Published in final edited form as:

Dev Psychol. 2016 July ; 52(7): 1073–1087. doi:10.1037/dev0000123.

Psychobiological Influences on Maternal Sensitivity in the Context of Adversity

Eric D. Finegood[1], **Clancy Blair**[1], **Douglas A. Granger**[2,3], **Leah C. Hibel**[4], **Roger Mills-Koonce**[5], and **The Family Life Project Key Investigators**[6,7]

[1]Department of Applied Psychology, New York University

[2]Institute for Interdisciplinary Salivary Bioscience Research, Arizona State University

[3]Johns Hopkins University School of Nursing, Johns Hopkins University Bloomberg School of Public Health, and Johns Hopkins University School of Medicine

[4]Department of Human Ecology, University of California, Davis

[5]Department of Human Development and Family Studies, University of North Carolina Greensboro

[6]Pennsylvania State University

[7]University of North Carolina at Chapel Hill

Abstract

This study evaluated prospective longitudinal relations among an index of poverty-related cumulative risk, maternal salivary cortisol, child negative affect, and maternal sensitivity across the first two postpartum years. Participants included 1,180 biological mothers residing in rural and predominantly low-income communities in the US. Multilevel growth curve analyses indicated that an index of cumulative risk was positively associated with maternal cortisol across the postpartum (study visits occurring at approximately 7, 15, and 24 months postpartum) over and above effects for African American ethnicity, time of day of saliva collection, age, parity status, having given birth to another child, contraceptive use, tobacco smoking, body mass index, and breastfeeding. Consistent with a psychobiological theory of mothering, maternal salivary cortisol was negatively associated with maternal sensitivity observed during parent-child interactions across the first two postpartum years over and above effects for poverty-related cumulative risk, child negative affect, as well as a large number of covariates associated with cortisol and maternal sensitivity. Child negative affect expressed during parent-child interactions was negatively associated with observed maternal sensitivity at late (24 months) but not early time points of observation (7 months) and cumulative risk was negatively associated with maternal sensitivity across the postpartum and this effect strengthened over time. Results advance our understanding of

Address correspondence to: Eric D. Finegood, edf237@nyu.edu, New York University, 627 Broadway, Floor 8, New York, NY, 10012. Douglas A. Granger is now at the Institute for Interdisciplinary Salivary Bioscience Research, University of California, Irvine.

In the interest of full disclosure, Douglas A. Granger is Founder and Chief Scientific and Strategy Advisor at Salimetrics and SalivaBio and these relationships are managed by the policies of the committee's on conflict of interest at Johns Hopkins University School of Medicine and the Office of Research Adherence and Integrity at Arizona State University. Douglas A. Granger is in the process of transferring to the University of California, Irvine where similar oversight arrangements will be established. No other author has conflicts to disclose.

the dynamic, transactional, and psychobiological influences on parental caregiving behaviors across the first two postpartum years.

Keywords

cortisol; parenting; HPA axis; cumulative risk; poverty

Parenting behaviors are critical to child development and have been conceptualized as being primary mechanisms, or conduits through which information about the environment is communicated pre- and postnatally to offspring—effectively "preparing" offspring for what the future may hold (Cameron et al., 2005). Although there is a multiplicity of influences on parenting behavior, relatively few studies have assessed these influences to examine their contributions. Several variables including parents' own material and psychological resources, social and contextual factors, as well as child characteristics all contribute to variation in parent-child interactions (Belsky & Jaffee, 2006). In this analysis, we examine these diverse factors in the investigation of a psychobiological model of parenting behavior; one that is in line with a growing literature (Barrett & Fleming, 2011) that also considers maternal stress physiology to have a unique and potentially mediating influence on individual variation in parenting behaviors in context.

Sensitive caregiving is characterized by a mother's ability to effectively attend, interpret, and respond to her child's cues and signals (Ainsworth, Blehar, Waters, & Wall, 1978). Thus, there are several aspects of the mother including her attention deployment, emotion regulation, and behavior that contribute to her ability to act contingently and in a responsive manner with her child. These factors, which make up a caregiving system, do not develop devoid of context. There are proximal factors including characteristics of the child as well as distal or societal factors including the larger socioeconomic context that contribute to the development and expression of sensitive maternal behaviors across individuals. With regard to mechanism, the cognitive and emotion-regulatory nature of the caregiving system lends support for the idea that this system may be subserved, in part, by a mother's own neurophysiology—itself "tuned" to the demands of a given context. Thus, the goal of the current paper is to characterize several of these distal and proximal influences on maternal sensitivity, and as well, to characterize the potentially mediating influence of the maternal stress physiological system in the relation between contexts of disadvantage and maternal sensitivity.

Child characteristics and maternal sensitivity

Parent-child interactions are inherently dyadic and transactional. For this reason, researchers have highlighted the necessity of considering child factors including temperament and affect when exploring how and why parent-child interactions unfold as they do (Belsky & Jaffee, 2006; Putnam, Sanson, & Rothbart, 2002; van den Boom, 1997). Indeed, high negative affect expressed by infants during dyadic interactions has been shown to be associated with decreased maternal sensitivity (Mills-Koonce et al., 2007) and longitudinal research has

demonstrated that the relationship between child temperament and maternal sensitivity may vary in strength as the child ages. As an example of this, research has found that difficult child temperament is inversely associated with maternal sensitivity in toddlerhood (two years of child age) but not in infancy (six and thirteen months of age) (Bates, Olson, Pettit, & Bayles, 1982; Lee & Bates, 1985; Pettit & Bates, 1984; Putnam et al., 2002). High negative affect expressed by children may compromise a mother's ability to act sensitively during interactions by placing extraneous load and taxing her resources, often at the expense of optimal expressions of sensitive caregiving. In addition to the proximal effects of child characteristics, distal factors including the larger socioeconomic context of families are associated with parent-child interactions that have implications for optimal child development.

The context of poverty

In contrast to the emotional (Walker & Best, 1991) and physiological stress (Hibel, Mercado, & Trumbell, 2012) experienced by middle-income mothers, mothers raising children below the poverty line do so with fewer support systems and financial resources. Often, their home environments are highly chaotic and located in neighborhoods with scarce resources and high crime rates. The psychological consequences of such conditions of hardship on mothers can be significant, and in many cases, can have negative effects on the quality of their interactions with their children (McLeod & Shanahan, 1993; McLoyd, 1998). The circumstances of deep and chronic poverty have the potential to limit a mother's capacity for sensitive caregiving by placing large constraints on her own psychological wellbeing.

In addition to psychological threats to wellbeing, poverty is physiologically taxing. Poverty is associated with alterations in the functioning of the hypothalamic pituitary adrenal (HPA) axis, measured most often by its chemical output cortisol, a glucocorticoid hormone. In adults, socioeconomic status (SES) has been shown to be inversely related to total diurnal cortisol concentrations as measured repeatedly throughout the day (Cohen, Doyle, & Baum, 2006) and also when measured from 45 minutes to three hours after waking (Li, Power, Kelly, Kirschbaum, & Hertzman, 2007). Low SES has been shown to be associated with higher evening levels of cortisol (Cohen et al., 2006b), and this effect has also been shown in a study of mothers of infants (Clearfield, Carter-Rodriguez, Merali, & Shober, 2014). Thus, in a number of studies, low SES has been associated with HPA axis activity at varying times throughout the day, although evidence is mixed, with several studies reporting no relation between SES and HPA axis activity (for a review see Dowd, Simanek, & Aiello, 2009).

Stress physiology and maternal sensitivity

Several studies have explored relations between the maternal HPA axis and parenting behaviors (Fleming, Steiner, & Corter, 1997; Giardino, Gonzalez, Steiner, & Fleming, 2008; Hibel et al., 2012; Kiel & Buss, 2013; Krpan, Coombs, Zinga, Steiner, & Fleming, 2005; Martorell & Bugental, 2006; Mills-Koonce et al., 2009; Seltzer et al., 2009; Sethre-Hofstad, Stansbury, & Rice, 2002; Sturge-Apple, Davies, Cicchetti, & Cummings, 2009; Thompson & Trevathan, 2008). Interestingly, research suggests that the functioning of the maternal HPA axis is associated with various aspects of maternal caregiving, although the direction of

these effects seems to vary as a function of the postpartum time period at which they are measured. Specifically, research has shown that in the first postpartum days, maternal salivary cortisol is positively correlated with first-time mothers' approach responses toward their newborn, which includes with their affectionate touch, their attraction to their own infant's odors, and their sympathy towards baby cries (Fleming, Steiner, & Anderson, 1987; Fleming et al., 1997; Stallings, Fleming, Corter, Worthman, & Steiner, 2001). Notably, by three months postpartum, research suggests that heightened maternal cortisol is associated with negative mood and fatigue in mothers (Krpan et al., 2005) and also with less sensitive behaviors (Thompson & Trevathan, 2008). Furthermore, at six months postpartum, heightened basal cortisol is associated with increases in negative intrusive behaviors in mothers interacting with their children (Mills-Koonce et al., 2009). Thus, prior literature provides evidence that over-activation of the maternal HPA axis is associated with decreases in sensitive caregiving behaviors beginning sometime during the middle of the first postpartum year. Less is known, however, about whether this relation extends into the second postpartum year, where we might expect that influences on parent-child interactions would transform as parents and their children grow.

Adversity, stress physiology, and maternal sensitivity

With respect to the link between environmental experiences of stress, stress physiology, and parenting behaviors, research has shown that heightened diurnal cortisol levels measured throughout the day in mothers mediate the association between adverse early life experiences, including inconsistent care and maltreatment, and maternal sensitivity in adulthood (Gonzalez, Jenkins, Steiner, & Fleming, 2012). In addition to diurnal measures, measures of cortisol reactivity to emotional stress have also been associated with parenting behavior. For example, in a middle-income sample of mothers, cortisol reactivity to an interparental interaction task mediated the relation between interparental withdrawal and psychologically controlling parenting/inconsistent discipline, even when controlling for earlier measures of these parenting behaviors (Sturge-Apple et al., 2009). From the perspective of a psychobiological model of mothering, multiple aspects of stress physiological functioning are hypothesized to mediate, in part, relations between experiences of adversity and maternal behaviors. To the best of our knowledge, no prior study with a predominantly low-income sample has examined longitudinal relations among experiences of adversity, maternal cortisol levels, and maternal sensitivity within a mediational framework.

The Present Study

Relatively little is known about longitudinal influences of accumulated poverty-related stressors on HPA axis functioning in low-income mothers across the first two postpartum years. Similarly, relatively little is known about the ways in which accumulated poverty-related stressors, child negative affect, and the maternal HPA axis are associated with maternal sensitivity in low-income mothers across the first two postpartum years.

