe than are the homes of White participants.

*Structural equation modeling*

To examine the relation of cognitive ability at age 3 years to child cortisol adjusted for time of day of data collection, observed parenting at 7, 15, and 24 months, and household risk at 7 and 24 months, we used structural equation modeling. We modeled executive function using a single latent variable with the working memory, inhibitory control, and attentional set shifting tasks as indicators. Similarly, IQ was modeled using a single latent variable with the Block Design and Vocabulary subtests as indicators. The latent cortisol and positive and negative parenting variables were indicated by measures at 7, 15, and 24 months. All cortisol measures were adjusted for time of day of saliva sample collection and log transformed. The latent household risk variable was indicated by measures of household density and the combined noise and safety rating at 7 and 24 months. We also included observed independent variables for mean income-to-need, maternal education measured at child age 3 years, African American ethnicity, child age at the 3 year assessment ($M = 37.05, SD = 1.8$), and child sex.

*Measurement model.* A measurement model with correlations among all latent indicators and observed variables fit the data well $\chi^2 (174) = 467.0, p = .0001$, CFI = .95, RMSEA = .036. Correlations are presented in Table 3 and loadings of observed variables on latent indicators are reported in Table 4. Inspection of parameter estimates indicated that all of the factor loadings were statistically significant and in the expected direction and that all of the latent variances were statistically significant. All correlations between latent variables were large and significant (all $p < .001$.) Correlation between EF and IQ latent variables was very high ($\phi = .92$), a finding consistent with numerous studies examining relations between latent variables for executive
functions, particularly working memory, and intelligence. The executive function latent variable was moderately correlated with the latent positive ($\phi = .59$) and negative ($\phi = -.59$) parenting variables as was IQ ($\phi = .61$ and -.61). Both executive function and IQ were also correlated with the latent variable indicated by household risk ($\phi = -.44$ and = -.53, respectively). Executive function and IQ were negatively related to the cortisol latent variable, with a larger relation for executive function ($\phi = -.56$) than for IQ ($\phi = -.37$). Cortisol was negatively related to positive parenting ($\phi = -.47$), and positively related to negative parenting ($\phi = .37$) and household risk ($\phi = .26$).

**Structural model.** In Figure 1 we present our hypothesized structural model. In this model we examined direct effects of poverty indicators, including income-to-need, maternal education, and African American ethnicity on all latent variables (direct paths of each poverty indicator to executive function and IQ are indicated jointly in the figure for clarity of presentation.) We also examined indirect effects of poverty indicators on executive function and IQ through household risk, positive and negative parenting, and cortisol to determine the extent to which these variables mediate effects of poverty on child cognitive outcomes. As well, indirect effects of household risk through positive and negative parenting and cortisol were examined. Finally, to examine the extent to which cortisol mediates effects of positive and negative parenting on child cognitive outcomes, we examined indirect effects of positive and negative parenting on executive function and IQ through cortisol.

The observed structural model is presented in Figure 2. This model fit the data well, $\chi^2 (188) = 474.4, p = .0001$, CFI = .94, RMSEA = .035, SRMR = .031. All effects are reported as standardized coefficients. **Direct effects** were observed in which executive function was negatively predicted by cortisol, $\beta = -.42, p < .0001$, and negative parenting, $\beta = -.26, p = .003$,
and positively predicted by positive parenting, $\beta = .19, p = .04$. Executive function was also
marginally negatively predicted by household risk, $\beta = -.12, p = .07$, but was not directly
predicted by observed variables maternal education, income-to-need ratio, African American
ethnicity, age at assessment, or sex.

In contrast, IQ was positively predicted by positive parenting, $\beta = .26, p < .0001$, and was
negatively predicted by negative parenting, $\beta = -.27, p < .0001$, and household risk, $\beta = -.25, p <
.0001$, and was marginally negatively predicted by cortisol, $\beta = -.15, p = .06$. IQ was not directly
predicted by maternal education, income-to-need ratio, or African American ethnicity, but males
had significantly lower IQ compared to females, $\beta = -.15, p < .0001$.

In the model in Figure 2, we also tested for *indirect effects* of latent and observed
variables on executive function and IQ, summarized in Table 5. Specifically, as noted above, we
were interested in the extent to which effects for cortisol on executive function mediated effects
for parenting on this aspect of child cognitive ability. We were also interested in the extent to
which associations of cortisol and parenting with child cognitive ability mediated the conditions
of poverty, including income-to-need, maternal education, household risk, and in this sample
African American ethnicity, on child outcomes.

