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Child Care and Cortisol Across Early Childhood: Context Matters

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A considerable body of literature suggests that children's child-care experiences may impact adrenocortical functioning in early childhood. Yet emerging findings also suggest that the magnitude and sometimes the direction of child-care effects on development may be markedly different for children from higher risk contexts. Using data from a large population-based sample of families from predominantly low-income backgrounds in rural communities, we tested the degree to which links between children's child-care experiences (at 7–36 months) and their subsequent cortisol levels (at 48 months) were moderated by their level of cumulative environmental risk. Our results provided evidence of a crossover interaction between cumulative risk and child-care quantity. For children from low-risk contexts, greater weekly hours in child care were predictive of higher cortisol levels. In contrast, for children facing several cumulative risk factors, greater hours in child care per week were predictive of lower cortisol levels. These effects were robust after adjusting for several controls, including children's cortisol levels in early infancy. Child-care quality and type were not predictive of children's cortisol levels, and neither mitigated the conditional effect of child-care quantity on cortisol. These findings suggest that links between child care and children's development may differ as a function of children's broader ecologies.

Keywords: child care, cortisol, poverty, cumulative risk

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The findings from multiple large-scale longitudinal studies of children in context (Belsky et al., 2007; Loeb, Bridges, Bassok, Fuller, & Rumberger 2007), as well as those from early-childhood interventions (Barnett & Masses, 2007; Schweinhart et al., 2005), indicate that high-quality child-care experiences in early childhood can positively impact children's cognitive development. However, there has also been some evidence of iatrogenic effects, such that children spending more time in child care in early childhood also

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tend to show somewhat more problematic social outcomes (e.g., aggression; Belsky et al., 2007; NICHD Early Child Care Research Network [NICHD ECCRN], 2003).

Such findings have led some to inquire about the possible mechanisms through which children's early child-care experiences "get under the skin" to affect their development. Spurred by the seminal work of Gunnar and colleagues (e.g., Dettling, Gunnar, & Donzella, 1999; Tout, de Haan, Campbell, & Gunnar, 1998; Watamura, Sebanc, & Gunnar, 2002), particular attention has been paid to the effects of child care on adrenocortical functioning, as indexed by cortisol—the primary end-product of the hypothalamic–pituitary–adrenal (HPA) axis. Through normative daily diurnal rhythms (Sapolsky, Romero, & Munck, 2000), as well as through acute responses to psychological stress (Kirschbaum & Hellhammer, 1989), the HPA axis supports the organism's ability to adapt to environmental demands (McEwen, 2000; Sapolsky et al., 2000).

A convergent literature finds that children's child-care experiences may affect HPA-axis functioning. Across multiple studies and samples, there is rather consistent evidence that compared with days in which they do not attend care and contrary to normative diurnal rhythms, children tend to show cortisol increases across the day on days in which they attend child care (see Geoffroy, Côté, Parent, & Séguin, 2006; Phillips, Fox, & Gunnar, 2011; Vermeer & van IJzendoorn, 2006). Some work has shown that this may be particularly the case in toddlerhood and the beginning of the early childhood years (Vermeer & van IJzendoorn, 2006; Watamura, Donzella, Alwin, & Gunnar, 2003) and for those attending lower quality care (Dettling, Parker, Lane, Sebanc, & Gunnar, 2000; Sims, Guilfoyle, & Parry, 2006). Beyond effects on diurnal rhythms, other work has shown long-term associations between spending greater proportions of time in center-based care in infancy and early childhood and children's subsequent cortisol awakening response in adolescence (Roisman et al., 2009).

To date, it has been largely unclear whether the effects of child care on children's cortisol levels have any long-term secondary impacts on children's health or behavior. However, recent evidence suggests that the atypical diurnal cortisol profiles stemming from child-care attendance may have negative secondary effects on children's immune-system functioning (Watamura, Coe, Laudenslager, & Robertson, 2010). Similarly, although such specific indirect effects are rarely considered with respect to children's cognitive and social development, increasing work suggests that normative variation in children's basal cortisol levels—that is, levels that are *not* reflective of intentionally evoked, particularly high cortisol levels—are predictive of children's cognitive and social development in early childhood. For instance, prior work with the same low-income sample considered in the present study has shown that higher levels of basal cortisol in infancy and toddlerhood (earlier time points than those examined here), are predictive of less effective executive functioning at 3 years of age (Blair, Granger, et al., 2011). In other samples, similar findings are evident for substantively related aspects of children's self-regulation (Watamura, Donzella, Kertes, & Gunnar, 2004; however, see Davis, Bruce, & Gunnar, 2002). Meta-analytic work has also suggested that higher levels of basal cortisol are associated with elevated levels of externalizing problems in early childhood (Alink et al., 2008). Collectively, these findings suggest that normative variation in children's basal cortisol levels may be positively associated with less optimal outcomes across multiple aspects of children's development in early childhood.