The first goal of this analysis is to determine whether a cumulative index of poverty-related risks is associated with heightened salivary cortisol in mothers over the child's first two

years, over and above a set of covariates known to be associated with cortisol. Considering the literature suggesting relations between experiences of adversity and alterations in stress physiology, we hypothesize that cumulative poverty-related risk will be associated with increased cortisol in mothers and that this relationship will remain stable across the first two postpartum years.

The second goal of this analysis is to statistically model a confluence of potential correlates of maternal sensitivity in line with theoretical models that highlight the influence of socio-contextual variables, maternal physiological variables, as well as child characteristics. More specifically, we aim to model the associations of cumulative poverty-related risk, maternal cortisol, and child negative affect with maternal sensitivity observed longitudinally across the first two postpartum years. Given past research, we hypothesize that cumulative risk, maternal cortisol, and child negative affect will each be uniquely and inversely associated with maternal sensitivity across the first two postpartum years. That is, we expect that mothers at higher cumulative risk, with higher cortisol levels, and having a child expressing higher amounts of negative affect (that is more irritable or fussy during parent-child interactions) will express lower amounts of sensitivity in observed interactions with their infants and toddlers.

From a developmental perspective, we are also interested in whether relations between the substantive predictor variables—cumulative risk, maternal cortisol levels, and child negative affect— and the dependent variable, maternal sensitivity, vary as a function of postpartum time. That is, allowing the effect of each of the predictors to vary with time in the same statistical model will afford the opportunity to observe the *relative* and potentially *changing* influences of each variable in explaining variance in maternal sensitivity over the child's first two years. Consistent with prior research showing the time-varying effect of child temperament and affect on maternal sensitivity, we hypothesize that child negativity expressed during parent-child interactions will be inversely, and more strongly related to maternal sensitivity during toddlerhood than during infancy. In contrast, we expect the relation between cumulative risk and maternal sensitivity to remain stable across this postpartum period. Although research has shown that the direction of the relation between cortisol and maternal sensitivity varies as a function of postpartum time, particularly within the first postpartum year, less is known about how this relation extends into the second postpartum year. Given our theoretical model in which increased cortisol levels are seen as a marker of risk, we hypothesize that the influence of cortisol on maternal sensitivity will be stable across the two postpartum years.

Method

Participants

The Family Life Project (FLP) was designed to study families in two regions of the United States with high child poverty rates (Dill, 1999)—three counties in eastern North Carolina (NC) and three counties in central Pennsylvania (PA) were selected to represent the Rural South and Appalachia regions, respectively. A total of 1,292 children and their primary caregivers (99.61% biological mothers) who resided in one of the six counties at the time of the child's birth were recruited at the initial study visit when the child was approximately

two months old. Low-income families were oversampled in both states and African American families were oversampled in NC (African American families were not oversampled in PA because the communities there were at least 95% non-African American). A comprehensive description of the sampling plan and recruitment procedures are provided by Vernon-Feagans, Cox, & The Family Life Project Key Investigators (2013).

The data used in this analysis come from 1,180 biological mothers who were seen on at least one occasion (mean number of visits = 2.48, range 1–3) occurring when their child was approximately 7 months of age (range 5.16 – 15.39 months, M = 7.83), 15 months (range 14.01 – 22.36 months, M = 15.74), and 24 months (range 22.59 – 34.79 months, M = 25.07). Mothers were approximately 25.88 years old (range 14.58 – 44.57) at study entry at 2 months postpartum, 40% were African American and 60% resided in NC. At the 7-month visit, the average income-to-needs ratio was 1.91 (range 0 – 16.49), 62% of mothers were married or constantly partnered, and 39% were primiparous.

Procedures

Data collection took place at three home visits when parents were approximately 7, 15, and 24 months postpartum. The visits lasted approximately 2–3 hours in duration, during which time self-report measures, semi-structured parent-child interactions, and saliva samples were collected from mothers and their children. The saliva samples, which were later assayed for salivary cortisol, were collected from mothers via passive drool after research assistants had been in the participant's home for at least 1 hour. Three saliva samples were collected from mothers before and after an emotional challenge task administered directly to the child. The first saliva sample was collected immediately prior to the emotional challenge task administered to the child. The second sample was collected 20 minutes after the completion of the emotional challenge task, or earlier, if the child expressed peak emotional arousal. The third saliva sample was collected 20 minutes after the second sample. The current study analyzed the average of these three saliva samples per mother per study visit. Families were seen at times that were convenient for them and as a result, time of day of saliva collection varied across families (M = 1:45pm, SD = 3 hours, range = 8:12am – 8:48pm, with 68% of participants seen between 10:45am and 4:45pm; see Table 1 for time of day information across the three study visits).

Mothers and their children engaged in a 10-minute semi-structured free-play task at the 7- and 15-month assessments. Mothers were provided with a standardized set of toys and were instructed to play with their child as if they had some free time. At the 24-month assessment, mothers and their children engaged in a puzzle task, which involved presenting the child with a jigsaw puzzle to complete and asking the mother to assist the child in any way that she chose. After a puzzle was completed, another puzzle of increased complexity was presented to the child (up to a total of three standardized puzzles).

Extensive data collection procedures at the 7- and 24-month waves of study made it necessary for families to be seen at two separate home visits at each of these waves. Because of this, not all of the mothers provided saliva and engaged in the parent-child interaction procedure at the same home visit. Mothers did however provide saliva at the same visit in which they engaged in the parent-child interaction task at the 15-month visit.

Measures

Salivary cortisol—After collection at each assessment (7, 15, and 24 months), saliva samples were immediately placed on ice, then stored frozen at −20°C and subsequently stored at −80°C until they 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, Carlsbad, CA). The samples were assayed in duplicate. Inter and intra-assay coefficients of variation were, on average, less than 15 and 10 percent. The cortisol distributions were subjected to natural log transformation to correct for positive skew. Outliers greater than +/− 3 SD after transformation were treated as missing (n = 36, 30, 24 saliva samples at 7, 15, and 24 months, respectively).

Within study visit, maternal cortisol levels collected around the emotional challenge were highly correlated with one another (r's ranged from .77 to .90 at 7 months; r's ranged from . 76 to .83 at 15 months; r's ranged from .76 to .87 at 24 months). Consistent with the fact that the emotional challenge task was administered to the child, and not to the parent, maternal cortisol levels declined across the emotion challenge task at each study visit, on average—at 7 months, cortisol was significantly higher at sample 1 than at sample 2 (paired $t(991)$ = 17.39, $p < .001$) and cortisol was significantly higher at sample 2 than at sample 3 (paired $t(985) = 10.78$, $p < .001$; this pattern of decline in cortisol levels replicated at 15- and 24-month study visits). Given the strong correlations between the three samples within each study visit, we averaged the three cortisol values at each study visit to arrive at a more reliable estimate of a mean cortisol level for each mother at each study visit. Thus, each mother in the analysis dataset had a total of up to three mean-composite cortisol values (if they participated in all three study visits). Visual inspection of histograms and skewness statistics indicated that the natural log transformed mean-composite cortisol data were normally distributed (natural log transformed cortisol values: Skewness = 0.15, SE = 0.04, Kurtosis = 0.25, SE = 0.09, Kolmogorov-Smirnov test $D(2530) = 0.02$, $p = 0.009$; raw cortisol values: Skewness = 3.13, SE = 0.04, Kurtosis = 16.80, SE = 0.09, Kolmogorov-Smirnov test $D(2530) = 0.16$, $p < 0.001$). Time of day of saliva collection was significantly related to this mean cortisol level at each study visit (r = −.48 at 7 months; r = −.45 at 15 months, r = −.53 at 24 months). Thus, consistent with much prior literature showing the diurnal pattern of cortisol, it was important that we control for time of day in all analyses. Inclusion of a quadratic effect for time of day on cortisol levels was non-significant at each study visit indicating a linear relation between time of day of saliva collection and cortisol levels.

Cumulative risk—Based on prior work with these data (Vernon-Feagans et al., 2013), we used a cumulative risk composite of 7 variables—family income-to-needs ratio, maternal education, constant spouse/partner living in the home, hours of employment, occupational prestige, household density, and neighborhood noise/safety—each measured at 7, 15 and 24 months postpartum. A continuous cumulative risk index was generated by reverse-scoring the positively framed indicators, standardizing each risk measure, and averaging the standardized risk variables. Three cumulative risk scores were generated, one for each study wave, with higher scores reflecting higher levels of risk. At 7 months, correlation coefficients among the seven indicators included in the cumulative risk index ranged from r

= −.12 to r = .52, p's < 0.01. At 15 months, correlation coefficients among the seven cumulative risk indicators ranged from r = −.15 to r = .55, p's < 0.01. At 24 months, correlation coefficients among the cumulative risk indicators ranged from r = −.08 to r = .53, p's < 0.01. The reader is referred to Vernon-Feagans et al. (2013), which presents correlations among the seven cumulative risk indicators averaged across time and additionally details the measurement work involved in the generation of this cumulative risk composite.

Maternal sensitivity—Free play and puzzle interactions were video recorded and the following seven aspects of parenting were coded at the three time points: sensitivity, detachment, intrusiveness, stimulation, positive regard, negative regard, and animation in interacting with the child (Cox & Crnic, 2002; NICHD Early Child Care Research Network, 1999). Ratings were made on a scale ranging from 1 ("not at all characteristic") to 5 ("highly characteristic") at the 7- and 15-month assessments and on a scale ranging from 1 ("not at all characteristic") to 7 ("highly characteristic") at the 24-month assessment. To maintain consistency across time, scores at the 24-month visit were rescaled to range from 1 to 5 (score of 7 recoded to 5, scores of 6 & 5 recoded to 4, 4 recoded to 3, 3 & 2 recoded to 2, 1 coded as 1; Mills-Koonce et al., 2011). Principal Component Analysis conducted with oblique rotation (i.e. Promax) at each time point indicated distinct dimensions of parenting behaviors. Maternal sensitivity included five maternal characteristics—sensitivity, detachment (reversed), stimulation of development, positive regard, and animation, which were averaged together to create the maternal sensitivity index used in the current analyses. Coders were trained and certified by a master coder and reliability was determined by calculating the intra-class correlation coefficients (ICC) for ratings made by pairs of trained coders for each scale of the maternal sensitivity dimension ($ICC_{Sensitivity}$ = .75, .84, .85; $ICC_{Detachment}$ = .74, .79, .83; $ICC_{Stimulation}$ = .75, .77, .89; $ICC_{Pos.\ regard}$ = .78, .85, .87; and $ICC_{Animation}$ = .79, .81, .82; at 7, 15, and 24 months, respectively). A minimum of 30% of all observations were double coded; discrepant codes were resolved by conferencing.