*Does cortisol mediate the effects of parenting on child cognitive ability?* Examination of
direct effects of positive and negative parenting and household risk latent variables on the
cortisol latent variable indicated that cortisol was inversely related to positive parenting, $\beta = -.32,
p = .008$, but was unrelated to negative parenting and to household risk. The relation between
positive parenting and cortisol resulted in an indirect effect of positive parenting on executive
function through cortisol, $\beta = .13, p = .03$. The test of the indirect effect of positive parenting on
IQ through cortisol was not significant, $\beta = .05, p = .12$. 
Do cortisol and parenting variables mediate the effects of poverty including income-to-need, maternal education, household risk, and African American ethnicity on child cognitive ability? Analysis of indirect effects indicated that income-to-need was related to executive function through negative parenting, $\beta = .04, p = .02$, but not through positive parenting or through the path including positive parenting and cortisol. In contrast, maternal education was indirectly related to executive function through negative parenting, $\beta = .07, p = .004$, and also through positive parenting, $\beta = .07, p = .03$, and through the path including positive parenting and cortisol, $\beta = .05, p = .04$.

Income-to-need and maternal education were both indirectly related to IQ through positive parenting, $\beta = .03, p = .03$ and $\beta = .06, p = .008$, negative parenting, $\beta = .04, p = .01$ and $\beta = .07, p = .002$, and household risk, $\beta = .10, p < .0001$ and $\beta = .08, p < .0001$, respectively.

Indirect effects of African American ethnicity on executive function were observed through negative parenting, $\beta = -.10, p = .006$, positive parenting, $\beta = -.05, p = .05$, and through the path including positive parenting and cortisol, $\beta = -.04, p = .04$. Similarly, indirect effects of African American ethnicity on IQ were observed through negative parenting, $\beta = -.10, p < .0001$, positive parenting, $\beta = -.07, p < .0001$, and also through household risk, $\beta = -.04, p = .001$.

Cortisol was higher in African American children, $\beta = .46, p < .0001$. Cortisol was unrelated to income-to-need and maternal education. Higher level of cortisol in African American relative to White participants in this sample resulted in an indirect effect of African American ethnicity on executive function through cortisol, $\beta = -.19, p < .0001$. This indirect effect was not present for IQ.
Discussion

In this analysis, level of salivary cortisol as measured at child ages 7, 15, and 24 months and adjusted for time of day of saliva sample collection was significantly higher in children in poverty and shown to partially mediate effects of poverty and parenting on child cognitive ability. These associations are consistent with well defined neurobiological models linking early experience with the development of the HPA axis component of the stress response system (Meaney & Szyf, 2005) and linking stress and stress hormones with cognition, particularly executive functions (Arnsten, 2000; Lupien, Maheu, Tu, Fiocco, & Shramek, 2007). The novel contributions of this analysis are in demonstrating 1) that relations between the glucocorticoid hormone cortisol and cognitive ability are present in early childhood, 2) that the effect of parenting on cortisol is associated with positive rather than negative aspects of parenting behavior, and 3) that cortisol and both positive and negative parenting mediate associations between the conditions of poverty, including African American ethnicity, likely a marker for deep poverty in this sample, and child cognitive ability at age 3 years.

Findings from this analysis extend the study of poverty, stress physiology, and executive functions to early childhood and provide increased specificity in the identification of relations among variables. As noted in the introduction, several studies have demonstrated that measures of what can be considered the typical rearing environment of poverty are associated with increased levels in physiological stress response systems in children (Evans, 2003; Fernald & Gunnar, 2009; Lupien, King, Meaney, & McEwen, 2001). Only one prior study (with an adolescent sample) has reported effects linking chronic poverty assessed as income-to-need with both stress physiology and cognitive ability (Evans & Schamberg, 2009). No previous study of which we are aware has examined multiple aspects of the context of poverty in a sample in early
childhood with the goal of linking poverty with stress physiology and cognitive development. The analysis presented here examined both positive and negative aspects of parenting and found that positive parenting was reduced in lower income homes and inversely related to cortisol level over the child’s first two years. This finding suggests that aspects of parenting such as the provision of nurturance and opportunities for stimulation rather than negative aspects of parenting, including intrusiveness and negative regard, are most relevant to child stress levels. It was expected that maternal negative behavior would also impact child stress physiology. It was also assumed that adverse physical characteristics of the home would be associated with increased levels of cortisol in children. Although both maternal negativity and household risk were significant predictors of child cognitive ability and mediators of the effects of poverty on child outcomes, their effects were independent of those associated with salivary cortisol. The absence of relations here is surprising and it may be that positive aspects of parenting were more comprehensively and accurately measured than were negative parenting and household risk.