Low-Income Contexts and Stress Physiology

Notably, the majority of the literature considering the effects of child care on HPA-axis functioning has been conducted with rather small, homogeneous samples of children from middle- to high-socioeconomic-status families, attending predominantly moderate- to high-quality care (however, see Rappolt-Schlichtmann et al., 2009; Roisman et al., 2009). There is growing reason to suspect that child-care effects for children from lower income families may differ markedly from those of their more affluent peers (McCartney & Berry, 2009).

Children growing up in low-income ecologies often face a host of risk factors that are predictive of less optimal development (Dearing, Berry, & Zaslow, 2006; Evans, 2004)—including HPA-axis functioning (Blair, Granger, et al., 2011, Blair, Raver, et al., 2011; Evans & English, 2002). At the neighborhood level, children in families struggling with financial adversity are more apt to experience social and physical stressors, such as deteriorated buildings, disorganization, crime, and inadequate access to services (Ellen, Mijanovich, & Dillman, 2001; Israel et al., 2006). A growing literature suggests that such neighborhood-level risk factors are associated with HPA-axis functioning. For example, in adults, high levels of perceived and observed neighborhood stressors or low levels of neighborhood-level social support have been linked to blunted diurnal cortisol profiles, such that those with higher levels of objective or perceived stress or less social support tend to maintain higher cortisol level over the course of the day, rather than showing normative diurnal declines (Karb, Elliott, Dowd, & Morenoff, 2012).

Children from low-income contexts are also more likely to experience household environments that are more disorganized and chaotic—represented, for instance, by greater levels of noise, crowding, and unpredictability (Evans, 2004; Vernon-Feagans, Garrett-Peters, DeMarco, & Bratsch-Hines, 2012). The inability to effectively predict and control experiential inputs is a key factor driving HPA-axis stress response (Dickerson & Kemeny, 2004), and increasing work suggests that such unstable and chaotic environments are predictive of comparatively higher levels of resting cortisol (Blair, Raver, et al., 2011), as well as indicators of autonomic nervous system functioning, such as epinephrine and blood pressure (Evans & English, 2002).

Stressors inside and outside the home can also impact the quality of parent–child interactions. On average, children from families facing economic adversity are more likely to experience less sensitive caregiving at home (Dearing et al., 2006; Linver, Brooks-Gunn, & Kohen, 2002). Similarly, prior work with the sample considered in the present study has shown that such lower quality parenting behaviors are, in turn, associated with higher cortisol levels in infancy and toddlerhood (Blair, Granger, et al., 2011). Indeed, this work builds on a notable literature indicating that stress physiology in young children is highly regulated by social experience (Gunnar & Donzella, 2002).

Child Care, Environmental Risk, and Stress Physiology

Thus, taken together, there is a growing indication that risk factors across children's ecologies—ranging between proximal experiences with their parents, more distal contextual indicators of chaos and neighborhood quality, to broader markers of risk, such as parental education and family income—have been linked with adrenocortical functioning. Although some evidence suggests that children's child-care experiences may also impact HPA-axis activity, these findings have been based largely on studies of children growing up in lower risk contexts. To date, it is largely unclear whether the effects of children's child-care experiences on HPA-axis functioning may differ for children from high- as opposed to low-risk contexts.

At least two contrasting hypotheses can be proposed. The first is that the stressors outside child care, which tend to be more prevalent in families struggling with material hardship, may metaphorically "prime the pump," such that the normative child-care effects on stress physiology are particularly pronounced for these children. For instance, some work has indicated that when children are first entering child care, the association between child-care attendance and cortisol increases over the course of the day is particularly pronounced for children with outside risks, such as insecure maternal attachment relationships (Ahnert, Gunnar, Lamb, & Barthel, 2004).

Alternatively, child care—especially, when quality is high—may serve as a respite for those facing stressful environments in their broader ecologies. That is, attending child care may buffer young children from high-risk contexts against the deleterious effects of those environments. Indeed, one might predict a dose–response relation, such that greater hours in child care are associated with the largest benefits for children from high-risk contexts.