Child negative affect—Parent-child interactions at 7, 15, and 24 months were coded for child negative affect, which assessed the extent to which the child cried, fussed, frowned, tensed body, or otherwise expressed his or her discontentment during the interaction. Children received a score between 1 and 5, where 1 represented "being not at all characteristic of the child's behavior during the interaction" and 5 represented "being highly characteristic of the child's behavior during the interaction". Coders were trained and certified by a master coder and subsequently each pair of coders maintained ICC's greater than .80. The same training, certification, and ongoing reliability procedures were used for both parent and child codes.

Control covariates—We included a number of behavioral and demographic covariates in our models predicting cortisol and maternal sensitivity, including an indicator for whether the mother reported breastfeeding (1) or not (0) at 7 months, given that breastfeeding has been shown to be related to decreases in the cortisol response (Tu, Lupien, & Walker, 2006). Research shows that tobacco smoking is associated with higher salivary cortisol levels (Granger et al., 2007), thus we included an indicator for tobacco smoking (1) or not (0) at 7

and 24 month visits. Smoking data were not collected at 15 months. Mothers who reported smoking at both, or at neither 7- nor 24-months were designated as smokers or non-smokers at 15 months, respectively. If mothers reported being a smoker at only one time point and not the other, their 15-month smoking data were treated as missing. Research using these data has shown that oral or transdermal contraceptive use is associated with higher cortisol levels in mothers (Hibel, Granger, Kivlighan, Blair, & The Family Life Project Investigators, 2006), thus we controlled for its use (1) or not (0) at each time point. Other time-varying covariates included body mass index (BMI) collected at 7 and 24 months. BMI was not collected at 15 months and we therefore averaged mothers' BMI at 7 and 24 months to get a 15-month measure. At each time point we included an indicator for whether the mother had given birth to another child during the course of the study (1) or not (0). As mentioned previously, timing of saliva collection at 7, 15, and 24 months (24-hour clock) was also controlled in each model to adjust for diurnal variation in cortisol levels.

Time-invariant covariates included maternal age (at 2-month study entry) and parity status at study entry (primiparous = 1, multiparous = 0). Both the mother's self-reported ethnicity as either Black or White and her state of residence (NC or PA) were used to categorize mothers as members of the following categories: Black mothers from PA, Black mothers from NC, White mothers from PA and White mothers from NC. Because so few mothers who identified as Black resided in Pennsylvania (n = 22), these mothers were excluded from the following analyses. Two ethnicity/region categories were then created using dummy codes (0 or 1) in order to separately estimate the role of ethnicity for mothers living in North Carolina (i.e North Carolina Black coded as 1, all other categories coded as 0) on the dependent variables. In addition, this coding scheme allowed for the control of state of residence for mothers who identified as White (Pennsylvania White coded as 1 versus all other categories coded as 0). Mothers residing in North Carolina who identified as White were therefore the reference group. All continuous variables were mean centered.

Missing Data

The total sample at study entry was 1,292 with 1,204 families seen at 7 months postpartum, 1,169 at 15 months, and 1,144 at 24 months. Mothers missing parent-child interaction data at all time points were excluded from the analysis sample. In addition, given the relationship between cortisol and gestation time (Mastorakos & Ilias, 2003), we excluded mothers from the analysis sample at a given time point if they reported being pregnant ($n = 52$ at the 7-month visit, $n = 92$ at 15 months, and $n = 87$ at 24 months). These considerations yielded an analysis dataset of $n = 1{,}180$ non-pregnant, biological mothers. To assess possible differences between those included in the analysis dataset and those not included, we examined a number of variables for which we had complete information collected at 2 months postpartum including state of residence, ethnicity, child age at 2 months, an income screen, total number of household members, number of children in the household, primary caregiver age, education, marital status, and employment. Examination of characteristics of participants excluded from the sample indicated few differences with those retained. For example, participants excluded from the analysis sample were less educated, on average, $t(1290) = 1.98$, $p < 0.05$, more likely to be African American χ^2 (1, $N = 1292$) = 8.29, $p < 0.01$, and less likely to be married χ^2 (1, $N = 1292$) = 3.97, $p < 0.05$. Participants included in

the analysis sample were missing data on a number of variables, and of them, cortisol had the most missing values. At 7 months, 3% of mothers in the analysis sample were missing cortisol values. At 15 months, 19% of mothers were missing cortisol values. At 24 months, 21% of mothers were missing cortisol values. We used full information maximum likelihood (FIML) estimation to reduce the potential for bias in the analysis related to missing data (Enders, 2010). FIML requires that missing data patterns be at least Missing at Random (MAR), that is, that missingness on an analysis variable is associated with other analysis variables but not to the values of the variable with missingness itself (Enders, 2010). To test this assumption, we conducted independent samples t-tests to assess whether cases with missing cortisol data differed systematically on any analysis variables compared to cases with complete cortisol data. At 7 months, mothers missing cortisol had higher cumulative risk scores $t(1154) = 4.24$, $p < .01$, had their 7-month home visit later in time postpartum $t(1104) = 3.05$, $p < .01$, were younger in age $t(1178) = -2.27$, $p < .05$, had higher BMIs $t(1081) = 3.03$, $p < .01$, and were less likely to be breastfeeding χ^2 (1, $N = 1{,}180$) = 9.31, $p < 0.01$. At 15 months, mothers missing cortisol measures had their 15-month home visit later in the day $t(1110) = 6.29$, $p < .01$ and were seen earlier in the postpartum $t(562.45) = -2.62$, $p < .01$. At 24 months, mothers missing cortisol measures had their 24-month home visit later in the day $t(1049) = 6.95$, $p < .01$ and were younger $t(1178) = -3.08$, $p < .01$ and were less likely to have given birth to another child by 24 months χ^2 (1, $N = 1180$) = 23.34, $p < 0.01$. These differences associated with missing cortisol data necessitated the inclusion of covariates in the model to assist in reducing the potential for nonresponse bias. Because of the associations between analysis variables and the propensity for missingness of cortisol, and additionally, because cortisol values are unlikely to be associated with the propensity for missing cortisol data, the MAR mechanism is a plausible assumption. This makes FIML an appropriate technique for handling missing data in the current analysis. FIML uses all of the information in the independent variables to estimate the covariance matrix, thus allowing us to include all biological, non-pregnant mothers that had maternal sensitivity data, regardless of their amount of missingness on the independent variables.

Data Analysis Plan

Following descriptive analyses (Table 2), we use multilevel modeling using Mplus 7 software (Muthén & Muthén, 1998–2012) including maximum likelihood estimation (MLE) with robust standard errors to predict the intercept and slope of salivary cortisol in mothers from 7 to 24 months postpartum. Model-building strategies included Satorra-Bentler scaled chi-square difference tests (Satorra & Bentler, 2001) to compare the fit of nested models. The data for this analysis were unbalanced and not time-structured (Singer & Willett, 2003) meaning that the number of study visits varied across families in the sample (range = 1–3) and that the timing of each study visit reflected the actual number of months postpartum of each mother. Time was centered at the between-person group mean of the 7-month visit (which was 7.83 months as shown in Table 1) and rescaled into years so that the coefficient for time reflects change in cortisol levels over the course of one year. For example, a mother whose 7-month study visit actually occurred when she was exactly 8 months postpartum would be scored to be 0.17 after group-mean centering (8.00 – 7.83 = 0.17). Furthermore, her score after rescaling from months into years would be 0.01 (0.17 / 12 = 0.01). Model-building strategies included testing an unconditional growth model including fixed linear

effects for time. In a next step, we added terms for cumulative risk and the covariates to the model (Table 3).

To address our second research question regarding the influences of cortisol, cumulative risk, and child negative affect on maternal sensitivity, we conducted a second series of multilevel models including MLE with robust standard errors to predict the intercept and slope of maternal sensitivity from 7 to 24 months postpartum. As in the first analysis, the data for this second analysis were unbalanced and not time-structured. That is, the number of study visits varied across families in the sample (range = 1–3) and each study visit reflected the actual number of months postpartum of each mother. Time was centered at the between-person group mean of the 7-month visit (which was 7.83 months as shown in Table 1) and rescaled into years so that the coefficient for time reflects change in maternal sensitivity over the course of one year. First, we ran an unconditional growth model including fixed and random linear time effects. Second, we included terms for child negative affect along with cumulative risk and the demographic and behavioral covariates (Model 1 in Table 4). Third, we added a residualized cortisol score (the unstandardized residuals of cortisol regressed onto time of day and contraceptive use, in order to partial out the variance associated with time of day of saliva collection and contraceptive use; Model 2 in Table 4). Finally, we included fixed linear trends (predictors of linear slope) for all variables and subsequently removed non-significant predictors of linear slope in the final model (Model 3 in Table 4). To assess our hypotheses regarding the relative changes in the prediction of the intercept of maternal sensitivity at 7 months vs. at 24 months postpartum, this final model was re-run with time re-centered at 24 months. Re-centering time in this way does not affect the statistical significance of any interactions of predictors with linear time (predictors of linear slope) retained in the model. Rather, in the presence of significant interaction terms, re-centering time to be around the 24-month visit allows the potential to see changes in the strength and/or direction of the main effects predicting the intercept at 24 months.

Effect sizes—Effect sizes were computed for statistically significant effects by taking the product of the coefficient of the independent variable and the SD of the independent variable and dividing by the square root of the variance term of the intercept in the unconditional growth model. These effect sizes are standardized estimates and can be interpreted as the shift in the intercept of the outcome in SD units that would be expected from a one SD change in the independent variable, net of the other variables in the model.

Results

Preliminary Analyses

Table 2 displays zero order correlations of the analysis variables. Cumulative risk was moderately associated with parenting sensitivity ($r = -.50$, $p < .01$), age of the mother ($r = -.48$, $p < .001$), breastfeeding at 7 months ($r = -.32$, $p < .01$), and tobacco smoking ($r = .27$, $p < .01$). Cumulative risk was also associated, albeit to a lesser extent, to cortisol ($r = .08$, $p < .01$), child negative affect ($r = .06$, $p < .01$), time of day of saliva collection ($r = -.19$, $p < .01$), BMI ($r = .13$, $p < .01$), having given birth to a new child over the course of the study ($r = .08$, $p < .01$), and using oral or transdermal contraceptives ($r = -.05$, $p < .01$). There was a

small correlation between cortisol levels and maternal sensitivity ($r = -.09$, $p < .05$). Notably, there was a moderate inverse association between cortisol and months postpartum ($r = -.22$, $p < .01$) indicating that that cortisol declined on average across the postpartum. In contrast, there was no correlation between maternal sensitivity and months postpartum ($r = -.01$, p = ns) indicating that maternal sensitivity was stable, on average, across the postpartum.