Be this as it may, findings emphasizing the relation of positive parental behavior to child stress physiology are consistent with data from animal models indicating it is the absence of nurturing behavior rather than high levels of negative parenting that may be most relevant to development (Cameron et al., 2005; Meaney, 2001). It is not clear in the present study, however, whether positive maternal behavior is affecting stress physiology through a tactile and kinesthetic nurturing process or whether other aspects of parenting behavior, such as structuring of opportunities and appropriate levels of stimulation are the operative mechanism, or even perhaps if positive parenting behavior is a marker for other aspects of early experience important for the development of stress physiology such as exposure to novelty (Tang et al., 2006) and
types of experience that promote flexible regulation of stress physiology (e.g., Parker, Buckmaster, Sundlass, Schatzberg, & Lyons, 2006).

*Increased cortisol levels in African American children.* Although findings confirm expected relations among poverty, parenting, stress physiology, and cognitive ability in children, no studies to our knowledge have previously examined these associations in an ethnically diverse sample. A major finding of this analysis is that typical level or set point for cortisol is higher in African American children than in white children in infancy and early childhood and that higher cortisol partially mediates an association between African American ethnicity and lower executive function ability at age 3 years. The percentage of African American families in poverty in the U.S. is highly disproportionate to their representation in the population as a whole and deficits in measured intelligence and academic achievement among African Americans relative to the U.S. population continue to be topics of intense interest and concern (Nisbett, 2009). This analysis provides evidence indicating that high levels of poverty experienced by African Americans in the U.S. are associated with increases in stress and with stress hormones such as cortisol that can impede the development of cognitive abilities, including executive functions. In so doing, it suggests that increases in stress physiology associated with poverty in African Americans might contribute to longstanding racial gaps in cognitive ability and school achievement. We cannot explicitly test the extent to which the effect of ethnicity is accounted for by poverty, however, due to the low representation of higher income African American participants in our sample, a characteristic of the communities from which the sample was drawn. Alternatively, it may be the case that the data reflect some preexisting differences between African American and White participants in the sample relating to genetic background and to epigenetic processes of development. The two possibilities are not mutually exclusive.
Unmeasured psychological risk in the African American sample in this study might include proximal influences of inequality that act directly on stress physiology and cognitive development but also intergenerational influences of social inequality on physiological stress response systems such as have been hypothesized to contribute to persistent racial disparities in birth and health outcomes (Kuzawa & Sweet, 2009; Lu & Halfon, 2003). Such an intergenerational mechanism may be one contributor to long standing disparities in health and educational outcomes in African Americans relative to the U.S. population as a whole. In combination with poor quality schools and reduced educational and employment opportunities, findings for higher cortisol levels in African American children in this sample may describe one process through which social inequality perpetuates racial disparities in physical and mental health and well being.

*Biological sensitivity to context.* Associations among poverty, African American ethnicity, cortisol, and cognitive ability take on particular meaning in that the mechanisms through which physiological stress affects physical and mental health are well established. The concept of allostasis describes a process by which stress physiology responds to repeated activation by adjusting resting levels or set points upward (McEwen, 2000). Increased levels, although adaptive in responding to stress in the short term are ultimately injurious to health and psychological well being, leading to wear and tear and to a decreased ability to respond to stress as needed. Consistent with an understanding of allostasis as adaptation of stress physiology to environmental demands, results linking poverty and early experience with salivary cortisol and cognitive ability in this sample are perhaps best understood within the framework of biological sensitivity to context (Boyce & Ellis, 2005). In the biological sensitivity theory, early experience is understood to shape stress response systems to meet expected environments with
consequences for behaviors important for regulating behavior in that environment (Blair, 2010). The model is supported by neurobiological research indicating the role of early experience in the development of stress response systems and the ways in which stress hormones potentiate aspects of cognition and behavior important for adaptive functioning. In the model, both highly supportive and highly unsupportive environments lead to elevated stress physiology (Ellis, Essex, & Boyce, 2005). In unsupportive environments, however, this increase is not well regulated and stress hormones remain elevated, facilitating reactive and inflexible rather than reflective and flexible forms of behavior and cognition. In supportive and structured environments, however, regulation of stress hormones is accomplished and facilitates reflective and flexible forms of behavior and cognition, such as executive functions.

