Published in final edited form as:

Child Dev. 2015 July ; 86(4): 1142–1158. doi:10.1111/cdev.12376.

Tracing Differential Pathways of Risk: Associations Among Family Adversity, Cortisol, and Cognitive Functioning in Childhood

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Abstract

Guided by family risk and allostasis theoretical frameworks, the present study utilized a prospective longitudinal design to examine associations among family risk experiences, basal cortisol patterns, and cognitive functioning in children. The sample included 201 low-income children living within a mid-size city in the Northeastern United States. Children were assessed at ages 2, 3, and 4 years. Growth-mixture modeling (GMM) analyses revealed three basal cortisol patterns (elevated, moderate, low) and these remained relatively stable across time. Exposure to greater levels of family instability and maternal emotional unavailability predicted elevated and low cortisol patterns, which were associated with lower child cognitive functioning at age 4. Findings have implications for family risk processes that may underlie risk-related disparities in child cognitive outcomes.

Keywords

Family Adversity; Cortisol; Child Cognitive Functioning

In the United States, 22% of children live in poverty and 45% come from low-income families (Jiang, Ekono, & Skinner, 2014). Children living in poverty are 1.3 times more likely than higher income children to experience learning and developmental delays

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(Brooks-Gunn & Duncan, 1997). Documenting these effects has been paramount in establishing associations between economic adversity and cognitive development, with research suggesting that prior to kindergarten, children from higher socioeconomic backgrounds have cognitive abilities that are 60% higher than those of children in the lowest socioeconomic group (e.g., Lee & Burkam, 2002). However, socioeconomic status only represents one global indication of risk and much of the prior research has lacked precision in identifying the proximal family risk factors associated with poverty that might have the most pathogenic influence on children's developing cognitive skills. A smaller body of literature that has focused on family-level risk factors has shown that family instability (e.g., Vernon-Feagons, Garrett-Peters, Willoughby, & Mills-Koonce, 2012), unsupportive and harsh parenting (e.g., Linver, Brooks-Gunn, & Kohen, 2002) and exposure to interpartner violence (e.g., Margolin & Gordis, 2004) may have detrimental effects on children's cognitive development. However, we still know very little about the underlying mechanisms that might account for the transmission of family risk processes within the context of poverty in shaping children's cognitive functioning.

Family risk frameworks offer a theoretical guide for delineating the pathways through which different types of family adversity may shape individual differences in human development across the lifespan (Repetti, Robles, & Reynolds, 2011; Repetti, Taylor, & Seeman, 2002). For example, Repetti and colleagues' (2002, 2011) "risky" family model proposes that physiological stress response systems, and in particular the hypothalamic-pituitary-adrenal (HPA) axis, represent biological mechanisms through which family risk processes influence physical and mental health outcomes across the lifespan. The primary aim of the present study was to examine whether differential exposure to family risk processes during the first two years of life was associated with variations in children's basal HPA activity over time and, in turn, their cognitive functioning at age 4. To date, only one study, conducted by Blair and colleagues (2011a), has simultaneously examined associations among poverty, parenting practices, and cortisol on children's developing cognitive abilities in early childhood. In this seminal study, Blair and colleagues (2011a) found evidence to suggest that cortisol and lack of positive parenting may both play critical mediating roles in influencing poor cognitive outcomes among preschool-age children living in poverty. Utilizing a family risk framework, the present analysis sought to expand upon this research in three primary ways. First, the present study aimed to increase precision in identifying the specific social and emotional aspects of family life that low-income children experience that may differentially shape their HPA activity during this developmental window. Second, it has been long assumed that multiple phenotypes of stress physiology may emerge based on environmental expectancies in order to maximize organismic functioning within risk contexts (e.g., Blair & Raver, 2012); however, these patterns cannot be fully determined with variable-centered approaches, which prior studies have predominately utilized. Therefore, our second aim was to employ a pattern-based analysis to examine whether specific types of family risk processes might differentially shape trajectories of children's HPA axis functioning across early childhood. Our final aim was to determine whether patterns of basal cortisol activity would be further differentiated by differences in cognitive outcomes when children were preschool age.

Family Risk and Children's Adrenocortical Functioning

Economic distress and ecological risk factors (e.g., neighborhood crime, unstable housing conditions) are known to place a considerable amount of strain on the family unit and as a result, disproportionately expose children to multiple forms of family adversity (e.g., Evans, 2004). Within contexts of poverty, proximal family stressors may dramatically shape children's developing physiological systems as they must repeatedly adapt to the threatening and stressful events that chronically occur in their home environments (Repetti et al., 2002, 2011). According to recent applications of family risk frameworks to early child developmental research, the HPA axis and its end product cortisol is one of the primary allostatic mechanisms that link associations between exposure to specific family stressors and negative developmental sequelae (Repetti et al., 2011). The regulation of the HPA axis is directed by neural structures that support emotional and cognitive responses to environmental stress, and it is through a complex set of direct influences and feedback interactions among hormones produced by the hypothalamus, pituitary gland, and adrenal glands, that cortisol is released. In response to threat and challenge, cortisol mobilizes metabolic resources in the brain and body, and modulates the processing, encoding, and memory consolidation of emotionally significant and distressing events. Based on family risk models, efficient calibration of the HPA axis within adverse family contexts is essential for developing regulatory strategies that can meet the demands of these environments (e.g., Blair et al., 2011b; Repetti et al., 2011). Disruptions in family functioning due to poverty (Cutuli, Wiik, Herbers, Gunnar, & Masten, 2010; Evans & Kim, 2007), interpartner violence (Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009), and emotionally withdrawn and unsupportive caregiving practices (Bugental, Martorell, & Barazza, 2003; Fisher, Stoolmiller, Gunnar, & Burraston, 2007) are defining features of risky family environments and each have been linked to influencing alternations in HPA axis regulation in children exposed to these family stressors.

First, family instability has been conceptualized as the experience of heightened levels of adverse, disruptive family events (e.g., such as caregiver changes, housing instability), which often occur at higher frequencies within low-income contexts (Evans, 2004). Moreover, family instability reflects a general breakdown in the ability of the family to provide a cohesive, predictable, and safe socialization environment for the child. In turn, a lack of consistent structure may lead to an increase in sensitivity or dampening in children's HPA activity in order to promote survival within a high threat and unpredictable family environment. For example, Cutuli and colleagues (2010) demonstrated that family instability, defined as a history of family level disruptions and problems, was linked to higher baseline cortisol among homeless children. Conversely, a few recent investigations have documented low cortisol patterns among children exposed to multiple negative family life events (Zalewski, Lengua, Kiff, & Fisher, 2012) and heightened levels of financial strain (Badanes, Watamura, & Hankin, 2011).