Given the fact that lower income/higher risk samples have rarely been employed in studies of child care and children's stress physiology, questions regarding such potential interactive processes between children's experiences inside and outside child care remain largely unaddressed. The limited available work considering links between child care and stress physiology with low-income samples, however, provides some indication that—unlike the well-replicated finding with comparatively more affluent samples—low-income children attending child care tend to show a normative diurnal cortisol decline over the course of the child-care day (Rappolt-Schlichtmann et al., 2009).

Further, at the behavioral level, there is growing evidence that child care may have a buffering effect against the negative impacts of high-risk environments on development. For example, the beneficial effects of child-care exposure on early academic achievement have been found to be especially pronounced for children from low-income contexts (Loeb et al., 2007). This is particularly the case when child-care quality is high (Dearing, McCartney, & Taylor, 2009). Further, in contrast to evidence suggesting that more extensive hours in child care may be "detrimentally" associated with aspects of children's social development (Belsky et al., 2007; NICHD ECCRN, 2003), other work suggests that such effects may be inversed for children from low-income/high-risk contexts. For young children raised in higher risk environments, attending child care may be *beneficial* for their social development (Côté et al., 2007; Côté, Borge, Geoffroy, Rutter, & Tremblay, 2008; Crosby, Dowsett, Gennetian, & Huston, 2010; Votruba-Drzal, Coley, & Chase-Lansdale, 2004). Although some findings suggest that this may be contingent upon higher quality care (e.g., Votruba-Drzal et al., 2004) and evident for center-based care (Crosby et al., 2010), others have shown similar effects stemming from simply attending nonmaternal care more broadly (Côté et al., 2007, 2008). Interestingly, child-care effects may be represented by a crossover interaction, such that the *direction* of child-care effects on behavioral outcomes differs at the low and high ends of the environmental-risk distribution. It is thus far unclear whether such interactive effects may extend to individual differences in HPA-axis functioning.

The Present Study

The aim of our proposed study was to consider the degree to which links between children's child-care experiences and their subsequent cortisol levels may vary as a function of broader environmental risk. Specifically, informed by prior work with these data (see Vernon-Feagans, Cox, & the Family Life Project Key Investigators, in press) and growing evidence that understanding the confluence of risk factors associated with low-income contexts may be more informative than considering any single risk factor alone (Burchinal, Roberts, Hooper, & Zeisel, 2000; Evans, 2004), we adopted a cumulative risk approach to address the following questions: (a) Is the relation between number of weekly hours in child care and children's cortisol levels at 48 months (controlling for 7-month levels) moderated by their broader level of cumulative risk; if so, is the interaction consistent with the potential buffering role of child care for those experiencing heightened environmental risk; and (b) Are such effects contingent upon child-care quality and type?

We hypothesized that greater hours in child care would be associated with higher levels of cortisol at age 48 months for those facing low levels of environmental risk. In contrast, we hypothesized that this relation would be comparatively more negative for those experiencing higher levels of risk—possibly to the extent to which the conditional relation reverses direction, such that greater hours in child-care exposure are predictive of subsequently *lower* cortisol levels. In addition, we hypothesized that the positive conditional relation between child-care hours and higher cortisol levels for those facing low levels of cumulative risk would be less pronounced (and the negative relation for those in higher levels of cumulative risk would be more pronounced) for those in higher quality care. We advanced no specific hypotheses with regard to child-care type, given that center-based care has been linked with potential supports (e.g., more predictable schedules, better trained caregivers, and richer learning environments; Fuller, Kagan, Loeb, & Chang, 2004), as well as potential stressors (e.g., large peer groups; Morrissey, 2010).

Method

Participants

The Family Life Project (FLP) was designed to study young children and their families in two of the four major geographical areas of the United States with high poverty rates (Dill, 1999)—eastern North Carolina (NC) and central Pennsylvania (PA). Spe-

cifically, 1,292 children whose families resided in one of the six counties at the time of the child's birth were sampled. Low-income families in both states and African American families in NC were oversampled. A comprehensive description of the sampling plan is provided by Vernon-Feagans et al. (in press). The present analytic sample comprises the 1,235 children who provided child-care data during at least one of the four points in which information about child care was collected between the ages of 7 and 36 months. Those excluded from the analytic sample did not differ, with respect to race or family income or primary-caregiver education level at the 7-months assessment.