*Executive functions and IQ in early childhood.* Given the link between stress hormones and synaptic activity in PFC, it was expected that cortisol would be substantially related to executive functions in this sample. In addition, given the strong relation between the executive function of working memory and measures of intelligence in latent variable analyses (Friedman et al., 2006), it was expected that cortisol would also be related to IQ. The substantial relation of cortisol with executive functions but marginal relation with IQ, however, is consistent with the research described above linking stress hormones with PFC and linking PFC with executive functions. Furthermore, as with research on adult samples (see Kane et al., 2005), this study noted a very strong correlation between executive function and IQ latent variables. Given that the two aspects of cognition were measured at the same time point, no directional relationship could be established. The close association between the constructs, however, is consistent with theory and research indicating that executive functions are important building blocks for the development of children’s thinking (Zelazo, Muller, Frye, & Marcovitch, 2003) and key
contributors to the development of intelligence (Piaget, 1952). A number of prior studies indicate that executive functions are central to the development of fluid intelligence in children (Fry & Hale, 1996; Kail, 2007) and adults (Engle, Tuholski, Lauglin, & Conway, 1999). Findings here are in agreement with these prior studies and indicate the influence of early experience on executive functions as one pathway through which intelligence develops.

Although the close relation between executive functions and intelligence is of interest in this predominantly low-income sample, it is perhaps more relevant to consider the findings in light of studies indicating executive functions to be unique influences on the development of self-regulation and early academic achievement (Riggs, Jahromi, Razza, Dilworth-Bart, & Mueller, 2007). Studies have shown that executive functions longitudinally predict math and reading achievement (McClelland Cameron, Connor, Farris, Jewkes, & Morrison, 2007; Welsh, Nix, Blair, & Bierman, 2009) and the development of theory of mind (Carlson, Mandell, & Williams, 2004; Hughes & Ensor, 2007) above and beyond measures of IQ. Consistent with findings reported here, a number of studies have also demonstrated that executive functions are amenable to the effects of experience. Benefits to executive functions have been shown in randomized controlled evaluations of innovative preschool school readiness programs focusing on the development of self-regulation (Bierman, Nix, Greenberg, Domitrovich, & Blair, 2008; Diamond, Barnett, Thomas, & Munro, 2007; Raver, Jones, Li-Grining, Zhai, Bub, & Pressler, in press).

*Limitations and directions for future research.* Questions concerning specific pathways through which early experience may influence the development of stress physiology important for cognitive development and self-regulation highlight key directions for future research as well as limitations of the current study. Although the data examined in this study are longitudinal, the
findings are correlational and can only indirectly address mechanisms through which the conditions of poverty and activity in stress response systems are causally related to cognitive ability in children. The primary inference in the study lies in the consistency of results with prior studies examining the neurobiology of relations among early experience, the stress response, and cognitive ability. Results are consistent with these prior studies and suggest that one way in which the environment of poverty affects children’s development is through increased stress, leading to elevated set points in stress response systems. In this analysis, however, only one aspect of stress physiology was measured, salivary cortisol as an indicator of the activity of the HPA axis. Cortisol expressed in saliva is the end product of cascade of physiological processes that can be influenced at multiple points by a number of aspects of the organism (Hellhammer, Wust, & Kudielka, 2009). Future studies that collect data on multiple indicators of stress physiology including measures of sympathetic and parasympathetic systems are needed to further establish the relations among early experience and stress physiology as an influence on the development of cognitive ability in children.

Furthermore, it is important to emphasize that the focus of this study was on the typical level for cortisol measured over the child’s first two years. Although longitudinal measurement is a strength of the study and the multiple time points bolster inference in the relation between early experience and stress physiology, additional data on cortisol are desirable. For one, saliva samples were collected in the home by data collectors and there may have been some effect of the presence of the data collector on children. It would be desirable to have typical day saliva samples collected by parents to assess potential effects of the data collection process on child cortisol levels. Given the standardized nature of the data collection protocol, however, and the collection of saliva near the end of the visit after the data collectors had been in the home for
more than an hour, it is likely that these effects were present but minimal. The analysis of Fernald and Gunnar (2009) of the effect of a conditional cash transfer poverty alleviation program on child cortisol levels suggests that our saliva collection protocol did not unduly influence observed cortisol levels in children. In that study, there was some effect of the data collector’s presence in the home on child cortisol levels; however, the effect was similar across children, dissipated within an hour, and as in the current study, cortisol levels were lower for children in more supportive homes, i.e., in homes receiving the conditional cash transfer in Fernald and Gunnar (2009) and in homes characterized by higher levels of positive parenting in our study.