Second, repeated exposure to parental conflicts containing hostility, violence, and escalating anger may represent a potent source of personal threat and potential disruption to the family unit (e.g., Davies & Sturge-Apple, 2006). Given the stress-sensitive nature of the HPA axis, exposure to interpartner conflict might have a profound influence on children's

adrenocortical activity. Prior research has detected elevated basal cortisol levels among children who live in family environments with heightened levels of interparental aggression (Davies et al., 2009).

Lastly, caregiver emotional unavailability and insensitive parenting practices undermine children's ability to utilize them as a source of protection and support (e.g., Ainsworth, Blehar, Waters, & Wall, 1978). Deviations from average levels of caregiver responsiveness and availability have been hypothesized to play a potent role in influencing atypical patterns in children's basal cortisol activity (Bugental et al., 2003; Bugental, Schwartz, & Lynch, 2010). Therefore, building off previous research, the present study simultaneously examined whether early exposure to family instability, interpartner violence, and emotionally unavailable caregiving practices were differentially linked to patterns of children's adrenocortical functioning across the ages of 2, 3, and 4 years.

Applying a Pattern-Based Approach to Adrenocortical Functioning in Family Risk

Much of the extant research examining the role of family contexts in shaping children's adrenocortical functioning has typically relied on variable-based approaches to delineate how risk experiences influence individual differences in children. However, person-based approaches may be preferable for capturing the dynamics of HPA axis activity and identifying multiple phenotypes of children's stress-response patterns that emerge under adverse caregiving conditions (e.g., Cicchetti & Rogosch, 1999). In particular, models of allostasis propose that different forms of basal HPA activity emerge within stressful contexts because "different circumstances demand different homeostatic set points" (Sapolsky, 1998, p. 7; see also, McEwen & Stellar, 1993). Concepts of allostasis and allostatic load have been utilized as guides for hypothesizing the different forms of stress response patterns that might be observed in children exposed to different types of family risk processes (e.g., Repetti et al., 2011).

First, the *hypercortisolism* hypothesis proposes that chronic exposure to environmental challenges may eventuate in the HPA system becoming increasingly sensitive and overactive in marshaling resources to cope with environmental threats. Consequently, repeated exposure to environmental difficulties may lead to heightened or amplified HPA activity and elevations in basal cortisol in children as a result of being in a constant state of preparation for threat to occur within the family context. In contrast, the *hypocortisolism hypothesis* posits that exposure to chronic environmental stressors may lead to enhanced negative feedback in the HPA axis, resulting in the suppression of adrenocortical activity. One hypothesis for this effect is that children exposed to chaos and frequent disruptions within the family system may develop lower basal cortisol patterns in order to prohibit the excessive expenditure of metabolic resources and protect the body from damage caused by the toxic effects of stress hormones on bodily tissues (e.g., Gunnar and Vazquez, 2001). Empirical research has identified the presence of both forms of adrenocortical functioning in children exposed to varying types and degrees of family risk processes (e.g., Cicchetti & Rogosch, 2001); however, few have sought to simultaneously model these patterns (e.g., Van

Ryzin, Chatham, Kryzer, Kertes, & Gunnar, 2009), which has limited current understanding of under which family risk conditions the development of a lower versus higher basal cortisol pattern is more probable. Therefore, in order to test for the presence of multiple patterns of HPA axis functioning, we utilized a pattern-based approach to parameterize children's basal activity across three consecutive years of early childhood. The developmental utility of a mixture approach for differentiating patterns of basal cortisol activity for family risk models is predicated upon the ability to meaningfully link these patterns with specific proximal risk factors. Therefore, the current study also examined whether differences in exposure to family instability, maternal emotionally unavailable caregiving, interpartner violence, and severity of economic hardship meaningfully predicted individual differences in basal cortisol trajectories among a sample of low-income children.

Patterns of Adrenocortical Activity and Cognitive Development

A final aim of the present study was to examine whether different patterns of adrenocortical activity were associated with meaningful differences in children's cognitive development. With prolonged or repeated exposure to environment stress, it has been proposed that individual differences in steady-state levels of cortisol may eventuate in structural and functional modifications to brain structures that influence the development of cognitive abilities (e.g., McEwen, 2006). This is particularly pertinent to early childhood when rapid changes in cognitive abilities occur, likely reflecting the protracted development of prefrontal and hippocampal brain regions. Studies in animal models and within the adult stress literature have demonstrated the inverted U-shape relation between glucocorticoids and cognitive functioning; such that, moderate quantities of cortisol seem to facilitate and sustain cognitive functioning and consolidate learning experiences, while chronically high or low levels of glucocorticoids have damaging effects on neuronal structures and can lead to impairments in a wide range of cognitive processes (e.g., attention, spatial learning, and memory) (e.g., de Kloet, Oitzl, & Joëls, 1999; Erikson, Drevets, & Schulkin, 2003). Extant research has largely focused on adrenocortical functioning as a developmental pathway between chronic stress and cognitive functioning in adults. This has limited knowledge of the developmental pathways through which early aberrations in basal HPA activity might eventuate in disturbances in cognitive functioning, and this might partially account for cognitive delays that are often observed among low-income preschool-age children.

In sum, the objectives of the present study were three-fold. First, couched within a family risk framework, the present investigation aimed to utilize a pattern-based approach to identify three primary forms of HPA activity within a sample of low-income children assessed across three consecutive years of early childhood. Drawing upon the concept of allostasis, we hypothesized that three distinct adrenocortical profiles— an elevated, low, and moderate— would emerge across time. Second, using a prospective longitudinal design, our next aim was to advance family risk frameworks by testing whether specific constellations of family risk factors measured at age 2 predicted different phenotypes of children's basal cortisol patterns across time. The application of a pattern-based approach was partly exploratory, and we did not propose any firm hypotheses with respect to the magnitude or direction of associations between specific family risk factors and children's cortisol patterns due to large inconsistencies in cortisol findings in the developmental literature. However,

based on findings from a few prior studies of preschool-age children, we expected that a low basal cortisol pattern would be predicted by family instability and severity of economic hardship (Badanes et al., 2011; Zalewski et al., 2012). In addition, consistent with prior studies of children exposed to marital violence (Davies et al., 2009) and maternal emotional unavailability (Bugental et al., 2003, 2010), we expected that exposure to interpartner aggression and increased levels of maternal emotional unavailability would predict higher resting cortisol patterns in children across time. Our third aim was to examine whether different patterns of cortisol were associated with meaningful differences in children's cognitive functioning at age 4. Based on research demonstrating an inverted U-shape relation between HPA axis activity and human cognitive functioning, we hypothesized that elevated and low basal cortisol patterns would be associated with poorer cognitive outcomes among children at age 4 (McEwen & Sapolsky, 1995).