Procedures

The present study focused on data collected across infancy and early childhood. Target children's primary caregivers (99.61% biological mothers) were interviewed regarding demographic and personal information when their children were approximately 2 months old (see Control Covariates section). The demographic and employment data used in the cumulative risk index as well as information about child-care were collected from primary caregivers during interviews when their children were 7, 15, 24, and 36 months old. When target children were approximately 7, 24, and 36 months old, independent observers rated neighborhood noise and safety, as well as the number of rooms within each household during home visits. Observational measures of parenting behavior during parent–child play task were obtained when target children were approximately 7, 15, 24, and 36 months old. When children were in nonparental care for at least 10 hr per week, interactions between the child-care caregiver and child were rated for caregiver quality by independent raters. Children's cortisol levels at the ages of 7 and 48 months were assayed using saliva samples collected during home visits.

Measures

Salivary cortisol. Saliva samples were collected after the data collectors had been in the home for at least 15–20 min interviewing the primary caregiver and prior to their conducting a number of assessments with children. Following Granger et al. (2007), unstimulated whole saliva was collected using either cotton or hydrocellulose absorbent material and expressing sample into 2-ml cryogenic storage vials using a needleless syringe (cotton) or by centrifugation (hydrocellulose). Prior work has indicated no differences in cortisol concentrations associated with the two collection techniques (Granger et al., 2007). Samples were immediately frozen at −20° C, shipped to the laboratory packed in dry ice, and subsequently frozen at −80° C. Saliva samples were assayed for salivary cortisol using a highly sensitive enzyme immunoassay that was cleared for use by a U.S. Food and Drug Administration 510k submission as an in vitro diagnostic measure of adrenal function (Salimetrics, State College, PA). The test used 25 μl of saliva (for singlet determinations), had a range of sensitivity from .007 to 3.0 ug/dL, and had average intra-and interassay coefficients of variation of less than 10% and 15%, respectively. All samples were assayed in duplicate. The criterion for repeat testing was variation between duplicates greater than 20%, and the average of the duplicates was used in all analyses. We log-transformed children's cortisol values at each time point to reduce positive skew.

Child temperature and primary caregiver ratings of hours since sleeping/napping, average hours of nightly sleep, hours of sleep the prior night, use of over-the-counter or prescription medications, and overall child health were considered as possible influences on child cortisol levels. Fewer average hours of sleep per night ($r = –.08$, $p = .02$), fewer hours of sleep the prior night ($r = -.10$, $p = .004$), and fewer hours since waking from sleeping/napping ($r = –.15$, $p < .001$) were modestly associated with higher cortisol levels. The modal time of day for the saliva collections was 10:00 a.m. at both 7 and 48 months. There was, however, some variability in collections times (*SD*s = 2.88 and 2.91, respectively), and, as expected, time of day (24-hr clock) showed moderate negative associations with children's cortisol levels at both 7 and 48 months ($r = -.24$, $p < .001$; and $r = -.36$, $p < .001$, respectively). All sleep and time-of-day variables were, thus, included as control covariates in the regression models discussed below.

Child-care quantity. Children's primary caregivers provided the average hours per week that the child attended child care when the child was 7, 15, 24, and 36-months old. We operationalized child-care quantity as the average number of child-care hours per week, averaged across the four measurement periods occurring between the ages of 7 and 36-months. We divided child-care hours by 10 to reduce the decimal places in the tables; the figures are on the original metric.

Child-care quality. Traditional measures of child-care quality, such as the Early Childhood Environment Rating Scale (ECERS; Harms & Clifford, 1980) or the Observational Record of the Caregiving Environment (ORCE; NICHD ECCRN, 1996) were unavailable in these data. We, thus, used independent ratings of caregivers' behavior toward the child, as scored with Home Observation for Measurement of the Environment scale (HOME; Caldwell & Bradley, 1984), as a proxy for child-care quality. HOME scale observations were made when the child was 7, 15, 24, and 36 months old. The infant/toddler version of the measure was used at the 7- through 24-month observations. Informed by prior psychometric work (Caldwell & Bradley, 1984), we created a Caregiver Responsivity subscale, based on 10 items tapping caregiver responsiveness and affection toward the child.[1] We adopted a similar strategy to model a latent Caregiver Responsivity factor across the seven items in the preschool version of the measure. Across both versions of the HOME measure, we hypothesized that a single latent factor explained observed caregiver responsivity across the items within a given time point. As items were scored dichotomously, these models were fitted using a robust weighted least squares estimator (Mplus; Muthén & Muthén, 2009). Descriptions of the confirmatory factor analysis models are provided in the supplemental materials.