In addition, it is important to further note that cortisol levels follow a well defined diurnal pattern in which levels are high shortly after waking in the morning and decline throughout the day. This variation requires that cortisol levels in studies such as this one be adjusted for time of day of saliva collection in order to remove this source of variability from the data. Future studies that collect multiple saliva samples throughout the day are needed to address questions concerning the ways in which the conditions of poverty are related to diurnal variation in cortisol in early childhood. Prior studies have shown that extreme early disadvantage is associated with a disrupted diurnal pattern in which cortisol levels are low in the morning and demonstrate little change throughout the day, resulting in elevated evening levels (Tarullo & Gunnar, 2006). Given that the sample participating in this study is predominantly low-income but not facing extreme disadvantage as seen in studies with samples of children in foster care or institutional rearing, it is likely that the diurnal pattern is typical but increased for low relative to higher income participants. Higher levels of morning cortisol have been observed in low SES samples (Evans, 2003; Lupien et al., 2001), suggesting that poverty leads to an increased but typical diurnal
pattern. There may be, however, children with atypical diurnal patterns in this sample that we are unable to identify.

Further points related to cortisol concern the dynamic nature of the HPA axis and our focus on a typical level for cortisol over the child’s first two years rather than cortisol reactivity. By combining a methodological approach in which we sampled saliva after being in the home for one hour and a statistical approach using structural equation modeling, we were able to identify what can be considered a general or typical level of cortisol in children over the first two years. This approach, however, in no way obviates that fact that the HPA axis is a very dynamic and reactive system and that measures of HPA reactivity provide very meaningful information about effects of stress on development. In this analysis, we focused on the typical cortisol level rather than reactivity in order to address hypotheses concerning allostatic load in early childhood and also for purposes of modeling the cortisol data as a single latent variable. The general model linking stress physiology with executive functions, however, is that of an inverted U relation in which moderate elevations are associated with better performance and very high or low levels are associated with poor performance. As with research examining stress and cortisol generally, our data indicate that elevations in stress physiology associated with poverty impact aspects of cognition important for the self-regulation of behavior. Presumably this is through problems modulating the reactivity and regulation of stress response systems, including the HPA axis. In follow-up analyses, it will be important to examine the relations of cortisol reactivity and regulation at individual time points to the measures of executive function and IQ. Prior studies with preschool children (Blair et al., 2005) and infants (Haley et al., 2006) have shown that lower baseline cortisol is associated with greater cortisol reactivity and with higher level of cognitive ability. Furthermore, by focusing on the typical cortisol level or set point over the child’s first
Cortisol and Cognition

two years, we did not address questions concerning the relative influence of cortisol and parenting variables at 7, 15, and 24 months on child cognitive ability at age 3 years. The conditions of poverty over the child’s first two years are stable in this sample but the impact of cortisol and/or parenting on cognitive ability may be greater at earlier as opposed to later time points in development.

It is also necessary to emphasize that the sample participating in this study is predominantly low-income. It may be that findings are most specific to low-income samples of this type, at least with respect to the distinct effects of positive and negative parenting and household risk on cortisol and cognitive ability. In more advantaged samples or samples in contexts substantially different from those of families participating in this study, sources of stress and effects of family and household variables on cortisol may vary from those reported here. The model relating early stress to alterations in stress sensitive physiological systems and to self-regulation, however, would seem to be highly generalizable. As well, the central role of early caregiving in this process seems highly generalizable. As such, studies employing randomized designs are needed to further establish associations among variables and to intervene to promote self-regulation and school readiness among children at risk. In this regard, an exemplary study demonstrated the reestablishment of a more typical diurnal cortisol pattern in 3 to 6 year old children receiving a therapeutic intervention in foster care (Fisher, Stoolmiller, Gunnar, & Burraaston, 2007).

The inclusion of measures of stress physiology and executive functions as well as other indicators of self-regulation in randomized intervention studies with low-income samples is an important direction for future research. The longitudinal findings of noted early intervention programs such as Abecedarian (Campbell, Pungello, Miller-Johnson, Burchinal, & Ramey,
2001) and Perry Preschool (Schweinhart et al., 2005) demonstrated that the programs
substantially improved life outcomes of program recipients. The overall benefits of these
programs in promoting advantageous outcomes such as educational attainment and reducing
disadvantageous outcomes such as criminality appear to have resulted to some extent from the
promotion of self-regulation in program recipients (Heckman, 2006, 2007). As such, it may be
that observed beneficial program outcomes occurred in part through effects on neurobiological
systems important for self-regulation including executive functions, as well as aspects of
personality and self-perception associated with self-regulation (Knudsen, Heckman, Cameron, &
Schonkoff, 2006). Currently a number of early parenting (Landry, Smith, Swank, & Guttentag,
2008) and preschool programs (Bierman, Domitrovich, Nix et al., 2008; Bodrova & Leong,
2007; Raver et al., in press; Raver, Jones, Li-Grining, Zhai, Metzger, & Solomon, 2009) have
demonstrated impressive benefits to child cognitive, social-emotional, and self-regulation
abilities using randomized designs. The inclusion of measures of stress physiology and multiple
aspects of self-regulation in future evaluations of similar early care and education programs can
help to further conclusively establish the point that such programs are highly effective at
promoting optimal outcomes for children at risk and represent an efficient and cost-effective
social policy response to persistent and pervasive threats to healthy child development associated
with the conditions of poverty.
References