Method

Participants

Participants included 201 2-year-old children and their mothers that were recruited from the Rochester area, a moderately sized city in the Western New York region of the United States. Mothers and their children participated in data collection over a three year period beginning in 2005 when children were 2 years old. In order to obtain a sample of families experiencing elevated levels of socio-demographic adversity and interpartner aggression, mothers and children were recruited through community agencies such as, Women, Infant, and Children assistance offices, Temporary Assistance to Needy Families rosters from the Department of Human and Health system, and the county family court system. Inclusionary study criteria included that a) female caregivers were biological mothers, b) child participants were 27 months old (± 5 months), and c) child participants did not have any serious developmental disabilities or brain injuries. Median annual income for the family household among the participants in the sample was \$18,300 (US) per year. A substantial portion of mothers (30%) and their partners (24%) did not complete high school. Most families were receiving public assistance (95%) and were living below the US Federal Poverty line (99.5%). Furthermore, based on the computed Hollingshead Four Factor Index, the majority of families (77%) were rated in the two lower social strata (i.e., unskilled or semiskilled workers).

The mean age of children at the first wave of assessment was 26 months ($SD=1.69$) with nearly half of the sample consisting of girls (44%, $n=92$). Of the 201 2-year-old children and mothers in the sample, the majority identified themselves as Black (56%), with smaller proportions of family members identified as White (23%), Latino (11%), Multiracial (7%), and Other (3%). Mothers also answered questions about their marital status and 63% reported not being married but living with someone, 23% were married, 5% were widowed, and 9% were separated. Of the children in sample, 73% of them lived with both their biological mother and biological father. The remaining children in the study lived with their mother and the target partner was either a stepfather or romantic partner. Inclusionary criteria for the study stipulated that the mother, the target partner, and the child were together at least 1 day, every 2 weeks over the past year.

The cumulative retention rate across the three annual measurement occasions was 87%. To test for selective attrition, we conducted statistical comparisons between the mother-child dyads that participated through the third measurement occasion and dyads that dropped out during the longitudinal component of the study along the primary, covariate, and demographic variables at the first assessment (e.g., family income, maternal education, maternal emotional unavailability, interpartner violence). No significant differences were identified in the analyses.

Procedure

Data collection was conducted at three annual measurement occasions, each spaced one year apart. During each annual wave of data collection, mothers and their children visited the research laboratory twice within a period of approximately two weeks. The University's institutional review board approved the research procedures prior to conducting the study.

Saliva Collection—For each annual wave of data collection, saliva samples were collected at two different visits on different days within a two-week window of time. Per visit, experimenters collected one saliva sample from children within 20 minutes after arrival to laboratory. A total of six saliva samples, two per annual time-point, were collected as baseline measures of children's cortisol across the three years of the study. In children it has been documented that there is a 20–25 minute delay in cortisol expression and so the timing of collection was specifically designed to measure HPA activation prior to their arrival to the laboratory (e.g., Kirschbaum & Hellhammer, 1989). Visit times across data collection were limited to a narrow period in the morning, which ensured uniformity in sampling procedures at each visit. The mean collection times per wave were as follows, Wave 1 ($M = 9:37$ AM, Range: 8:37 AM – 10:55 AM), Wave 2 ($M = 9:29$ AM, Range: 8:07 AM – 10:48 AM), Wave 3 ($M = 9:25$ AM, Range: 8:09 AM – 12:54 PM). To avoid the effects of dynamic cortisol awakening response, mothers were instructed to wake their toddlers at least one hour prior to the visit. In addition, we requested that toddlers not eat or drink at least 30 minutes prior to saliva collection (Susman et al., 2007). Experimenters followed identical saliva sampling procedures across all six visits, which included developing rapport with the families and inviting children to play with toys and get acquainted with the laboratory space. A sorbette was held under the child's tongue by a research assistant for one minute to ensure a sufficient quantity of saliva was obtained. Each sorbette was placed in a 2 mL cryovial and immediately stored at -80°C until shipped on dry ice to Salimetrics, LLC. (State College, PA).

Measures

Maternal Emotional Unavailability—At the first wave, maternal emotional unavailability was assessed through behavioral observation of the mother and her child during a 10-minute interaction task where mother-child dyads were instructed to play with various toys and puzzles within a room. To assess for maternal emotional unavailability, observer ratings were completed using the Iowa Family Interaction Rating Scales (IFIRS; Melby & Conger, 2001). The IFIRS is a global coding system that measures behavioral and emotional traits of parents during dyadic interactions. Based on previous conceptualizations of maternal emotional unavailability (e.g., Biringen & Easterbrook, 2012), two scales were

utilized to form a composite of parenting. *Insensitive/parent-centeredness* was a measurement of the extent to which the mother lacks an awareness of the child's needs, moods, interests, and capabilities. When the mother's interactions with the child are paced to her behavior and mood and are not well timed, the mother appears to "miss" opportunities to appropriately engage the child and appears to enforce rules, regulations, and constraints without considering the child's age-appropriate level of choice, control, or autonomy.

Disengagement was measured by maternal behaviors that seemed to serve the purpose of creating physical or emotional distance between the mother and the child. Examples of these behaviors include: when the mother ignores the child, chooses not to participate in play with the child, appears uninterested or bothered by the child, or shows a lethargic or apathetic attitude toward the child. Ratings were assessed on nine-point Likert-type scales ranging from 1 (not characteristic at all) to 9 (mainly characteristic). Inter-rater reliability was based on two independent coders for 25% of the interactions. Intra-class correlations were .83 and .94 for insensitive/parent-centeredness and disengagement scales respectively. Given overlap between the two scales ($r = .35$), a composite was formed to create a single indicator score for maternal emotional unavailability.