Unconditional Growth Model of Cortisol

In the unconditional growth model, there was a significant negative slope for cortisol across the two postpartum years ($b = -0.25$, SE = 0.01, $p < .001$), indicating that modeling time in a linear way contributed significantly to the modeling of cortisol compared to an unconditional means model in which just the mean of cortisol is modeled. Random intercepts ($\sigma^2 = 0.085$, SE = 0.012, $p < .001$) were retained and indicated that there was a significant amount of between-person variation in the intercept of cortisol at 7 months postpartum. A Satorra-Bentler scaled chi-square difference test indicated that including a term for a random linear slope, which would allow there to be random variation around the fixed linear slope, did not significantly improve model fit compared to a model with only random intercepts and a fixed linear slope. Thus, random intercepts were modeled in the following analyses where natural log-transformed cortisol is the dependent variable including covariates as well as predictors of interest.

Cumulative Risk, Demographic controls, and Cortisol

Effects on the intercept, shown in Table 3 indicate a significant positive association between cumulative risk and maternal salivary cortisol ($b = 0.06$, SE = 0.03, $p < 0.01$, effect size (ES) = 0.13). Given the diurnal rhythm of cortisol, there was a large effect for time of day of saliva collection on the intercept, indicating that time of day was inversely associated with cortisol, on average ($b = -0.10$, SE = 0.00, $p < .001$, ES = 1.03). African American mothers had higher cortisol than did White mothers ($b = 0.11$, SE = 0.04, $p < .01$, ES = 0.19). Oral or transdermal contraceptive use ($b = .10$, SE = 0.03, $p < .001$, ES = 0.16), smoking ($b = 0.11$, SE = 0.03, $p < .001$, ES = 0.18), and maternal age ($b = 0.01$, SE = 0.00, $p < 0.01$, ES = 0.14) were positively associated with the intercept of cortisol. In addition, first-time mothers had elevated cortisol compared to multiparous mothers ($b = 0.07$, SE = 0.03, $p < 0.01$, ES = 0.11). Having given birth to another child over the 7–24 month period ($b = -0.13$, SE = 0.05, $p < 0.01$, ES = 0.10), BMI ($b = -0.01$, SE = 0.00, $p < 0.001$, ES = 0.18), and breastfeeding at 7 months ($b = -0.11$, SE = 0.04, $p < 0.01$, ES = 0.13) were each negatively associated with the intercept of cortisol. No variables were associated with linear change in cortisol.

Table 3 indicated that variation in cortisol levels between-persons ($\sigma^2_{intercept} = 0.05$) was low relative to the amount of variation in cortisol levels within-persons ($\sigma^2_{residual} = 0.26$). Based on calculation of the intra-class correlation (ICC), between-person variation in cortisol accounted for approximately 16% of the total random variation in cortisol levels across time (ICC = $0.16 = \sigma^2_{intercept} / (\sigma^2_{intercept} + \sigma^2_{residual})$). This indicates that the majority of variation in cortisol levels was observed within-persons across time.

Unconditional Growth Model of Maternal Sensitivity

In the unconditional growth model, we observed no evidence of a fixed slope, suggesting that, on average, there was no change in maternal sensitivity levels across the first two postpartum years. Random intercepts (σ^2 = .34, SE = 0.02) were retained and indicated a significant amount of between-person variation in the intercept of maternal sensitivity at 7 months. A Satorra-Bentler scaled chi-square difference test indicated that although there was no average change in the sample overall across time, there was significant individual random variation in change. Including a term for a random linear slope significantly improved model fit compared to a model including only random intercepts and fixed linear slope. Thus, random linear slopes (σ^2 = 0.036, SE = 0.011, $p < .005$) were also modeled in the following analyses, including covariates and predictors of interest.

Cumulative Risk, Cortisol, Child Negative Affect, and Maternal Sensitivity

Table 4 displays the results from a series of multilevel growth curve analyses that assessed the extent to which cumulative risk, cortisol, and child negative affect were associated with maternal sensitivity across the first two postpartum years. Model 1 of Table 4 included cumulative risk, child negative affect, as well as covariates as predictors of maternal sensitivity. Model 2 of Table 4 included all the predictors of Model 1 with the addition of a cortisol term, which was the unstandardized residual of cortisol levels partialling out the variance associated with linear time-of-day of saliva collection and oral or transdermal contraceptive use. As noted in the Methods section, there was no quadratic effect of time of day on cortisol. Model 3 of Table 4 included all of the terms from previous models plus estimates of the predictors of the slope.

Results presented in Model 1 of Table 4 indicated that cumulative risk was negatively related to the intercept of maternal sensitivity ($b = -0.34$, SE = 0.03, $p < 0.001$, ES = 0.38). In addition, child negative affect during parent-child interactions ($b = -0.07$, SE = 0.01, $p < 0.001$, ES = 0.12), African American ethnicity ($b = -0.31$ SE = 0.05, $p < .001$, ES = 0.26), and BMI ($b = -0.01$, SE = 0.00, $p < 0.05$, ES = 0.06) were negatively related to the intercept of maternal sensitivity. Maternal age ($b = 0.02$, SE = 0.00, $p < 0.001$, ES = 0.17) and breastfeeding ($b = 0.25$, SE = 0.05, $p < 0.001$, ES = 0.15) were positively associated with the intercept of maternal sensitivity.

Results from Model 2 of Table 4 indicated that maternal cortisol levels were inversely associated with the intercept for maternal sensitivity ($b = -0.05$, SE = 0.02, $p < 0.05$, ES = 0.05) net of other variables in the model. Notably, the addition of cortisol to Model 2 did not change the coefficient for cumulative risk from what it was in Model 1, indicating that the association between cortisol and maternal sensitivity is unique and provides no evidence of any mediation of cumulative risk on maternal sensitivity through cortisol.

Results from Model 3 of Table 4 included predictors of the slope and indicated that cumulative risk ($b_{\text{cumulative risk x time}} = -0.09$, SE = 0.03, $p < 0.01$, ES = 0.30) and child negative affect ($b_{\text{negative affect x time}} = -0.07$, SE = 0.02, $p < 0.001$, ES = 0.39), but not maternal cortisol levels, were associated with the slope of maternal sensitivity across time.

The effect for cortisol on the intercept was unchanged with the addition of predictors of slope to the model.

Figure 1 depicts the significant association between cumulative risk and the slope of maternal sensitivity. The coefficient for the main effect of cumulative risk on the intercept for maternal sensitivity ($b = -0.29$, SE = 0.03, $p < .001$, ES = 0.32) shown in Model 3 of Table 4 is interpreted as the association between cumulative risk and maternal sensitivity at the 7-month study visit (time is centered at the 7-month visit). Subsequent re-centering of time to be around the 24-month study visit (rather than the 7 month visit) and re-examination of the main effect for cumulative risk in this new model indicated that cumulative risk was associated with an even greater differential in maternal sensitivity at 24-months ($b = -0.42$, SE = 0.04, $p < .001$; not shown in Table 4). Figure 1 depicts how the association between cumulative risk and maternal sensitivity increases with time.

Figure 2 depicts the significant association between child negative affect and the linear slope of maternal sensitivity. The coefficient for the main effect of child negative affect on the intercept of parenting sensitivity ($b = -0.02$, SE = 0.02, *ns*) shown in Model 3 of Table 4 is interpreted as the non-significant association between child negative affect and maternal sensitivity at the 7-month study visit. Re-centering the intercept to represent the last time point, 24-months, indicated that child negative affect was negatively associated with maternal sensitivity ($b = -0.13$, SE = 0.02, $p < .001$; not shown in Table 4). Figure 2 depicts this increasing association between child negative affect and maternal sensitivity across time. Table 4 indicated that variation in maternal sensitivity between-persons ($\sigma^2_{\text{intercept}} = 0.20$) represented approximately 45% of the total random variation in maternal sensitivity across time (ICC = 0.45 = $\sigma^2_{\text{intercept}}$ / ($\sigma^2_{\text{intercept}} + \sigma^2_{\text{linear slope}} + \sigma^2_{\text{residual}}$).

Robustness checks

Given that time of day of saliva collection varied between and within participants in the sample, we reran our models with subgroups of the full sample in which saliva was collected either in the morning or in the afternoon/evening. We also examined the portion of the sample in which saliva was collected within one standard deviation of the mean collection time (between the hours of 10:00 and 16:00). In the morning subsample ($n = 717$; 1,005 cortisol samples; 1,005/717 = 1.40 samples per person, on average) the association between cortisol and maternal sensitivity was larger than in the sample as a whole ($b = -0.13$, SE = 0.06, $p = .02$) as was the association between cumulative risk and maternal cortisol ($b = 0.08$, SE = 0.04, $p = .06$). In the afternoon/evening subsample ($n = 993$; 1,523 cortisol samples; 1,523/993 cases = 1.53 samples per person, on average), the association between cortisol and maternal sensitivity was reduced although the direction of the coefficient is the same as in the full sample ($b = -0.04$, SE = 0.04, $p = .38$). The association between cumulative risk and cortisol was also reduced relative to the coefficient for the full sample ($b = 0.05$, SE = 0.03, $p = .11$). In the 10:00 to 16:00 hour subsample ($n = 975$; 1,725 cortisol samples; 1,725/975 = 1.77 samples per person, on average), the association between cortisol and maternal sensitivity was again larger than in the sample as a whole ($b = -.12$, SE = 0.04, $p = .005$). The association between cumulative risk and maternal cortisol in this subsample was slightly larger than in the sample as a whole ($b = 0.07$, SE = 0.03, $p = .02$).

Discussion

To our knowledge, this is the first study to explore longitudinal relations among a cumulative index of poverty-related risk, maternal HPA axis activity, child negative affect, and maternal sensitivity from 7 to 24 months postpartum in a large sample of mothers from primarily low-income and rural communities in the United States. In general, the findings align with a psychobiological model of mothering in which the effects of accumulated adversity are seen both physiologically as well as behaviorally.

Consistent with our hypothesis, our analyses indicated that poverty-related cumulative risk was associated with elevated salivary cortisol in mothers, over and above several demographic and behavioral variables with which cortisol and cumulative risk are both associated. The size of the effect of cumulative risk on maternal cortisol was small but consistent with prior studies that have found direct inverse associations between SES and HPA axis activity in adults (Clearfield et al., 2014; Cohen et al., 2006a; Cohen et al., 2006b; Li et al., 2007). It is important to acknowledge, however, that while this finding is consistent with some prior studies, many studies have found no direct associations between SES and HPA axis activity and others have found positive relationships between SES and HPA axis activity (for a comprehensive review of this topic see Dowd et al., 2009). A number of other variables including tobacco smoking, BMI, breastfeeding, oral or transdermal contraceptive use, parity status, having given birth to a new child during the course of the study, and maternal age were each unique correlates of maternal cortisol levels over and above the effect of cumulative risk. Including these covariates allowed for a somewhat conservative test of the hypothesis that cumulative risk would be associated with HPA axis activity in mothers. It is important to note that many of these covariates are themselves correlated with cumulative risk and to some extent may represent causal processes through which risk is associated with maternal stress physiological functioning.