Cortisol and Cognition

in the rat through variations in maternal care. *Neuroscience and Biobehavioral Reviews*, 29, 843-865


Table 1.

Descriptive statistics for variables in the analysis

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cortisol 7mos (log µg/dl)</td>
<td>1106</td>
<td>-1.88</td>
<td>.69</td>
<td>-3.9 - .22</td>
</tr>
<tr>
<td>Cortisol 15mos (log µg/dl)</td>
<td>991</td>
<td>-1.99</td>
<td>.76</td>
<td>-4.3 - .49</td>
</tr>
<tr>
<td>Cortisol 24mos (log µg/dl)</td>
<td>939</td>
<td>-2.08</td>
<td>.73</td>
<td>-4.4 - .25</td>
</tr>
<tr>
<td>Positive parenting 7mos</td>
<td>1141</td>
<td>2.90</td>
<td>.79</td>
<td>1.0 – 4.8</td>
</tr>
<tr>
<td>Positive parenting 15mos</td>
<td>1100</td>
<td>2.79</td>
<td>.80</td>
<td>1.0 – 5.0</td>
</tr>
<tr>
<td>Positive parenting 24mos</td>
<td>1055</td>
<td>2.89</td>
<td>.81</td>
<td>1.0 – 4.8</td>
</tr>
<tr>
<td>Negative parenting 7mos</td>
<td>1141</td>
<td>2.41</td>
<td>.77</td>
<td>1.0 – 5.0</td>
</tr>
<tr>
<td>Negative parenting 15mos</td>
<td>1100</td>
<td>2.27</td>
<td>.69</td>
<td>1.0 – 5.0</td>
</tr>
<tr>
<td>Negative parenting 24mos</td>
<td>1055</td>
<td>2.43</td>
<td>.87</td>
<td>1.0 – 5.0</td>
</tr>
<tr>
<td>Executive functions 36mos</td>
<td>950</td>
<td>.49</td>
<td>.21</td>
<td>.00 – 1.0</td>
</tr>
<tr>
<td>IQ 36mos</td>
<td>1046</td>
<td>93.64</td>
<td>16.5</td>
<td>45 – 142</td>
</tr>
<tr>
<td>Income-to-need</td>
<td>1236</td>
<td>1.76</td>
<td>1.5</td>
<td>.00 – 16.5</td>
</tr>
<tr>
<td>Maternal education</td>
<td>1123</td>
<td>12.97</td>
<td>2.00</td>
<td>7 – 20</td>
</tr>
<tr>
<td>Household density 7mos</td>
<td>1152</td>
<td>1.55</td>
<td>.62</td>
<td>.67 – 5.0</td>
</tr>
<tr>
<td>Household density 24mos</td>
<td>1098</td>
<td>1.51</td>
<td>.58</td>
<td>.50 – 5.5</td>
</tr>
<tr>
<td>Household safety 7mos</td>
<td>1177</td>
<td>3.00</td>
<td>.58</td>
<td>1.0 – 4.0</td>
</tr>
<tr>
<td>Household safety 24mos</td>
<td>1105</td>
<td>3.00</td>
<td>.49</td>
<td>1.0 – 4.0</td>
</tr>
</tbody>
</table>
Table 2. Correlation among observed variables