Interpartner Violence—Interpartner violence was assessed using a semi-structured, narrative interview with the mother on the Interparental Conflict Characteristics (ICC) Module of the Interparental Disagreement Interview (IDI: Davies, Sturge-Apple, Cicchetti, Manning, & Vonhold, 2012). The ICC module of the IDI is a semi-structured, narrative interview with the mother designed to assess the frequency, nature, course, and aftermath of interpartner conflicts witnessed by child participants. Video recordings of the interview were subsequently coded for maternal and partner aggression and anger in interpartner conflicts. Aggression was operationalized by acts of physical or verbal hostility directed toward the partner in the interpartner relationship, whereas signs of irritation, tension, anger, or fury were used as indications of anger. Coders rated each construct along seven-point scales ranging from 0 (no sign of anger or aggression) to 6 (high levels of anger or aggression); at the extreme, these are partner behaviors that reflect considerable dysregulation, disorganization or loss of control. Intraclass correlation coefficients reflecting inter-rater agreement indices for ratings of aggression and anger across mother and partner ranged from .78 to .92. Due to high correlations between ratings of partner and spousal aggression ($r = .54$) and anger ($r = .47$), the codes were summed together to form target constructs of interpartner aggression and anger. Finally, the aggression and anger scores were averaged to create a composite score of interpartner violence ($r = .89$). Research has provided evidence for the validity of IDI codes through its correlations with established assessments of interpartner discord and child psychological and physiological functioning (Davies et al., 2009).

Socioeconomic Status—Mothers completed the Mt. Hope Demographics Interview (Cicchetti & Carlson, 1979) at the first wave of data collection. Mothers' report of their own and their partner's educational level and occupational status were used to compute a social strata score using the Hollingshead Four Factor Index of Social Status (Hollingshead, 1975). Scores ranged from 1 to 9, with higher scores indicating higher social status. In addition,

mothers reported on the earned annual income of the family unit, excluding public assistance supplements.

Family Instability—At the first wave, family instability was measured through maternal report on the Family Instability Questionnaire (FIQ; Ackerman, Kogos, Youngstrom, Schoff, & Izard, 1999; Forman & Davies, 2003). On the FIQ, caregivers answer questions that assess the cumulative number of occurrences of eight disruptive family events over the past three years across domains of (a) caregiver changes, (b) residential changes, (c) caregiver intimate relationship changes, (d) job/income loss, and (e) family member deaths. Prior research has shown strong associations between the FIQ and child adjustment, supporting its importance as a theoretical construct in ecological models of child development (Forman & Davies, 2003).

Salivary Basal Cortisol—All samples were assayed for salivary cortisol in duplicate using a highly sensitive enzyme immunoassay (Salimetrics, PA). The test uses 25 µl of saliva per determination, has a lower limit of sensitivity of 0.003 µg/dl, standard curve range from 0.012 to 3.0 µg/dl, and average intra- and inter-assay coefficients of variation 3.5 % and 5.1 % respectively. Method accuracy, determined by spike and recovery, and linearity, determined by serial dilution, were 100.8 % and 91.7 % respectively. Values from matched serum and saliva samples showed the expected strong linear relationship, $r(63) = 0.89$, $p < 0.0001$ (Salimetrics, 2005).

Cortisol data were checked for possible outliers, and 12 subjects (6%) evidenced values greater than 3.5 standard deviations away from the mean. These values were removed and their cortisol assessment for that wave was based upon the second sample collected. For two participants, cortisol samples across both assessments were unusable and were removed from analyses. Intra-class correlations between the two cortisol assessments collected at each wave, on two separate occasions and two weeks apart, ranged from $ICC = .57$ to $.64$ ($p < .001$) and were averaged to create a composite basal cortisol score for each annual assessment. To further test the reliability of our cortisol measures, we conducted principle component analyses on the cortisol measures within each wave and found that across all three waves, only a single component was extracted. Cortisol variable loadings ranged from $r = .85$ to $.86$ across the three waves and 72% to 74% of the cumulative variance in the original cortisol values was explained by the single component at each wave. Our cortisol methods are also in alignment with prior studies, which similarly averaged two morning cortisol measures to form an index of basal cortisol with samples of high-risk children (Bruce, Fisher, Pears, & Levine, 2009; Lupien, King, Meaney, & McEwen, 2001).

Cognitive Functioning—The Vocabulary and Block Design subtests of the Wechsler Preschool and Primary Scale of Intelligence (WPPSI; Wechsler, 2002) were administered to assess child global cognitive ability at age 4. We used the IQ full-scale estimate derived from the two subtests in the primary analyses.

Results

Preliminary Analyses

Tables 1 and 2 provide the raw means, total N , standard deviations, ranges, and correlations among the primary variables in the analyses. Missing data analyses for the primary variables and covariates ($Median = 7\%$, $Range = 0 - 26\%$) indicated that data were missing completely at random (MCAR) based on Little's MCAR test (Little, 1988; Schlomer, Bauman, & Card, 2010), $\chi^2 = 109.38$, $df = 107$, $p = .42$. Therefore, in order to retain the maximum amount of statistical power, we utilized missing data estimation available in Mplus 7.0 and included the full sample in the analyses (Muthén & Muthén, 2012).

Primary Analyses

To parameterize our model of children's basal cortisol levels over time, we performed a growth-mixture modeling analysis (GMM; e.g., Li, Duncan, Duncan, & Acock, 2006). Given the even spacing of assessment waves, we specified a linear growth model for the trajectory of the data. Factor loadings on the growth curve were centered on the initial cortisol assessment, identifying the mean cortisol levels at baseline as the intercept value of the growth function. Although children were assessed in the morning, cortisol shows a strong diurnal variation whereby the highest levels occur at wake-up time in the morning, followed by a steep decline over the morning hours, and then waning in the afternoon. Children's cortisol samples were correlated with maternal reports of their wake-up times, r 's ranged from .28 to .41, p 's $< .01$. Following previous recommendations (e.g., Sturge-Apple, Davies, Cicchetti, & Manning, 2012), we controlled for the effects of variability in the length between child wake-up time and assessment time on cortisol in the growth curve analyses. We accomplished this by regressing maternal reports of child wake-up times (Wave 1 $M = 7:37$ AM, $Range = 4:45$ AM – $9:53$ AM; Wave 2 $M = 7:42$ AM, $Range = 6:00$ AM – $9:30$ AM; Wave 3 $M = 7:36$ AM, $Range = 4:15$ AM – $10:15$ AM) onto the manifest cortisol indicators of the latent growth curve. Time of day of cortisol collection was not controlled for in analyses because collection time was not consistently associated with children's cortisol values.