Additionally, we found that cortisol, child negative affect, and cumulative risk were uniquely negatively correlated with maternal sensitivity, also over and above a number of covariates. That cortisol was uniquely associated with maternal sensitivity is consistent with prior research showing that increases in diurnal cortisol secretion (Gonzalez et al., 2012) basal cortisol levels (Mills-Koonce et al., 2007) and cortisol reactivity (Thompson & Trevathan, 2008) are associated with reductions in maternal sensitivity throughout the first postpartum year. Findings from the present study suggest that this relationship between heightened HPA axis activity and lower maternal sensitivity may extend throughout the second postpartum year as well. Maternal sensitivity concerns the extent to which a mother can effectively interpret and find appropriate meaning in her child's cues and to what extent she is responsive to her child's needs (Ainsworth et al., 1978). Sensitivity relies on the coordinated actions of top-down and bottom-up cognitive and emotion-regulatory networks in the brain that modulate attention and affect by way of hypothalamic-midbrain-limbic and prefrontal cortical circuitry, as well as the influence of several neurohormones, including cortisol, on this circuitry (Comprehensive reviews include Barrett & Fleming, 2011; Swain, Kim, & Ho, 2011; Swain, Lorberbaum, Kose, & Strathearn, 2007). Heightened or otherwise dysregulated patterns of HPA axis activity in the context of high stress have negative effects on the neural networks that underlie self-regulation (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007) and

therefore may impact maternal sensitivity. Indeed, our findings regarding the positive association between poverty-related cumulative risk and maternal cortisol as well as the unique associations among cumulative risk, maternal cortisol and maternal sensitivity may be indicative of difficulty in flexibly regulating emotional, executive, and attentional responses to infant cues during parent-child interactions in the context of adversity. With this in mind, however, we found no evidence that the association between poverty-related cumulative risk and maternal sensitivity was mediated through activity of the maternal HPA axis. At least one prior study has shown such mediation. Specifically, Gonzalez and colleagues (2012) used path analyses to show that maternal diurnal cortisol levels mediated the relation between experiences of early life adversity and maternal sensitivity in adulthood. The present study differs from this previous study in a number of ways, however, which may have contributed to the somewhat divergent findings. Whereas the prior study was conducted at a single time point with a sample of mothers earlier in the postpartum (2–6 months), the present study was conducted in a sample of mothers seen longitudinally and later in the postpartum (throughout the first two postpartum years). In addition, the current study utilized a mean-composite of cortisol samples collected during a home visit, which cannot be parameterized in the same way as diurnal levels collected in the early morning and evening. Lastly, we did not incorporate measures of adverse early life experiences, which may be uniquely associated with adult outcomes even when adversity in adulthood in controlled for (Cohen, Janicki-Deverts, Chen, & Matthews, 2010). The measure of cumulative risk utilized in the current study was with respect to current life circumstances, not to reports of early life adversity.

A general strength of our study is its longitudinal design. Specifically, having repeated measures allowed us to examine changes in relations among variables across the child's first two years of life, an important developmental period during which parents constitute the child's primary social environment and lay the foundations for growth in many domains of development including in cognitive, social, and emotion regulation (Kopp, 1982; Sroufe, 1997; Tronick, 1989). Consistent with our hypothesis, results from this analysis indicated that the strength of the relation between maternal HPA axis activity and maternal sensitivity did not vary across time, suggesting that maternal stress physiological regulation continues to be uniquely associated with maternal sensitivity across the early years of parenthood. In contrast, the associations between child negative affect and maternal sensitivity and also between poverty-related cumulative risk and maternal sensitivity increased in strength over time. In the particular case of child negative affect, we found no association with maternal sensitivity at 7 months. Rather, a significant association was seen at 24 months. Such an increase in the influence of child negative affect on maternal sensitivity is consistent with prior research (Bates et al., 1982; Lee & Bates, 1985; Pettit & Bates, 1984), which has suggested that child negative affect may be more associated with maternal sensitivity during toddlerhood than during early infancy. It makes sense from an interactional perspective on the process of development in general (Sameroff & Fiese, 2000) and on the development of parenting behaviors in particular (Belsky & Jaffee, 2006) that the relation between cumulative risk and maternal sensitivity, and the relation between children's negative affect and maternal sensitivity, would increase across time. The development of parenting behaviors across the postpartum is dynamic and influenced by ongoing bidirectional

interactions between the parent and the environment across time. It may be the case, for instance, that high amounts of stress in the context of high cumulative psychosocial and sociodemographic risk are associated with cognitive and emotion-regulatory difficulties in parents, which may influence their capacity for sensitive caregiving early in the postpartum. As time goes on, these difficulties might further elicit more dysregulation and negative behaviors on the part of children so that by 24 months, children's negative affect and psychosocial/sociodemographic risks compound and explain a wider differential in sensitive maternal behaviors than were observed earlier in time. Child negative affect may also be associated with maternal sensitivity through interactions with aspects of parents themselves, including their perceptions of self-efficacy in the caregiving role, instances of depression, and their perceived social support (see comprehensive review by Belsky & Jaffee, 2006). These factors may be particularly relevant for families living in rural poverty who tend to have smaller social networks and social support systems than middle-income families living in rural settings (Evans, Boxhill, & Pinkava, 2008). Findings from the current study highlight the need for future work to examine interactive effects between accumulated risk and both parent and child characteristics in the prediction of maternal sensitivity in the context of rural and low-income communities.

Study Limitations, Implications, and Conclusions

The primary limitation of our study concerns the fact that time of day of saliva collection varied between and within participants across time. Variation in time of day of saliva collection is potentially a problem for our analysis not only because of the diurnal rhythm of the HPA axis, but also because elevations in morning cortisol may have different biologic and etiologic interpretations than elevations in afternoon/evening cortisol (Miller, Chen, & Zhou, 2007). Statistical control for time of day may be able to address potential problems concerning the diurnal rhythm of the HPA axis, but it cannot, however, address issues relating to the interpretation of elevations in HPA axis activity in the morning versus the evening. For instance, prior studies have shown that certain forms of severe stress (i.e. maltreatment, neglect, institutionalization, or Posttraumatic Stress Disorder) are associated with depressed morning levels and elevated evening levels of cortisol (Gunnar & Quevedo, 2007; Heim, Ehlert, & Hellhammer, 2000), which differs from cortisol patterns often associated with stress exposures considered in the typical range. Although this is a limitation of the current study, concerns are reduced somewhat through our robustness checks restricting samples to specific time windows.

Our robustness checks restricting the sample to specific times of the day indicated that our results are for the most part robust across the day and in fact are larger for the relation between cumulative risk and cortisol and cortisol and maternal sensitivity in the morning and midday relative to the afternoon/evening collection times. We suggest that this might be due to the fact that families in our sample represent risk that is within the typical range rather than risk that is extreme, where we might expect to observe a negative association between risk and cortisol levels in the morning and a positive relation between risk and cortisol levels in the evening. Although the present study utilized a cumulative risk approach, which is somewhat distinct from simpler measures of SES, it is notable that prior research remains somewhat inconsistent with respect to the time of day at which the link

between SES and HPA axis activity (when it is observed) is present. Some studies have shown low SES to be associated with elevated morning cortisol levels (Li et al., 2007), while others have shown low SES to be only associated with elevated evening cortisol levels (Clearfield et al., 2014; Cohen et al., 2006b). Still others have shown associations between SES and cortisol throughout the entire working day (Cohen et al., 2006a; Steptoe et al., 2003). Our findings are generally consistent with a prior meta-analysis showing that psychosocial stress tends to be associated with elevated cortisol across the day (Miller et al., 2007) and with prior research demonstrating relations between parenting behavior and maternal cortisol sampled in the morning (Giardino et al., 2008; Martorell & Bugental, 2006; Thompson & Trevathan, 2008) or late morning/early afternoon (Fleming, Ruble, Krieger, & Wong, 1997; Krpan et al., 2005; Mills-Koonce et al., 2009; Sethre-Hofstad et al., 2002; Stallings et al., 2001).

One potential alternative explanation for larger associations in morning and midday samples than in evening samples in our analysis might have to do with differences in the stability of cortisol levels throughout the waking day. Some studies (Doane, Chen, Sladek, Van Lenten, & Granger, 2015; Riese, Rijsdijk, Rosmalen, Snieder, & Ormel, 2009) though not all (Ross, Murphy, Adam, Chen, & Miller, 2014) have shown morning cortisol to be more stable and trait like than evening samples. These differential controls over HPA axis activity might influence the strength of the associations of morning and midday samples vs. afternoon and evening samples with our measure of maternal sensitivity, which we believe taps into a more stable construct of parenting behavior.

Although our findings are consistent with prior literature regarding relations between maternal HPA axis activity and parenting behavior, other measures of HPA axis functioning including cortisol reactivity to a stressor or diurnal cortisol levels sampled repeatedly over the course of the day would have been preferable given their relative ease of interpretation and precedence in the literature. Measures of diurnal activity would have provided us with a more direct test of our hypotheses and with unique information about the relation of risk to maternal HPA axis regulation and about the relation of maternal stress physiological regulation to maternal sensitivity. We are also mindful of other approaches to capture HPA axis functioning including latent state-trait modeling (LST; Doane et al., 2015; Steyer, Mayer, Geiser, & Cole, 2015) in which variation in repeated measures of cortisol is partitioned into both stable trait variation (capturing individual differences) and state variation (capturing occasion or state-specific variation in HPA axis functioning). The ability to partition and predict variance in cortisol levels in this way is informative given that cortisol levels are affected by both endogenous and exogenous variables and because researchers are often interested in addressing questions related to individual differences in HPA axis functioning specifically. Given the very high correlations among mothers' three measures of cortisol within each study wave, and the relatively modest correlation of cortisol between each wave, we did not employ this modeling approach in the present study.

A further limitation concerns the correlational nature of our analyses, which precludes causal interpretations to be made with respect to associations between elevated HPA axis activity and reductions in maternal sensitivity. Specifically, because measures of maternal sensitivity and cortisol occurred simultaneously no directional conclusions can be made.