|         | C7  | C15 | C24 | P7  | P15 | P24 | N7  | N15 | N24 | EF  | IQ  | INR | Ed  | D7  | D24 | S7  | S24 | AA  |
|---------|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| Cort 7  | 1.00|     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| Cort 15 | .03 | 1.00|     |     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| Cort 24 | .09 | .11 | 1.00|     |     |     |     |     |     |     |     |     |     |     |     |     |     |
| Positive7| -.11| -.07| -.08| 1.00|     |     |     |     |     |     |     |     |     |     |     |     |     |
| Positive15| -.10| -.10| -.03| .65| 1.00|     |     |     |     |     |     |     |     |     |     |     |     |
| Positive24| -.10| -.13| -.09| .56| .63| 1.00|     |     |     |     |     |     |     |     |     |     |     |
| Negative7| .07 | .03 | .09 | -.19| -.19| -.23| 1.00|     |     |     |     |     |     |     |     |     |     |
| Negative15| .07 | .02 | .01 | -.22| -.31| -.31| .41| 1.00|     |     |     |     |     |     |     |     |     |
| Negative24| .06 | .09 | .09 | -.27| -.36| -.53| .35| .41| 1.00|     |     |     |     |     |     |     |     |
| EF      | -.09| -.11| -.12| .29 | .30 | .33 | -.21| -.21| -.25| -.25| 1.00|     |     |     |     |     |     |
| IQ      | -.07| -.09| -.08| .32 | .36 | .43 | -.21| -.25| -.36| .46 | 1.00|     |     |     |     |     |     |
| Income  | -.05| -.03| -.04| .35 | .43 | .42 | -.28| -.29| -.31| .28 | .38 | 1.00|     |     |     |     |     |
| Mat ed  | -.03| -.10| -.04| .40 | .44 | .46 | -.24| -.29| -.32| .27 | .37 | .58| 1.00|     |     |     |     |
| Dense7  | .03 | .11 | .03 | -.21| -.24| -.27| .15 | .18 | .19 | -.22| -.28| -.38| -.33| 1.00|     |     |     |
| Dense24 | .01 | .03 | .01 | -.19| -.25| -.22| .16 | .20 | .16 | -.18| -.24| -.36| -.33| .51 | 1.00|     |     |
| Safety7 | -.03| -.08| -.05| .21 | .25 | .28 | -.16| -.12| -.17| .11 | .23 | .37 | .34 | -.21| -.16| 1.00|     |     |
| Safety24| -.02| -.08| -.06| .16 | .22 | .22 | -.09| -.13| -.20| .11 | .20 | .35 | .29 | -.15| -.16| .42 | 1.00|     |
| AA vs W | .16 | .10 | .13 | -.36| -.34| -.37| .35 | .27 | .34 | -.35| -.37| -.38| -.24| .28 | .25 | -.20| -.12| 1.00|

Correlations larger than .10 are significant at $p < .01$ level
Table 3.

*Correlation among latent and observed variables*

<table>
<thead>
<tr>
<th></th>
<th>CORT</th>
<th>POS</th>
<th>NEG</th>
<th>RISK</th>
<th>EF</th>
<th>IQ</th>
<th>Income</th>
<th>Mat ed</th>
<th>AA</th>
</tr>
</thead>
<tbody>
<tr>
<td>CORT</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>POS</td>
<td>-.42</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NEG</td>
<td>.37</td>
<td>-.60</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RISK</td>
<td>.26</td>
<td>-.54</td>
<td>.48</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EF</td>
<td>-.56</td>
<td>.59</td>
<td>-.59</td>
<td>-.44</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IQ</td>
<td>-.37</td>
<td>.61</td>
<td>-.61</td>
<td>-.53</td>
<td>.92</td>
<td>.42</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Income</td>
<td>-.15</td>
<td>.53</td>
<td>-.50</td>
<td>-.64</td>
<td>.42</td>
<td>.46</td>
<td>.57</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Mat ed</td>
<td>-.18</td>
<td>.57</td>
<td>-.48</td>
<td>-.57</td>
<td>.41</td>
<td>.46</td>
<td>.57</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>AA</td>
<td>.48</td>
<td>-.46</td>
<td>.55</td>
<td>.39</td>
<td>-.52</td>
<td>-.45</td>
<td>-.38</td>
<td>-.24</td>
<td>1.00</td>
</tr>
</tbody>
</table>

*all correlations significant at* *p < .05 level or greater*
Table 4.