Our next analytic step was to examine whether there were latent groups in the linear growth model. All models were estimated using the latent variable software Mplus, Version 7.0 (Muthén & Muthén, 2012). The flexibility of MPlus for growth mixture modeling allows researchers to freely explore how the patterns emerge. Based on our *a priori* hypotheses, we conducted our nested GMMs with the expectation that three profiles would emerge from the data including, a regular or moderate cortisol group, a low cortisol group, and an elevated or high cortisol group. To determine the optimal number of profiles, we used three tests for comparing nested models, including the Bayesian information criterion (BIC; Schwarz, 1978), Entropy (e.g., Muthén, & Muthén, 2004), and the Vuong–Lo–Mendell–Rubin likelihood ratio test (VLMR; Lo, Mendell, & Rubin, 2001). The model with the lowest BIC value is generally considered the best-fitting model. As a further test of model fit, the VLMR provides a p value that indicates whether the $k-1$ profile model is rejected in favor of the k profile model (see Nylund, Asparouhov, & Muthén, 2007). Nested models are tested until the VLMR indicates no significant difference in fit for the k class solution compared to the

k-1 solution, indicating that the more parsimonious model should be retained. Finally, examination of entropy shows the confidence level for how individuals were classified, with values higher than .70 indicating greater confidence (Muthén & Muthén, 2004). In addition, it is also important to consider the practical applications of profile solutions. Therefore, substantive implications were also taken into account when determining the optimal number of profiles (Muthén & Muthén, 2004).

We explored several possible parameterizations of latent trajectories of basal cortisol. To test for the best profile solution, we extracted one, two, three, and four growth-mixtures in the model analyses and compared fit indices as well as substantive interpretations across the different solutions. In our modeling, we specified that mean level differences in latent intercept and latent slope factors could vary across classes. We also constrained error variances on the manifest indicators of cortisol to be equal to one another across waves, with the assumption that random fluctuations in cortisol are equal to one another in the time series. Finally, to maximize our class distinctions, we constrained the variances of the mean level intercept and slope constructs to be equal to one another across classes.

The results of model testing across the number of classes are presented in Table 3 and revealed that model fit was best for the three-class solution. The 3-class model had the lowest BIC value when compared to the 2-class and 4-class solutions. In addition, the VLMR indicated that the 3-class solution was also a fit and more parsimonious than the 4-class solution. As for substantive considerations, the three-class solution was consistent with our hypothesized trajectories of cortisol, including a normative or moderate cortisol profile, an elevated or high profile, and a low cortisol profile. Inspection of the classes in the 4-class solution revealed a very small class that had a similar trajectory profile as the elevated class in the 3-class solution, thus suggesting minimal differences between the two classes. Taking both statistical and substantive considerations into account, we retained the 3-class model for analyses. Figure 1 shows the model implied basal cortisol trajectories for the three-class solution.

Inspection of the latent basis intercept and slope means show how the three profiles were identified (see Table 4). Examination of the intercept values revealed that the children in the moderate class had initial cortisol levels that fell in the middle of the two other groups. Conversely, children in the elevated class evidenced the highest levels of initial basal cortisol levels in comparison to the other two groups; and lastly, the low cortisol class had the lowest initial levels of basal cortisol. We ran Wald statistical tests (Engle, 1983) to test the equality of these means in MPlus. We specifically compared each mean to another resulting in three separate tests. Results indicated that the means were significantly different from one another (Wald Statistics ranged from 124.13 to 284.12, $ps < .01$) suggesting that the three profiles exhibited significantly different levels of basal cortisol at the first measurement occasion of the study. Additionally, the results revealed that the latent slope means across the different classes were not significant, suggesting basal cortisol levels remained relatively stable across the ages of 2 and 4 across all three classes (Table 4). Wald tests on these means suggested that on average, there were not significant differences in the slopes across cortisol profiles across time (Wald Statistics ranged from 0.02 to 3.01, ps ranged from .08 to .88).

We performed the next steps of our analysis with the aim to delineate whether family and socioeconomic risk constructs differentially predicted class membership and further, to examine average level of cognitive functioning within classes. To accomplish this, we performed logistic regressions, including covariates in our model analysis as predictors of class membership and then estimated the mean of children's cognitive functioning within each cortisol trajectory class. Inclusion of covariates in mixture models may lead to changes in class definition; however, our model results revealed that it did not significantly change trajectory profiles or membership. A summary of these findings are presented in Table 4. Logistic regression analysis requires setting one class as the comparison group. In the first analysis, we set the moderate cortisol trajectory as the comparison class. In the second comparison, we set the elevated profile as the comparison class in order to compare it with the low cortisol group. The results of the logistic regressions suggested two general patterns. First, family instability was associated with membership within the elevated and low cortisol classes compared with the moderate cortisol group. Specifically, the regression coefficient suggests that for every one-unit increase in family instability, the log odds of being in the low and elevated cortisol groups versus the moderate cortisol group increases by .37 and .43 respectively. Thus, early exposure to disruptive and unstable caregiving conditions may be a particularly potent predictor of both high and low resting levels of basal cortisol functioning during the first four years of childhood. Second, exposure to maternal emotional unavailability was primarily associated with membership in the elevated class as compared with the low and moderate basal cortisol classes. In particular, the logistic regression coefficients suggest that for every one-unit increase in maternal emotional unavailability, the log odds of being in the elevated cortisol group versus the moderate group increases by .37. Furthermore, for every one-unit increase in maternal emotional unavailability, the log odds of being in the low cortisol group versus the elevated cortisol group decreases by -.51. Exposure to greater levels of emotionally unsupportive and withdrawn parenting was a unique predictor of elevated cortisol, which remained stable between the ages of 2 and 4. None of the other family and socioeconomic risk predictors differentiated basal cortisol profile membership.

Finally, we tested for mean differences in children's cognitive functioning according to basal cortisol profile (Table 4). Wald tests revealed that the means were significantly different from one another (Wald Statistics ranged from 15.23 to 1755.62, $ps < .01$). In particular, membership in the elevated profile was associated with the lowest mean level of child cognitive ability in comparison to the mean levels of cognitive functioning in the moderate and low cortisol profiles. The moderate cortisol class was associated with the highest mean level of cognitive functioning at age 4. Given the large range of IQ scores in the current sample, we re-ran analyses excluding 5% of the IQ scores on the tail ends of the distribution to test for the potential influence of these scores on our results. Findings were similar to the original analysis, thus the full sample was retained.