Notably, however, prior experimental research with non-human animals suggests that manipulation of cortisol levels in non-human animal mothers is associated with significant changes in observed maternal behaviors. In multiparous 3–5 week postpartum marmoset primates, for instance, daily cortisol injection has been associated with less carrying of infants in non-stressful, baseline conditions compared to controls (Saltzman & Abbott, 2009). Other research has shown that primiparous rats who were adrenalectomized displayed lower amounts of maternal care in the early postpartum period (Rees, Panesar, Steiner, & Fleming, 2004), and that subsequent corticosterone replacement in these adrenalectomized rats was associated with increases in maternal behaviors. This suggests a potentially causal link between glucocorticoids and maternal care in non-human animal models, but suggests that prior experiences of mothering as well as time since birth may moderate the extent of this relationship.

In addition, it is important to note that approximately 66% of the total analysis sample was low-income, meaning that the majority of families' reported total household income was below 200% of the federal poverty threshold—$19,157 for a family of four with two children in the year 2004 (U.S. Census Bureau, 2014). Due to the large proportion of low-income families in the analysis sample, the generalizability of the findings may be limited and the effects may be attenuated or increased due to the restricted range. It is essential to note, however, that even within this sample of primarily low-income families, cumulative risk was still associated with maternal cortisol levels. Recent analyses of brain development in children in poverty have shown that effects of risk factors on gray matter volume (Hair, Hanson, Wolfe, & Pollak, 2015) and on cortical surface area (Noble et al., 2015) are most pronounced for children at or below the poverty line. Effects of risk for children in families at 150% of poverty were substantially smaller. If the effects of risk on maternal cortisol follow a similar pattern, our effect size estimates may be somewhat larger than would be observed in a lower risk sample.

Results from the current study have implications for understanding the role that stress hormones, and perhaps more generally, the role that maternal self-regulation, plays in the facilitation and maintenance of parenting behaviors that are responsive, contingent, and warm. With this mind, a promising parenting intervention program, Play and Learning Strategies, PALS (Landry, Smith, & Swank, 2006) is worth noting. PALS is designed to increase maternal responsiveness via the use of video-feedback teaching tools in which a parent's own interactions with her child are videotaped and subsequently discussed and critiqued by the mother with support from program implementers. This reflective process may have the effect of increasing behavioral and affective regulation in mothers, with alterations to the functioning of underlying stress physiological systems including the maternal HPA axis also taking place. Such shifts in the broad indices of maternal self-regulation in the context of intervention would be hypothesized to lead to interactions with children that are more sensitive in nature and to have positive downstream effects on child development in the context of high stress and adversity.

Acknowledgments

The Family Life Project was supported in part by a grant from the National Institute of Child Health and Human Development grant P01 HD39667, with co-funding from the National Institute on Drug Abuse.

References

Ainsworth MDS, Blehar MC, Waters E, Wall S. Patterns of attachment: A psychological study of the strange situation. 1978

Arnsten AF, Li BM. Neurobiology of executive functions: Catecholamine influences on prefrontal cortical functions. Biological Psychiatry. 2005; 57(11):1377–1384. DOI: 10.1016/j.biopsych.2004.08.019 [PubMed: 15950011]

Atkinson L, Leung E, Goldberg S, Benoit D, Poulton L, Myhal N, … Kerr S. Attachment and selective attention: Disorganization and emotional stroop reaction time. Development and Psychopathology. 2009; 21(01):99–126. [PubMed: 19144225]

Barrett J, Fleming AS. Annual research review: All mothers are not created equal: Neural and psychobiological perspectives on mothering and the importance of individual differences. Journal of Child Psychology and Psychiatry. 2011; 52(4):368–397. DOI: 10.1111/j.1469-7610.2010.02306.x [PubMed: 20925656]

Bates JE, Olson SL, Pettit GS, Bayles K. Dimensions of individuality in the mother-infant relationship at six months of age. Child Development. 1982; 53(2):446–461. DOI: 10.2307/1128988 [PubMed: 7075328]

Belsky, J.; Jaffee, SR. The multiple determinants of parenting. In: Cicchetti, D.; Cohen, D., editors. Developmental psychopathology: Vol. 3. risk, disorder, and adaptation. 2. Hoboken, NJ: Wiley; 2006. p. 38-85.

Cameron NM, Champagne FA, Parent C, Fish EW, Ozaki-Kuroda K, Meaney MJ. The programming of individual differences in defensive responses and reproductive strategies in the rat through variations in maternal care. Neuroscience and Biobehavioral Reviews. 2005; 29(4–5):843–865. DOI: 10.1016/j.neubiorev.2005.03.022 [PubMed: 15893378]

Clearfield MW, Carter-Rodriguez A, Merali A, Shober R. The effects of SES on infant and maternal diurnal salivary cortisol output. Infant Behavior and Development. 2014; 37(3):298–304. DOI: 10.1016/j.infbeh.2014.04.008 [PubMed: 24813589]

Cohen S, Doyle WJ, Baum A. Socioeconomic status is associated with stress hormones. Psychosomatic Medicine. 2006a; 68(3):414–420. DOI: 10.1097/01.psy.0000221236.37158.b9 [PubMed: 16738073]

Cohen S, Janicki-Deverts D, Chen E, Matthews KA. Childhood socioeconomic status and adult health. Annals of the New York Academy of Sciences. 2010; 1186(1):37–55. [PubMed: 20201867]

Cohen S, Schwartz JE, Epel E, Kirschbaum C, Sidney S, Seeman T. Socioeconomic status, race, and diurnal cortisol decline in the coronary artery risk development in young adults (CARDIA) study. Psychosomatic Medicine. 2006b; 68(1):41–50. 68/1/41 [pii]. [PubMed: 16449410]

Cox, MJ.; Crnic, K. Unpublished Manuscript. University of North Carolina; Chapel Hill: 2002. Qualitative ratings for parent-child interaction at 3–12 months of age.

Dill, BT. Literature review prepared for the Annie E. Casey Foundation. 1999. Poverty in the rural US: Implications for children, families, and communities.

Doane LD, Chen FR, Sladek MR, Van Lenten SA, Granger DA. Latent trait cortisol (LTC) levels: Reliability, validity, and stability. Psychoneuroendocrinology. 2015; 55:21–35. [PubMed: 25705799]

Dowd JB, Simanek AM, Aiello AE. Socio-economic status, cortisol and allostatic load: A review of the literature. International Journal of Epidemiology. 2009; 38(5):1297–1309. [PubMed: 19720725]

Enders, CK. Applied missing data analysis. New York: Guilford Press; 2010.

Evans GW, Boxhill L, Pinkava M. Poverty and maternal responsiveness: The role of maternal stress and social resources. International Journal of Behavioral Development. 2008; 32(3):232–237.

Fleming AS, Ruble D, Krieger H, Wong P. Hormonal and experiential correlates of maternal responsiveness during pregnancy and the puerperium in human mothers. Hormones and Behavior. 1997; 31(2):145–158. [PubMed: 9154435]

Fleming AS, Steiner M, Anderson V. Hormonal and attitudinal correlates of maternal behaviour during the early postpartum period in first-time mothers. Journal of Reproductive and Infant Psychology. 1987; 5(4):193–205. DOI: 10.1080/02646838708403495

Fleming AS, Steiner M, Corter C. Cortisol, hedonics, and maternal responsiveness in human mothers. Hormones and Behavior. 1997; 32(2):85–98. DOI: 10.1006/hbeh.1997.1407 [PubMed: 9367716]

Giardino J, Gonzalez A, Steiner M, Fleming AS. Effects of motherhood on physiological and subjective responses to infant cries in teenage mothers: A comparison with non-mothers and adult mothers. Hormones and Behavior. 2008; 53(1):149–158. DOI: 10.1016/j.yhbeh.2007.09.010 [PubMed: 18076883]

Gonzalez A, Jenkins JM, Steiner M, Fleming AS. Maternal early life experiences and parenting: The mediating role of cortisol and executive function. Journal of the American Academy of Child & Adolescent Psychiatry. 2012; 51(7):673–682. DOI: 10.1016/j.jaac.2012.04.003 [PubMed: 22721590]

Granger DA, Blair C, Willoughby M, Kivlighan KT, Hibel LC, Fortunato CK, Wiegand LE. Individual differences in salivary cortisol and alpha-amylase in mothers and their infants: Relation to tobacco smoke exposure. Developmental Psychobiology. 2007; 49(7):692–701. DOI: 10.1002/dev.20247 [PubMed: 17943979]

Gunnar MR, Quevedo KM. Early care experiences and HPA axis regulation in children: A mechanism for later trauma vulnerability. Progress in Brain Research. 2007; 167:137–149. [PubMed: 18037012]

Hair NL, Hanson JL, Wolfe BL, Pollak SD. Association of child poverty, brain development, and academic achievement. JAMA Pediatrics. 2015; 169(9):822–829. [PubMed: 26192216]

Heim C, Ehlert U, Hellhammer DH. The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. Psychoneuroendocrinology. 2000; 25(1):1–35. [PubMed: 10633533]

Hibel LC, Mercado E, Trumbell JM. Parenting stressors and morning cortisol in a sample of working mothers. Journal of Family Psychology. 2012; 26(5):738–746. DOI: 10.1037/a0029340 [PubMed: 22866929]

Hibel LC, Granger DA, Kivlighan KT, Blair C. The Family Life Project Investigators. Individual differences in salivary cortisol: Associations with common over-the-counter and prescription medication status in infants and their mothers. Hormones and Behavior. 2006; 50(2):293–300. DOI: 10.1016/j.yhbeh.2006.03.014 [PubMed: 16682032]

Kiel EJ, Buss KA. Toddler inhibited temperament, maternal cortisol reactivity and embarrassment, and intrusive parenting. Journal of Family Psychology. 2013; 27(3):512–517. DOI: 10.1037/a0032892 [PubMed: 23750532]

Kopp CB. Antecedents of self-regulation: A developmental perspective. Developmental Psychology. 1982; 18(2):199–214. DOI: 10.1037/0012-1649.18.2.199

Krpan KM, Coombs R, Zinga D, Steiner M, Fleming AS. Experiential and hormonal correlates of maternal behavior in teen and adult mothers. Hormones and Behavior. 2005; 47(1):112–122. DOI: 10.1016/j.yhbeh.2004.08.006 [PubMed: 15579272]

Landry SH, Smith KE, Swank PR. Responsive parenting: Establishing early foundations for social, communication, and independent problem-solving skills. Developmental Psychology. 2006; 42(4): 627–642. DOI: 10.1037/0012-1649.42.4.627 [PubMed: 16802896]

Lee CL, Bates JE. Mother-child interaction at age two years and perceived difficult temperament. Child Development. 1985; 56(5):1314–1325. DOI: 10.2307/1130246 [PubMed: 4053745]

Li L, Power C, Kelly S, Kirschbaum C, Hertzman C. Life-time socio-economic position and cortisol patterns in mid-life. Psychoneuroendocrinology. 2007; 32(7):824–833. DOI: 10.1016/j.psyneuen.2007.05.014 [PubMed: 17644268]

Lupien SJ, Maheu F, Tu M, Fiocco A, Schramek TE. The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. Brain and Cognition. 2007; 65(3):209–237. DOI: 10.1016/j.bandc.2007.02.007 [PubMed: 17466428]

Martorell GA, Bugental DB. Maternal variations in stress reactivity: Implications for harsh parenting practices with very young children. Journal of Family Psychology. 2006; 20(4):641–647. DOI: 10.1037/0893-3200.20.4.641 [PubMed: 17176199]

Mastorakos G, Ilias I. Maternal and fetal hypothalamic-pituitary-adrenal axes during pregnancy and postpartum. Annals of the New York Academy of Sciences. 2003; 997(1):136–149. [PubMed: 14644820]

McLeod JD, Shanahan MJ. Poverty, parenting, and children's mental health. American Sociological Review. 1993:351–366.