Child-care type. Child-care type was reported by the child-care caregiver and was designated as center- versus other-based care (including parental) using dummy-codes at 7, 15, 24, and 36 months old. We aggregated these data as the number of spells—of the four time points measured—in which the child was reported to be in center-based care.

Cumulative risk. Informed by extensive prior work with these data (see Vernon-Feagans et al., in press), we created a cumulative-risk composite comprising 8 variables—family income, maternal education, constant spouse/partner living in the

[1] We dropped one item (i.e., caregiver permits messy play) typically used in the Responsivity scale, as the confirmatory factor analysis models indicated that it was unassociated with the latent construct at each point in time.

home, hours of employment, occupational prestige, household density, neighborhood noise and safety, and positive parenting—that were measured at several points across infancy and early childhood. As described in detail in the supplemental materials, we created a continuous cumulative-risk index by reverse-scoring the positively framed variables, standardizing each risk measure, and averaging across the risks. Internal-consistency reliability across the items was reasonable ($\alpha = .82$). Higher scores reflect higher levels of cumulative risk.

Control Covariates

Informed by prior studies (e.g., NICHD Study of Early Childhood and Youth Development; NICHD ECCRN, 2003), we adopted a variety of child and family covariates, in an attempt to adjust for potential confounding factors that could bias our estimates. Described in detail in the supplemental materials, our control covariates include: infant gender (male = 1), race (African American = 1), temperament, mental development; primary caregiver reading ability, age at birth, AFDC receipt, Medicaid receipt, and research site (NC = 1); as well as aspects relevant to the measurement of cortisol, including time of saliva collection, time since nap/sleep, typical quality of sleep and prior-night sleep quality.

Missing Data

As indicated in prior work (Blair, Raver, et al., 2011b), there is little evidence of selective attrition in the sample through 58 months, and missing data for the main covariates of interest in the present study were fairly minimal. For instance, 96% of children had data with respect to child-care quantity during at least one of the four measurement periods. Among these 1,235 children, the percentage of children with missing data with regard to child-care quantity ranged between 3% and 9% across the four measurement periods. Of those who had a spell of child care (i.e., 10 or more hours), the percentage with missing data for estimated child-care quality was 19%, and the percentage for those reporting center-based versus non-center-based care ranged from 23% to 31% for those attending 10 or more hours in care. Across the measures in the cumulative risk index, missingness ranged from 7%–16% at 7 months assessment to 14%–19% at 48 months assessment. Approximately 28% of children included in the analytic sample were missing salivary cortisol data at the 48 months assessment. To adjust for potential biases due to missing data, we used the robust full-information maximum likelihood (FIML) estimator available in Mplus (Version 5.21; Muthén & Muthén, 2009). FIML estimation helps to adjust for biases due to missing data under the assumption that missingness is conditional on observed variables that are included in the model and, after adjusting for these variables, are not conditional on unobserved values of the variables with missing data (i.e., "missing at random"; Collins, Schafer, & Kam, 2001).

Data-Analytic Plan

In a taxonomy of models, we first regressed children's 48-month salivary cortisol levels on their (a) prior cortisol levels at age 7 months; (b) child-care quantity, quality, and type; (c) the cumulative risk index; and (d) a set of additional control covariates. To test our key questions of interest, we subsequently allowed the respective effects of child-care quantity, quality, and type to vary as function of cumulative risk level by introducing the respective two-way interaction terms into the model. Each interaction term was fitted in a separate model. To consider the degree to which the two-way interaction between child-care quality and cumulative risk may be mitigated by quality or type, we subsequently added these respective three-way interactions into the model (along with their constituent two-way interactions).

All variables were centered on their respective grand means. All models were fitted using the robust maximum likelihood estimator available in the Mplus statistical platform (Muthén & Muthén, 2009). Sampling weights were included in all models to adjust for the sampling plan. All comparisons of nested models were based on Satorra–Bentler (S-B; Satorra & Bentler, 1994) adjusted likelihood ratio tests. Per convention, we probed statistically significant interactions at 1 standard deviation below (i.e., low cumulative risk) and 1 standard deviation above (i.e., high cumulative risk) the cumulative risk mean using tests of model constraints.