Discussion

The present study demonstrates the potential for family risk conceptualizations to enhance our understanding of how differences in children's experiences of family adversity, within poverty conditions, may shape individual differences in stress-response systems and the

implications this might have for children's cognitive development. Our findings point to the utility of pattern-based approaches for cataloging heterogeneity in basal cortisol activity over time in high-risk samples of young children. Using growth mixture modeling analysis, we identified three patterns (elevated, moderate, and low) of basal HPA activity among the children in the study, who were experiencing heightened levels of socioeconomic distress and different types of family adversity. Demonstrating how allostatic processes may operate within family risk contexts, children's basal cortisol patterns were uniquely differentiated from each other by specific family risk factors measured at age 2, and the cortisol levels across all the groups remained relatively stable across the following two years of the study. Finally, our results revealed that both the elevated and lower basal cortisol patterns were associated with lower cognitive functioning when children were 4 years-old.

First, as early as age 2, children displayed categorically different physiological "set points" of basal cortisol activity. Inspection of the slope means of the classes revealed no statistically significant changes in linear trajectories in basal cortisol levels across the classes. Therefore, children's basal cortisol levels within each group remained relatively stable across the years of the study, which converges with recent applications of experiential canalization theories to developmental research of self-regulation among children living in poverty as well as with concepts of biological embedding (Blair & Raver, 2012; Gotlieb, 1991). In particular, developmental scientists have increasingly proposed that immediate caregiving environments shape or canalize physiological "set points" of stress response systems to maximize organismic functioning in specific anticipated conditions (Blair & Raver, 2012). One interpretation of our finding of stability in children's basal cortisol trajectories is that early exposure and ongoing experiences of specific types of family risk may eventuate in the formation of specific phenotypes of basal HPA activity that are meant to promote adaptive functioning within particular caregiving environments.

Stability in basal cortisol patterns across all three classes may also reflect inherited genetic differences among children in the sample. This is certainly plausible; however, family risk frameworks propose that different allostatic adaptations in HPA axis system are heavily influenced by contextual factors and may take different forms if contextual demands change (Repetti et al., 2011; Susman, 2006) and furthermore, genes are understood to operate differently across environments. Similarly, evidence from studies of therapeutic interventions with foster care children has documented the potential of family-based programs, aimed at fostering positive caregiving experiences, to influence subsequent changes in children's adrenocortical activity (e.g., Fisher et al., 2007). Our findings also provide evidence to suggest that early caregiving experiences may play a unique and critical role in shaping individual differences in children's HPA activity within high-risk, low-income contexts. Future research is needed to elucidate the developmental timing of these processes in addition to examining the longitudinal stability of basal cortisol patterns among low-income children exposed to different constellations of family risk processes.

Family instability and maternal emotional unavailability were identified as two potent proximal risk processes that meaningfully differentiated trajectories of basal cortisol among young children living in poverty. With respect to family instability, greater levels of unpredictability in the family environment such as, residential, caregiver, and family

structure changes predicted both the elevated and the low basal cortisol trajectories. These findings are consistent with a large body of research that has indicated that the HPA axis is highly sensitive to perturbations and disruptions in the family context, and that particular adaptations in children's stress-response activity may serve to facilitate coping strategies that match the demands of the characteristics of an unpredictable family environment (Blair et al., 2011b; Chen, Cohen, & Miller, 2010). It is noteworthy that our findings suggest that children's experiences of family instability may eventuate in two different phenotypic patterns of basal cortisol. Given inconsistencies in the literature regarding the direction of associations between family instability and cortisol, with studies either finding heightened (e.g., Chen et al., 2010) or dampened (Badanes et al., 2011; Fernald, Burke, & Gunnar, 2008; Zalewski et al., 2012) levels of functioning, the results from the current study indicate that both may be plausible when considered within a pattern-based approach. This begs the question as to why this may be so.

With respect to the identification of a low basal cortisol pattern, it has been primarily hypothesized that early experiences of severe trauma (e.g., sexual abuse) or prolonged periods of non-traumatic stress provoke frequent elevations in cortisol, which in turn can result in the down-regulation of different components of the HPA axis in some individuals (e.g., Gunnar & Vazquez, 2001). Although the exact neurobiological mechanisms through which this occurs are complex and likely multiply determined, it has been generally proposed that low cortisol is preceded by a period of elevated cortisol (e.g., Gunnar & Vazquez, 2001; Susman, 2006). However, recent studies reporting low cortisol among non-maltreated children, with no documented trauma histories, have challenged the assumption that low cortisol is predominately preceded by periods of heightened activity in the HPA axis (e.g., Badanes et al., 2011). The lack of multi-wave longitudinal developmental research examining the influence of multiple aspects of family life adversity on basal cortisol patterns has limited interpretations of when and why lower basal cortisol activity might develop in early childhood. Therefore, the current study represents one of only a few developmental studies that have detected both types of basal cortisol patterns among same-age and socioeconomically similar non-maltreated child participants. In comparing our findings with studies that have documented a low-cortisol pattern among low-income, non-maltreated children (e.g., Badanes et al., 2011; Zalewski et al., 2012), we speculate that stable levels of low basal cortisol might represent a distinct allostatic adaptation of the HPA axis to an unstable and unpredictable family environment, which is characterized by lower steady-state HPA activity that serves to protect against the excessive expenditure of metabolic resources in the face of recurrent stress and chaos. This conclusion aligns with family risk and experiential canalization models that propose that specific aspects of the early caregiving environment are operative in shaping HPA axis activity in a direction that maximizes functioning within a specific expected environment (e.g., Blair & Raver, 2012).

Family instability and maternal emotional unavailability jointly predicted an elevated pattern of cortisol in young children, with exposure to unsupportive caregiving primarily differentiating this pattern from the low basal cortisol pattern. Within the context of poverty and family instability, exposure to emotionally unavailable caregiving practices might be an especially potent predictor of higher basal cortisol activity, representing one unique developmental pathway underlying the emergence of stable elevations in basal cortisol in

low-income children. The external validity of the present finding is supported by an extensive body of research from both human and nonhuman models that has shown that insensitive and unresponsive caregiving practices seem to play a unique and critical role in the up-regulation of activity within the HPA axis. In particular, numerous rodent studies have consistently shown that less frequent licking and grooming behaviors of adult dams towards their pups is associated with the development of hypercortisolism in their offspring (e.g., Meaney, 2001, for review). In human studies, higher cortisol levels have been detected among children whose mothers intentionally withdraw attention from their child's affective cues as a means of controlling child behavior (Bugental et al., 2003). In addition, harsh and insensitive parenting practices often emerge within chaotic family environments (Matheny, Wachs, Ludwig, & Phillips, 1995). As such, it was not surprising that we observed modest bivariate associations between maternal emotional unavailability and family instability. It seems plausible that the combination of repeated changes and disruptions in the family system and lack of primary caregiver emotional support might engender a hypervigilant coping style, which then contributes to the emergence of a higher basal cortisol pattern among children within these contexts. These interpretations are speculative in nature and future research is needed to examine transactional processes among harsh and insensitive parenting practices, chaotic family environments, and HPA axis functioning across early child development. For example, future studies should consider including psychological measures of children's perceptions of threat as this may further understanding of how children's subjective experiences of these family risk environments might contribute to the development of stably elevated or lowered basal cortisol activity.