McLoyd VC. Socioeconomic disadvantage and child development. American Psychologist. 1998; 53(2):185–204. DOI: 10.1037/0003-066X.53.2.185 [PubMed: 9491747]

Miller GE, Chen E, Zhou ES. If it goes up, must it come down? chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. Psychological Bulletin. 2007; 133(1):25. [PubMed: 17201569]

Mills-Koonce WR, Propper C, Gariepy J, Barnett M, Moore GA, Calkins S, Cox MJ. Psychophysiological correlates of parenting behavior in mothers of young children. Developmental Psychobiology. 2009; 51(8):650–661. DOI: 10.1002/dev.20400 [PubMed: 19739135]

Mills-Koonce WR, Gariépy J, Propper C, Sutton K, Calkins S, Moore G, Cox M. Infant and parent factors associated with early maternal sensitivity: A caregiver-attachment systems approach. Infant Behavior and Development. 2007; 30(1):114–126. [PubMed: 17292784]

Mills-Koonce WR, Garrett-Peters P, Barnett M, Granger DA, Blair C, Cox MJ. The Family Life Project Key Investigators. Father contributions to cortisol responses in infancy and toddlerhood. Developmental Psychology. 2011; 47(2):388–395. DOI: 10.1037/a0021066 [PubMed: 21142362]

Mizoguchi K, Ishige A, Takeda S, Aburada M, Tabira T. Endogenous glucocorticoids are essential for maintaining prefrontal cortical cognitive function. The Journal of Neuroscience. 2004; 24(24): 5492–5499. DOI: 10.1523/JNEUROSCI.0086-04.2004 [PubMed: 15201321]

Muthén, LK.; Muthén, BO. Mplus user's guide. 7. Los Angeles, CA: Muthén & Muthén; 1998–2012.

NICHD Early Child Care Research Network. Chronicity of maternal depressive symptoms, maternal sensitivity, and child functioning at 36 months. Developmental Psychology. 1999; 35:1297–1310. [PubMed: 10493655]

Noble KG, Houston SM, Brito NH, Bartsch H, Kan E, Kuperman JM, … Libiger O. Family income, parental education and brain structure in children and adolescents. Nature Neuroscience. 2015; 18(5):773–778. [PubMed: 25821911]

Pettit GS, Bates JE. Continuity of individual differences in the mother-infant relationship from six to thirteen months. Child Development. 1984; 55(3):729–739. [PubMed: 6734314]

Pfeffer FT, Danziger S, Schoeni RF. Wealth disparities before and after the great recession. The Annals of the American Academy of Political and Social Science. 2013; 650(1):98–123. DOI: 10.1177/0002716213497452 [PubMed: 25332508]

Putnam, SP.; Sanson, AV.; Rothbart, MK. Child temperament and parenting. In: Bornstein, MH., editor. Handbook of parenting: Vol. 1 children and parenting. 2. Mahwah, NJ: Lawrence Erlbaum Associates; 2002. p. 255-277.

Rees SL, Panesar S, Steiner M, Fleming AS. The effects of adrenalectomy and corticosterone replacement on maternal behavior in the postpartum rat. Hormones and Behavior. 2004; 46(4): 411–419. [PubMed: 15465526]

Riese H, Rijsdijk FV, Rosmalen JG, Snieder H, Ormel J. Neuroticism and morning cortisol secretion: Both heritable, but no shared genetic influences. Journal of Personality. 2009; 77(5):1561–1576. [PubMed: 19678874]

Ross KM, Murphy ML, Adam EK, Chen E, Miller GE. How stable are diurnal cortisol activity indices in healthy individuals? evidence from three multi-wave studies. Psychoneuroendocrinology. 2014; 39:184–193. [PubMed: 24119668]

Saltzman W, Abbott DH. Effects of elevated circulating cortisol concentrations on maternal behavior in common marmoset monkeys (callithrix jacchus). Psychoneuroendocrinology. 2009; 34(8):1222–1234. [PubMed: 19362777]

Sameroff, AJ.; Fiese, BH. Transactional regulation: The developmental ecology of early intervention. In: Shonkoff, JP.; Meisels, SJ., editors. Handbook of early childhood intervention. 2. New York: Cambridge University Press; 2000. p. 135-159.

Satorra A, Bentler PM. A scaled difference chi-square test statistic for moment structure analysis. Psychometrika. 2001; 66(4):507–514.

Seltzer MM, Almeida DM, Greenberg JS, Savla J, Stawski RS, Hong J, Taylor JL. Psychosocial and biological markers of daily lives of midlife parents of children with disabilities. Journal of Health and Social Behavior. 2009; 50(1):1–15. [PubMed: 19413131]

Sethre-Hofstad L, Stansbury K, Rice MA. Attunement of maternal and child adrenocortical response to child challenge. Psychoneuroendocrinology. 2002; 27(6):731–747. DOI: 10.1016/S0306-4530(01)00077-4 [PubMed: 12084665]

Singer, JD.; Willett, JB. Applied longitudinal data analysis: Modeling change and event occurrence. New York: Oxford University Press; 2003.

Sroufe, LA. Emotional development: The organization of emotional life in the early years. Cambridge University Press; 1997.

Stallings J, Fleming AS, Corter C, Worthman C, Steiner M. The effects of infant cries and odors on sympathy, cortisol, and autonomic responses in new mothers and nonpostpartum women. Parenting: Science and Practice. 2001; 1(1–2):71–100.

Steptoe A, Kunz-Ebrecht S, Owen N, Feldman PJ, Willemsen G, Kirschbaum C, Marmot M. Socioeconomic status and stress-related biological responses over the working day. Psychosomatic Medicine. 2003; 65(3):461–470. [PubMed: 12764220]

Steyer R, Mayer A, Geiser C, Cole DA. A theory of states and traits-revised. Annual Review of Clinical Psychology. 2015; 11:71–98.

Sturge-Apple ML, Davies PT, Cicchetti D, Cummings EM. The role of mothers' and fathers' adrenocortical reactivity in spillover between interparental conflict and parenting practices. Journal of Family Psychology. 2009; 23(2):215–225. DOI: 10.1037/a0014198 [PubMed: 19364215]

Swain JE, Lorberbaum JP, Kose S, Strathearn L. Brain basis of early parent–infant interactions: Psychology, physiology, and in vivo functional neuroimaging studies. Journal of Child Psychology and Psychiatry. 2007; 48(3–4):262–287. [PubMed: 17355399]

Swain J, Kim P, Ho SS. Neuroendocrinology of parental response to Baby-Cry. Journal of Neuroendocrinology. 2011; 23(11):1036–1041. [PubMed: 21848646]

Thompson LA, Trevathan WR. Cortisol reactivity, maternal sensitivity, and learning in 3-month-old infants. Infant Behavior & Development. 2008; 31(1):92–106. DOI: 10.1016/j.infbeh.2007.07.007 [PubMed: 17716739]

Tronick EZ. Emotions and emotional communication in infants. American Psychologist. 1989; 44(2):112. [PubMed: 2653124]

Tu MT, Lupien SJ, Walker C. Diurnal salivary cortisol levels in postpartum mothers as a function of infant feeding choice and parity. Psychoneuroendocrinology. 2006; 31(7):812. [PubMed: 16716531]

U.S. Census Bureau. The 2004 poverty threshold tables. 2014. Retrieved from https://www.census.gov/hhes/www/poverty/data/threshld/

van den Boom DC. Sensitivity and attachment: Next steps for developmentalists. Child Development. 1997; 64(4):592–594. DOI: 10.2307/1132108 [PubMed: 9306637]

Vernon-Feagans L, Cox M. The Family Life Project Key Investigators. The family life project: An epidemiological and developmental study of young children living in poor rural communities. Monographs of the Society for Research in Child Development. 2013; 78(5):1–150. DOI: 10.1111/mono.12046 [PubMed: 24147448]

Walker LO, Best MA. Well-being of mothers with infant children: A preliminary comparison of employed women and homemakers. Women & Health. 1991; 17(1):71–89. DOI: 10.1300/J013v17n01_05 [PubMed: 2048323]

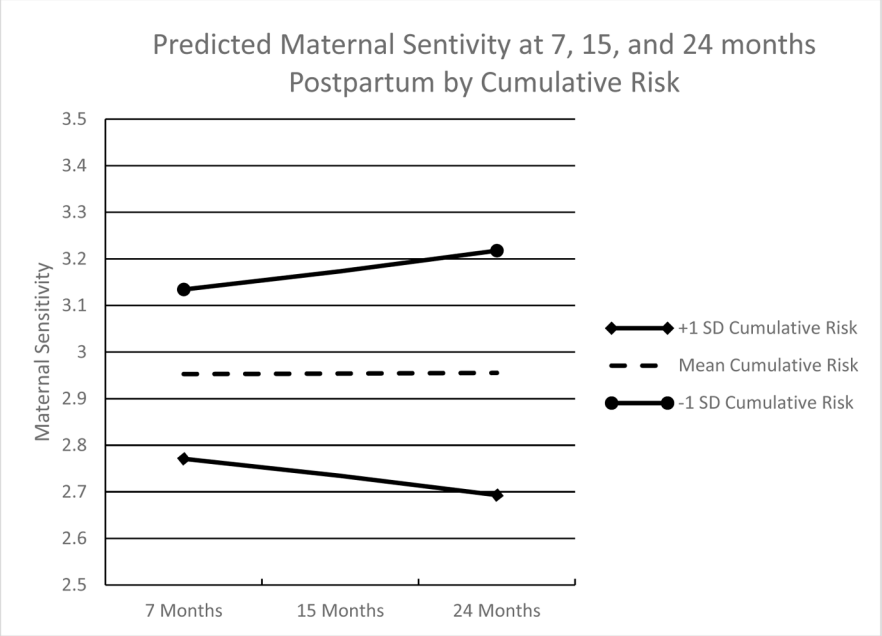

Figure 1.