Results

Preliminary Analyses

Across the first 36 months of life, children spent approximately 20.90 hr in child care per week, on average. Just over a third of the children (35%) were in 10 or fewer hours of care per week, on average, and approximately a third of the children (32%) were in 30 or more hours of child care per week. The average hours per week spent in child care tended to increase, on average, as children grew older, and there was moderate to strong rank-order stability in children's child-care hours over this period (rs = .35 $p < .001$ to .61 $p < .001$). Approximately 20% of the children attended at least two (of four) occasions in center-based care. As shown in Table 1, children spending more hours in child care per week and those spending a greater proportion of their time in center-based care tended to experience slightly lower quality care, on average ($r = -.11$; $p = .003$ and $r = -.29$; $p < .001$, respectively). Although there was some indication that lower quality care was correlated with higher levels of salivary cortisol at 7 months ($r = -.12$, $p = .002$), no such relation emerged with children's cortisol levels at the age of 48 months. Neither child-care hours nor type were correlated with children's cortisol levels at either 7 or 48 months. Rank-order stability in children's cortisol levels over time was rather modest ($r = .16$, $p < .001$).

On average, children with higher levels of cumulative risk tended to attend fewer hours in child care per week ($r = -.09$, $p = .001$), and when they did attend child care, they tended to experience lower quality care ($r = -.26$, $p < .001$). Notably, the former correlation was quite modest. Cumulative risk was not correlated with attending center-based care ($r = -.04$, $p = .19$). There was a weak positive correlation between number of cumulative risks and children's cortisol levels at both 7 ($r = .11$, $p < .001$) and 48 ($r = .11$, $p = .002$) months of age; higher levels of risk were rather weakly associated with higher cortisol levels.

Table 1
Zero-Order Correlation Between Salivary Cortisol, Cumulative Risk, Child Care Experiences, and Select Demographic Variables

Variable	1	2	3	4	5	6	7	8	9	Mean (*SD*)
1. Cortisol 48 months (log)	—									−2.11 (0.72)
2. Cortisol 6 months (log)	.16***	—								−1.84 (0.77)
3. Cumulative risk	.11**	.11***	—							0.00 (0.68)
4. Child care hours	.00	.01	−.09**	—						20.90 (16.52)
5. Child care quality	−.06	−.12**	−.26***	−.11**	—					0.00 (0.89)
6. Child care type	.03	.04	−.04	.47***	−.29***	—				.72 (1.17)
7. Target child temperament	−.07*	−.03	.02	−.03	.04	−.04	—			0.00 (0.07)
8. Target child–male	.02	.03	−.02	.01	−.05	−.02	−.05†	—		.50
9. Target child–African American	.15***	.17***	.46**	.29***	−.34***	.26***	−.05†	.01	—	.42
10. Target child–site	.10**	.11	.28**	.28***	−.34***	.27***	−.08**	−.07**	.61***	.60

† $p < .10$. * $p < .05$. ** $p < .10$. *** $p < .001$.

Child Care, Cumulative Risk, and Salivary Cortisol

As displayed in Column A of Table 2, there were no main-effect relations evident between any of the child-care variables and children's subsequent cortisol levels at 48 months. The main effect of cumulative risk also failed to reach statistical significance, after adjusting for the control covariates. As expected, children's prior levels of salivary cortisol at 7 months old were positively—albeit, modestly—associated with their 48-month cortisol levels ($B = 0.11$, $p < .001$). This relation

Table 2
*Fitted Regression Coefficients Testing the Degree to Which the Relations Between Children's Child-Care Experiences (7–36 Months) and Their Subsequent Cortisol Levels Are Moderated by Level of Cumulative Environmental Risk (*N = *1,235)*