Loadings of observed variables on latent indicators

<table>
<thead>
<tr>
<th>Latent Indicator</th>
<th>Observed Variable</th>
<th>Unstandardized Coefficient</th>
<th>Standardized Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>Executive Functions</td>
<td>Attention Shifting</td>
<td>1.00</td>
<td>.68</td>
</tr>
<tr>
<td></td>
<td>Working Memory</td>
<td>.84</td>
<td>.55</td>
</tr>
<tr>
<td></td>
<td>Inhibitory Control</td>
<td>1.17</td>
<td>.46</td>
</tr>
<tr>
<td>IQ</td>
<td>Vocabulary</td>
<td>1.00</td>
<td>.74</td>
</tr>
<tr>
<td></td>
<td>Block Design</td>
<td>.68</td>
<td>.61</td>
</tr>
<tr>
<td>Cortisol</td>
<td>7 months</td>
<td>1.00</td>
<td>.30</td>
</tr>
<tr>
<td></td>
<td>15 months</td>
<td>.97</td>
<td>.26</td>
</tr>
<tr>
<td></td>
<td>24 months</td>
<td>.93</td>
<td>.27</td>
</tr>
<tr>
<td>Positive Parenting</td>
<td>7 months</td>
<td>1.00</td>
<td>.68</td>
</tr>
<tr>
<td></td>
<td>15 months</td>
<td>1.11</td>
<td>.76</td>
</tr>
<tr>
<td></td>
<td>24 months</td>
<td>1.22</td>
<td>.82</td>
</tr>
<tr>
<td>Negative Parenting</td>
<td>7 months</td>
<td>1.00</td>
<td>.53</td>
</tr>
<tr>
<td></td>
<td>15 months</td>
<td>.89</td>
<td>.53</td>
</tr>
<tr>
<td></td>
<td>24 months</td>
<td>1.43</td>
<td>.67</td>
</tr>
<tr>
<td>Household Risk$^a$</td>
<td>7 months$^b$</td>
<td>1.00</td>
<td>.62</td>
</tr>
<tr>
<td></td>
<td>7 months$^c$</td>
<td>-.83</td>
<td>-.55</td>
</tr>
<tr>
<td></td>
<td>24 months$^b$</td>
<td>.89</td>
<td>.59</td>
</tr>
<tr>
<td></td>
<td>24 months$^c$</td>
<td>-.61</td>
<td>-.48</td>
</tr>
</tbody>
</table>

all coefficients significant at $p < .0001$

$^a$household risk included measures of $^b$density and ratings of $^c$home safety at 7 and 24 months
Table 5. 
*Standardized indirect effects*

<table>
<thead>
<tr>
<th>Path</th>
<th>EF</th>
<th>IQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive parenting → Cortisol</td>
<td>.13</td>
<td>ns</td>
</tr>
<tr>
<td>African American → Cortisol</td>
<td>-.19</td>
<td>ns</td>
</tr>
<tr>
<td>African American → Negative Parenting</td>
<td>-.10</td>
<td>-.10</td>
</tr>
<tr>
<td>African American → Household Risk</td>
<td>ns</td>
<td>-.04</td>
</tr>
<tr>
<td>African American → Positive Parenting</td>
<td>-.05</td>
<td>-.07</td>
</tr>
<tr>
<td>African American → Positive Parenting → Cortisol</td>
<td>-.04</td>
<td>ns</td>
</tr>
<tr>
<td>Income-to-Need → Negative Parenting</td>
<td>.04</td>
<td>.04</td>
</tr>
<tr>
<td>Income-to-Need → Household Risk</td>
<td>ns</td>
<td>.10</td>
</tr>
<tr>
<td>Income-to-Need → Positive Parenting</td>
<td>ns</td>
<td>.03</td>
</tr>
<tr>
<td>Income-to-Need → Positive Parenting → Cortisol</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>Maternal Education → Negative Parenting</td>
<td>.07</td>
<td>.07</td>
</tr>
<tr>
<td>Maternal Education → Household Risk</td>
<td>ns</td>
<td>.08</td>
</tr>
<tr>
<td>Maternal Education → Positive Parenting</td>
<td>.07</td>
<td>.06</td>
</tr>
<tr>
<td>Maternal Education → Positive Parenting → Cortisol</td>
<td>.05</td>
<td>ns</td>
</tr>
<tr>
<td>Household Risk → Positive Parenting</td>
<td>ns</td>
<td>-.04</td>
</tr>
<tr>
<td>Household Risk → Positive parenting → Cortisol</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

All coefficients significant at p < .05 level or greater
Figure Captions

1. Hypothesized model relating poverty indicators, household risk, positive and negative parenting, and cortisol to cognitive outcomes at age 3 years.

2. Observed model relating poverty indicators, household risk, positive and negative parenting, and cortisol to cognitive outcomes at age 3 years. All paths presented as standardized effects.

All paths $p < .05$ except † $p < .10$
Cortisol and Cognition

African American

- .23

.57

- .38

Maternal Education

Income-to-Need

POS
$R^2 = .47$

.46

- .15

- .27

.34

.17

RISK
$R^2 = .51$

- .30

- .12†

.26

- .25

NEG
$R^2 = .44$

- .15

- .41

.39

CORT
$R^2 = .40$

- .32

.19

- .27

.26

EF
$R^2 = .61$

- .42

.93

IQ
$R^2 = .55$

- .27