Contrary to expectations, interpartner violence was not a unique predictor of children's basal cortisol trajectories. Jaffe, Wolfe, & Wilson (1990) have proposed that the "aftermath" of domestic violence, such as relocation and parental stress, often serves as the primary mechanism through which interpartner aggression impacts children's functioning. Similarly, McCloskey, Figueredo, and Koss (1995) have also suggested that family dysfunction, which is either a byproduct of or a catalyst for interpartner aggression, might have a more robust effect on children's allostatic systems than the independent contribution of witnessing violence on its own. In support of this assumption, we observed moderate correlations between family instability and interpartner violence.

Severity of economic hardship was not a unique predictor of children's basal cortisol trajectories, contrasting with investigations that have documented the unique influence of socioeconomic status in shaping children's cortisol trajectories (e.g., Evans & English, 2002; Chen et al., 2010). At the bivariate level, earned income was moderately correlated with cortisol at ages 2 and 3, maternal emotional unavailability, family instability, and child cognitive functioning at age 4. One interpretation of this finding is that the effects of poverty may be transmitted through its impact on the social and emotional aspects of the family environment, which may explain the heterogeneity in developmental outcomes that is often observed in studies of low-income children. One caveat to this interpretation is that since we oversampled for families experiencing extreme levels of socioeconomic adversity, we may have lacked the statistical power to adequately test the influence of economic hardship on the child outcomes under investigation. Therefore, future studies should sample a broader range of socioeconomic backgrounds in order to fully test these assumptions.

Finally, our findings suggest different patterns of children's adrenocortical functioning may have important developmental implications for cognitive outcomes in early childhood. In particular, stable elevations and lower levels of resting basal cortisol activity in children was associated with lower cognitive abilities at age 4. Consistent with research demonstrating associations between elevations in cortisol and lower cognitive abilities (e.g., Blair et al., 2011a), our study found that the elevated pattern of basal cortisol was associated with the lowest mean level of cognitive functioning in children at age 4. Clinical and nonhuman studies have documented links between elevations in glucocorticoids and deficits in multiple areas of cognitive functioning (e.g., attention, spatial working memory) (e.g., Erickson, Drevets, & Schulkin, 2003). One explanation for this association is that the high rate of secretion of glucocorticoids by the adrenal glands may have neurotoxic effects on neuroanatomical structures (e.g., prefrontal cortex, hippocampus) that are understood to support cognitive functioning, and are highly plastic during early child development.

Testifying to the potential utility of pattern-based approaches, we found that the low basal cortisol trajectory was also associated with significantly lower levels of cognitive functioning in children in comparison to the moderate basal cortisol pattern. Only a few studies have documented associations between low cortisol activity and cognitive deficits in children (e.g., Gunnar & Vazquez, 2001). Research with adults has demonstrated a link between low basal cortisol and poorer cognitive performance (e.g., Erickson et al., 2003). For example, Fehm-Wolfsdorf, Reutter, Zenz, Born, Lorenz, (1993) found that cognitive performance was superior when participants were assessed in the morning, when their cortisol levels were higher, as opposed to the evening when cortisol their levels were lower. Gunnar and Vazquez (2001) have proposed that failure to produce a peak in morning cortisol might alter a child's capacity to maintain high engagement with the environment during the day. Although the low cortisol pattern among children in our study might not reflect hypocortisolism per se, the results suggest that children with this cortisol profile may possess significantly lower cognitive abilities relative to those with moderate levels of basal cortisol. One tentative hypothesis might be that children who display lower levels of basal HPA activity might not release adequate levels of cortisol that are essential for marshaling sufficient metabolic resources for optimal learning and memory consolidation to occur.

To our knowledge, this is the first study to document substantive differences in cognitive functioning across children displaying differentiated patterns of basal cortisol activity. These findings suggest pattern-based approaches may help to detect both high and low cortisol levels that are "risk" biomarkers for maladaptive cognitive outcomes. Although this interpretation is attenuated by the correlational nature of the study, one potential advantage of utilizing a GMM approach is that it may offer more precision in differentiating cortisol groups since heterogeneity is examined as it naturally occurs in the data, and individuals are classified into groups based on individual response patterns. However, experimental research is needed to validate the potential prognostic validity of this approach for delineating pathways of risk. In sum, these findings offer preliminary support for the family risk model proposition that early context-dependent adaptations within the HPA axis in response to family stress may confer a cascade of risk processes, such as disturbances in early cognitive functioning, which in turn might set the stage for early learning and academic difficulties as children transition into formal educational settings.

Despite these novel insights, the current study is not without its limitations. First, cortisol groups are characterized in relation to one another, and therefore, we cannot make interpretations with respect to whether the values in the high or low cortisol groups represent absolute indicators of heightened or lower basal cortisol activity, which might be observed in the general population. This may pose some difficulties in the interpretation of the present study results. However, as noted by previous investigators (e.g., Bruce et al., 2009), it is often difficult to systematically compare absolute cortisol values across studies because of heterogeneity in sampling and assaying techniques used and investigator differences in the reporting of cortisol results (i.e. transformed versus raw values). Second, because HPA functioning was measured at the level of the adrenals, we are unable to make definitive conclusions regarding the impact of resting levels of cortisol on neuroanatomical structures thought to support cognitive functioning. Furthermore, we did not use neuroimaging to identify whether structural and processing differences in specific cortical regions implicated in cognitive processing are observed in conjunction with reduced abilities; nonetheless, this represents an important direction for future research. We also utilized a narrow assessment of global cognitive ability and it will be important for future work to include a broader range of cognitive assessments. Third, the focus of our study was to examine resting or typical patterns of cortisol among a community sample of low-income children and thus, future research should examine whether these findings generalize to other populations and age groups (i.e., adolescents and adults). Fourth, the sampling of mid-morning basal cortisol precludes examination of diurnal patterns of cortisol levels at different points across the day. Although morning cortisol levels demonstrate a higher consistency across days in comparison to those found in afternoon and evening measures (Kirschbaum et al., 1990), increasing the number of samples collected per day would strengthen conclusions about children's cortisol profile membership. Lastly, it will also be important for future research to measure additional allostatic systems and assess health-related behaviors, such as sleeping and eating patterns, in order to gain a more complex and nuanced understanding of the multiple biological, environmental, and behavioral factors that shape children's physiological functioning under different family risk contexts.