	Model					
Variable	A	B	C	D	E	F
Substantive covariates						
Intercept	−2.15***	−2.15***	−2.15***	−2.15***	−2.16***	−2.16***
Cortisol (6 months)	0.11**	0.11**	0.11**	0.11**	0.11**	0.10**
Child-care hours	0.02	0.01	0.02	0.02	0.01	0.01
Child-care quality	0.01	0.00	0.01	0.02	0.02	0.01
Child-care type	−0.01	−0.01	0.00	−0.02	−0.01	−0.02
Cumulative risk	0.06	0.05	0.07	0.05	0.06	0.07
Risk × Child-Care Hours		−0.09**			−0.09	−0.10
Risk × Child-Care Quality			−0.03		0.00	
Risk × Child-Care Type				−0.05		0.03
Child-Care Hours × Child-Care Quality					−0.02	
Child-Care Quality × Child-Care Type						0.01
Risk × Hours × Quality					−0.01	
Risk × Hours × Type						−0.03
Control covariates						
Target child–male	0.00	−0.01	0.00	0.00	−0.01	−0.01
Target child–African American	0.15*	0.18*	0.14†	0.17*	0.18*	0.19*
Target child–temperament	−0.61	−0.60	−0.62	−0.60	−0.57	−0.58
Target child–Mental Development Index score	0.01†	0.01†	0.01†	0.01*	0.01†	0.01†
North Carolina site	−0.18**	−0.18**	−0.17**	−0.18**	−0.18**	−0.19**
Primary caregiver–reading	0.01	0.01	0.01	0.01	0.01	0.01
Primary caregiver age (Target child age = 2 months)	0.31	0.27	0.31	0.30	0.27	0.27
Primary caregiver age at first birth	−0.02*	−0.01†	−0.02*	−0.02*	−0.01*	−0.01†
Primary caregiver receiving AFDC	−0.05	−0.06	−0.05	−0.04	−0.06	−0.07
Primary caregiver receiving Medicaid	−0.09	−0.08	−0.09	−0.10	−0.08	−0.08
Time of day (6 months)	0.00	−0.01	0.00	0.00	−0.01	−0.01
Time of day (48 months)	−0.09***	−0.09***	−0.09***	−0.09***	−0.09***	−0.09***
Typical sleep	−0.01	0.00	−0.01	0.00	0.00	0.00
Sleep prior night	−0.01	−0.01	−0.01	−0.01	−0.01	−0.01
Last sleep/nap	−0.02***	−0.02***	−0.02***	−0.02***	−0.02***	−0.02***
R^2	.22	.24	.22	.23	.24	.24

Note. Models A–F test a taxonomy of main-effect, two- and three-way interactions between cumulative risk, and children's child-care experiences. AFDC = Aid to Families With Dependent Children.
† $p < .10$. * $p < .05$. ** $p < .10$. *** $p < .001$.

corresponded to a standardized regression coefficient of .12; a standard deviation difference in 7-month salivary cortisol level was associated with a .12 standard deviation cortisol difference at 48 months of age.

Subsequent models (Column B) indicated that the relation between child-care quantity and children's later cortisol levels was conditional on their level of cumulative risk. Specifically, there was evidence of a crossover interaction ($B = -0.09$, $p = .002$; $\Delta R^2 = .02$), such that the magnitude and direction of the association between child-care hours and children's 48-month cortisol levels was conditional on level of cumulative risk. As displayed in Figure 1, in the context of low environmental risk (i.e., 1 *SD* below the mean), greater hours in child care were associated with higher levels of salivary cortisol at 48 months ($B_{low\_risk} = 0.07$, S-B$\Delta\chi^2 = 12.48$, $\Delta df = 1$, $p < .001$). This conditional relation was evident, after adjusting for children's 7-month cortisol levels, child-care quality and type, and the other control covariates. This simple slope corresponded to a conditional standardized association of approximately .16.

In contrast, in the context of high levels of cumulative environmental risk—designated here as a standard deviation above the mean—the direction of the relation was inversed; greater hours in child care were predictive of lower cortisol levels at 48 months ($B_{high\_risk} = -0.05$, S-B$\Delta\chi^2 = 4.04$, $\Delta df = 1$, $p = .04$). This corresponded to standardized simple slope of –.12.

As shown in Columns C and D of Table 2, there was no evidence of similar interactions between child-care quality or type (respectively) with level of cumulative risk. As displayed in Columns E and F in Table 2, there was also no indication that the interaction between child-care hours and cumulative risk was conditional on child-care quality or type, respectively. In subsequent models (not shown), we tested a series of models in which we allowed the effects of child-care quantity, quality and type, cumulative risk, and the respective two-way interactions between these child-care variables and cumulative risk to vary as a function of gender and infant temperament and cortisol level at age 7 months, respectively. No two- or three-way interactions emerged at traditional or Bonferonni-adjusted levels of statistical significance.