The relationship between cumulative risk and maternal sensitivity varies as a function of time. Dashed line is prediction for those at average levels of cumulative risk. Solid line with squares is prediction for those at one standard deviation (SD) above the mean of cumulative risk. Solid line with circles is prediction for those at one SD below the mean for cumulative risk. $N = 1,180$

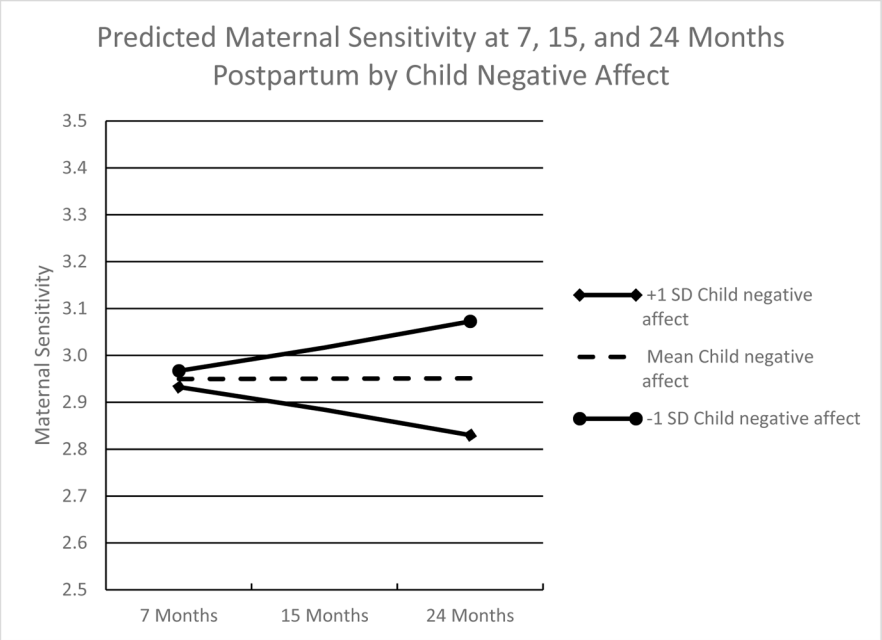

Figure 2.

The relationship between child negative affect and maternal sensitivity varies as a function of time. Dashed line is prediction for those whose children present amounts negative affect at the sample mean. Solid line with squares is prediction for those one standard deviation (SD) above the mean of child negative affect and solid line with circles is prediction for those one SD below the mean for child negativity. $N = 1{,}180$.

Table 1

Descriptives of the analysis variables

	7 months					15 months					24 months				
Time-varying variables	**N**	**Mean**	**SD**	**Min**	**Max**	**N**	**Mean**	**SD**	**Min**	**Max**	**N**	**Mean**	**SD**	**Min**	**Max**
Sensitive parenting	1055	2.90	0.79	1.00	4.80	972	2.79	0.80	1.00	5.00	906	2.91	0.81	1.00	4.80
Cortisol sample 1 (μg/dl)	1009	0.21	0.16	0.03	1.34	778	0.13	0.10	0.01	0.88	712	0.15	0.12	0.02	0.78
Cortisol sample 2 (μg/dl)	1003	0.16	0.12	0.02	1.11	774	0.10	0.09	0.01	0.73	705	0.11	0.09	0.01	0.79
Cortisol sample 3 (μg/dl)	991	0.14	0.10	0.02	0.93	751	0.10	0.07	0.01	0.67	698	0.10	0.08	0.01	0.63
Cortisol mean (ln μg/dl)	1021	−1.98	0.64	−4.07	0.30	791	−2.40	0.62	−4.47	−0.27	718	−2.31	0.65	−4.16	−0.52
Cumulative risk	1055	−0.02	0.66	−2.21	1.98	972	−0.02	0.65	−2.41	2.00	906	−0.01	0.61	−2.30	1.49
Child negative affect	1050	1.68	1.00	1.00	5.00	972	1.94	1.06	1.00	5.00	905	2.20	1.03	1.00	5.00
Months postpartum	1055	7.83	1.44	5.16	15.39	972	15.74	1.32	14.01	22.36	906	25.07	1.96	22.59	34.79
Time of day	1048	13.56	2.91	8.20	20.13	966	13.94	2.88	8.75	20.33	895	13.79	3.20	8.33	20.80
Body mass index	991	28.71	7.21	14.65	51.60	-	-	-	-	-	840	29.20	7.59	13.40	54.17
Had new child (% yes)	1055	0%				972	6%				906	11%			
Contraceptive use (% yes)	1054	28%				972	36%				905	29%			
Tobacco smoking (% yes)	1051	31%				-	-				904	35%			
Time-invariant variables	N	Mean	SD	Min	Max										
Age of mother	1180	25.88	5.80	14.58	44.57										
Primiparous (% yes)	1178	39%													
Breastfeeding 7 months (% yes)	1180	13%													
NC Black (% yes)	1180	40%													
NC White (% yes)	1180	20%													
PA White (% yes)	1180	40%													

Note: NC = North Carolina; PA = Pennsylvania; ln = natural log; Dash (−) indicates that variable was not collected at that time point; N = 1,180

Table 2

Zero order correlations among the analysis variables

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1 Cortisol (ln)	--													
2 Cortisol residual	.83**	--												
3 Cumulative risk	.13**	.08**	--											
4 Sensitive parenting	−.09**	−.09**	−.50**	--										
5 Child negative affect	−.01	.02	.06**	−.12**	--									
6 Months postpartum	−.22**	−.01	.02	−.01	.18**	--								
7 Time of day	−.46**	.00	−.19**	.09**	−.03	.03	--							
8 Body mass index	−.09**	−.08**	.13**	−.13**	.01	.03	.01	--						
9 Had new child	−.08**	−.05*	.08**	−.01	.02	.20**	−.05*	.06**	--					
10 Age of Mother	−.05**	−.02	−.48**	.33**	−.04*	.00	.06**	.05*	−.07**	--				
11 Primiparous	.03	.04	−.01	−.03	.06**	−.03	.04*	−.05*	.03	−.36**	--			
12 Tobacco smoking	.11**	.10**	.26**	−.14**	.06**	.02	−.07**	−.06**	.01	−.21**	.05*	--		
13 Breastfeeding	−.08**	−.07**	−.32**	.29**	−.02	−.03	.04	−.15**	.01	.30**	−.07**	−.16**	--	
14 Contraceptive use	.06**	.00	−.05**	.01	.01	.00	.06**	−.06**	−.01	−.16**	.22**	−.03	−.10**	--

* $p < .05$;

** $p < .01$

Table 3

Mixed model predicting maternal salivary cortisol at 7, 15, and 24 months postpartum

Fixed effects	**b (*SE*)**	**ES**
Intercept	−2.22 (0.04) ***	
Linear slope	−0.26 (0.02) ***	
NC Black	0.11 (0.04) **	0.19
PA White	0.09 (0.03) *	0.15
Time of day	−0.10 (0.00) ***	1.03
Age of mother	0.01 (0.00) *	0.14
Primiparous	0.07 (0.03) *	0.11
Had new child	−0.13 (0.05) **	0.10
Contraceptive use	0.10 (0.03) ***	0.16
Tobacco smoking	0.11 (0.03) ***	0.18
BMI	−0.01 (0.00) ***	0.18
Breastfeeding	−0.11 (0.04) **	0.13
Cumulative Risk	0.06 (0.03) *	0.13
Random effects	σ2 (*SE*)	
Intercept	0.05 (0.01) ***	
Residual	0.26 (0.01) ***	

Note: NC = North Carolina; PA = Pennsylvania; BMI = Body mass index; Time centered at 7 mos. visit ES = Effect size;

Effect sizes were calculated as (b * SD(x)) / SD(y) where SD(y) was based on the variance term for the intercept in the unconditional growth model;

* $p < .05$;

** $p < .01$;

*** $p < .001$

Table 4

Mixed models predicting maternal sensitivity at 7, 15, and 24 months postpartum

	Model 1		Model 2		Model 3	
Fixed effects	**b (*SE*)**	**ES**	**b (*SE*)**	**ES**	**b (*SE*)**	**ES**
Intercept	2.96 (0.05) ***		2.95 (0.05) ***		2.95 (0.05) ***	
Linear slope	0.00 (0.02)		0.00 (0.02)		0.00 (0.02)	
NC Black	−0.31 (0.05) ***	0.26	−0.31 (0.05) ***	0.26	−0.31 (0.05) ***	0.26
PA White	−0.01 (0.05)		0.00 (0.05)		0.01 (0.05)	
Age of mother	0.02 (0.00) ***	0.17	0.02 (0.00) ***	0.17	0.02 (0.00) ***	0.17
Breastfeeding	0.25 (0.05) ***	0.15	0.25 (0.05) ***	0.15	0.24 (0.05) ***	0.15
Primiparous	0.02 (0.04)		0.02 (0.04)		0.02 (0.04)	
Had new child	0.08 (0.05) †		0.08 (0.05)		0.08 (0.05) †	
Tobacco smoking	−0.06 (0.04) †		−0.06 (0.04) †		−0.06 (0.04) †	
BMI	−0.01 (0.00) *	0.06	−0.01 (0.00) *	0.06	−0.01 (0.00) *	0.06
Cumulative Risk	−0.34 (0.03) ***	0.38	−0.34 (0.03) ***	0.38	−0.29 (0.03) ***	0.32
Child negative affect	−0.07 (0.01) ***	0.12	−0.07 (0.01) ***	0.12	−0.02 (0.02)	
Cortisol			−0.05 (0.02) *	0.05	−0.08 (0.03) *	0.07
Cortisol X time					0.04 (0.04)	
Cumulative risk X time					−0.09 (0.03) **	0.30
Child negative affect X time					−0.07 (0.02) ***	0.39
Random effects	σ^2 (*SE*)		σ^2 (*SE*)		σ^2 (*SE*)	
Intercept	0.20 (0.01) ***		0.20 (0.01) ***		0.20 (0.01) ***	
Linear slope	0.02 (0.01)		0.02 (0.01)		0.01 (0.01)	
Residual	0.24 (0.01) ***		0.24 (0.01) ***		0.24 (0.01) ***	

Note: NC = North Carolina; PA = Pennsylvania; Time was centered at 7 month visit in all models; BMI = Body Mass Index ES = Effect size; Effect sizes were calculated as (b * SD(x)) / SD(y) where SD(y) was based on the variance terms for the intercept and slope in the unconditional growth model;

† $p < .10$;

* $p < .05$;

** $p < .01$;

*** $p < .001$;

N = 1,180.