In spite of these limitations, this report represents a conceptual and empirical advance in increasing our understanding of family processes that shape differential patterns of children's adrenocortical functioning within risky family environments and its implications for early cognitive development. In particular, these findings contribute to accumulating evidence that suggests emotionally unavailable and insensitive parenting practices and family unpredictability may be highly operative in differentiating patterns of children's adrenocortical functioning within low-income contexts, with subsequent negative implications for early cognitive functioning and academic readiness (e.g., Miller & Chen, 2013). Attachment-based interventions (e.g., Cicchetti, Rogosch, Toth, & Sturge-Apple, 2011) have been successful in normalizing children's basal cortisol levels, which underscores the potential for these types of interventions to prevent cognitive impairments. From a public health perspective, the study findings provide further empirical support for the need to increase investment in community-based interventions that are aimed to strengthen parent-child relationships and reduce family stress, as these might prove to have important short- and long-term benefits for children's physiological and cognitive functioning

Acknowledgments

This research was supported by grant R01 MH071256 from the National Institute of Mental Health awarded to Patrick T. Davies and Dante Cicchetti. The authors are grateful to the mothers and children who participated in this project.

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Figure 1.

Plot comparisons of estimated means and trajectories of three different cortisol profiles from latent basis model with three-class solution. Negative cortisol values reflect the impact of controlling for wake-up time in analyses. Groups are identified in the plot key.

Table 1

Descriptive Statistics for Primary Variables in Analysis

	<i>N</i>	<i>M</i>	<i>SD</i>	Range
Cortisol µg/dl, 2 years	180	0.24	0.17	.04 – .84
Cortisol µg/dl, 3 years	162	0.23	0.14	.05 – .68
Cortisol µg/dl, 4 years	172	0.24	0.14	.04 – .81
Wake-up Time AM, 2 years	199	7:37	46 min	4:45 – 9:53
Wake-up Time AM, 3 years	165	7:42	39 min	6:00 – 9:30
Wake-up Time AM, 4 years	173	7:36	51 min	4:15 – 0:15
Earned Income	200	1.56	1.60	0.00 – 4.52
Hollingshead Score	200	23.47	10.51	8.0 – 58.00
Family Instability	192	5.08	4.75	0.00 – 29.00
Interpartner Violence	184	4.89	3.23	0.00 – 12.00
Maternal Emotional Unavailability	174	4.09	1.61	1.00 – 8.00
Cognitive Functioning	169	90.65	16.39	50.00 – 130.00

Note. For child wake-up times, AM= ante meridiem. Min.= minutes.

Table 2

Correlations Among Primary Variables in Analyses

Variable	1	2	3	4	5	6	7	8	9
1. Cort, age 2	--								
2. Cort, age 3	.28**	--							
3. Cort, age 4	.35**	.35**	--						
4. Earned Income	-.26**	-.16*	-.06	--					
5. Hollingshead Score	-.22**	-.09	.03	.70**	--				
6. Family Instability	.21**	.04	.10	-.36**	.33**	--			
7. Mat. Emot. Unavail.	.32**	.21**	.27**	-.25**	-.28**	.16*	--		
8. IPV	.05	.06	-.03	-.07	-.01	.19*	-.05	--	
9. Child Cog. Funct.	-.24**	-.07	-.23**	.29**	.29**	-.22*	-.34**	.03	--

Note. Cort = cortisol; Mat. Emot. Unavail.= maternal emotional unavailability; IPV = interpartner violence; Child Cog. Funct. = Child Cognitive Functioning.

* $p < 0.05$.** $p < 0.01$.

Table 3
Fit Statistics for Growth Mixture Models With Freely Estimated Means and Pattern Loadings

	1 Class	2 Class	3 Class	4 Class
<u>Sample Size</u>				
Nc=1	199	93	80	56
Nc=2	---	86	58	6
Nc=3	---	---	61	79
Nc=4	---	---	---	58
<u>Fit statistics</u>				
df	18	25	32	39
BIC	663.87	486.22	482.05	496.06
Entropy	1	0.74	0.74	0.68
VLMR LRT	---	228.28 *	30.88 *	22.06

Note. Sample size is based upon estimated counts for latent classes based upon posterior probabilities; BIC = Bayesian Information Criterion; VLMR LRT = Vuong-Lo-Mendall-Rubin Likelihood Ratio Test of additional class.

* *p* .05.

Table 4

Parameter Estimates for 3-class Model

	Low Cortisol Class	Elevated Cortisol Class	Moderate Cortisol Class
Sample size	81	58	60
Avg. probability of class membership	.83	.90	.82
<u>Growth Model Results</u>			
Latent variable means by Cortisol Class			
Intercept mean, g0	−0.05 ^a	0.20 ^{**b}	0.02 ^c
Slope mean, g1	−0.03 ^d	−0.07 ^d	−0.03 ^d
<u>Logistic Regression Coefficients Predicting Cortisol Class Membership</u>			
<i>Moderate Cortisol Trajectory as Reference Class</i>			
Maternal Emotional Unavailability	0.13	0.37 [*]	Reference Class
Child Gender	−1.08	−0.95	Reference Class
Hollingshead Score	−0.05	−0.04	Reference Class
Interpartner Violence	0.01	0.08	Reference Class
Family Instability	0.37 [*]	0.43 [*]	Reference Class
Earned Income	−0.23	−0.47	Reference Class
<i>Elevated Trajectory as Reference Class</i>			
Maternal Emotional Unavailability	−0.51 [*]	Reference Class	
Child Gender	0.14	Reference Class	
Hollingshead Score	−0.01	Reference Class	
Interpartner Violence	−0.00	Reference Class	
Family Instability	0.03	Reference Class	
Earned Income	−0.24	Reference Class	
<u>Wave 3 Child IQ Means by Cortisol Class</u>			
Mean	91.36 ^{e**}	80.82 ^{f**}	102.06 ^{g**}
Standard Error	2.38	2.08	4.33

Note. Significant differences between groups were examined by the Wald test of significance at $p < .05$. Values with the same superscripts denote non-significant between-group differences in parameter estimates, whereas values with different superscripts denote significant differences. Regression coefficients are unstandardized beta weights.

*
 $p < .05$

**
 $p < .01$.