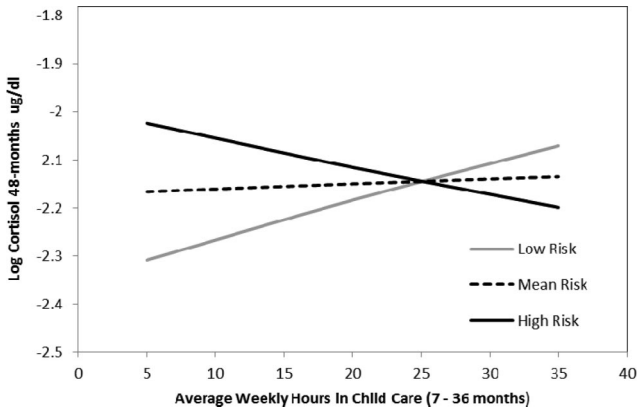

Figure 1. Estimated association between average weekly child-care hours (at ages 7–36 months) and children's cortisol levels at age 48 months, conditional on the level of cumulative risk. Scale of *y* axis = 1 standard deviation.

Discussion

A convergent literature suggests that children's child-care experiences may impact HPA-axis functioning (see Geoffroy et al., 2006; Phillips et al., 2011; Vermeer & van IJzendoorn, 2006). However, the majority of the extant findings have been based on studies of children from relatively low-risk samples. Growing evidence from multiple literatures suggests that links between children's child-care experiences and their subsequent development may differ markedly for children from comparatively higher risk environments. The aim of the present study was to test whether relations between children's child-care experiences and their subsequent cortisol levels at 48 months (controlling 7-month levels) were moderated by children's broader levels of risk. Our results provided some support for such interactive effects. Specifically, there was evidence of a crossover interaction. In the context of low levels of risk, greater hours in child care per week between the ages of 7 and 36 months were predictive of higher cortisol levels at 48 months. In contrast, in the context of high levels of environment risk, greater hours in child care per week were predictive of *lower* cortisol levels at 48 months old. No similar interaction effects emerged for child-care quality or type, nor did child-care quality or type mitigate the interactive effects of child-care quantity and cumulative risk.

Child-Care Quantity, Cumulative Risk, and HPA-Axis Functioning

Prior work with lower risk samples has shown that attending child care may be associated with elevated adrenocortical activity over the course of the child-care day. Although our study considered a different metric of children's HPA-axis activity—basal cortisol levels, rather children's daily diurnal rhythms—our findings suggest that links between children's level of child-care exposure and their subsequent cortisol levels were contingent on their broader levels of environmental risk. Specifically, for children with low levels of environmental risk, greater average hours in child care per week between the ages of 7 and 36 months were associated with higher cortisol levels at 48 months. The direction of this relation, however, was inversed for children with higher levels of cumulative risk factors. For instance, for children with cumulative risk levels that were a standard deviation above the mean, greater hours in child care per week were predictive of *lower* cortisol levels at 48 months. Although this conditional association was somewhat modest—corresponding to a standardized regression coefficient of approximately .12—the effect was robust after adjusting for several control covariates, including children's prior cortisol levels at 7 months. In fact, this standardized conditional association for child care hours is almost as strong a predictor of children's 48-month cortisol levels as children's 7-month cortisol levels.

These findings are consistent with prior studies considering interactive effects between environmental risk and child-care exposure in the context of children's behavioral outcomes. For instance, Côté et al. (2007, 2008) found that the prototypical link between greater hours of child care and heighted levels of child aggression is less pronounced and sometimes reversed for children from higher risk environments.

Further, the positive association between weekly hours of child care and cortisol for children with no or limited environmental risks is to some extent consistent with a convergent body of findings from studies of lower risk samples. Specifically, this work has shown that contrary to their normative diurnal cortisol patterns on non-child-care days, children tend to show increases in their cortisol levels over the course of the day on days in which they attend child care (Geoffroy et al., 2006; Phillips et al., 2011; Vermeer & van IJzendoorn, 2006). Although measures of children's diurnal cortisol levels over time were not collected in the present study, theory suggests that regular and repeated activation of the HPA axis may lead to systemic changes in HPA-axis functioning. Models of allostasis and allostatic load (Ganzel, Morris, & Wethington, 2010; McEwen, 2002) posit that the HPA axis, in coordination with other stress-response systems (e.g., sympathetic and parasympathetic nervous systems), shows regular changes and adaptations that allow the organism to anticipate and respond to normative, transient environmental stressors. However, repeated and chronic activation is also proposed to cause "wear and tear" on these systems, leading to potential changes in their homeostatic set points over time (Ganzel et al., 2010; McEwen, 2002).

Empirically, links between repeated HPA-axis activation and long-term changes in children's overall cortisol levels are somewhat unclear. Indeed, some work suggests that chronic activation of the HPA axis—perhaps, particularly given nonspecies-typical environmental stressors, such as the extreme deprivation—may lead to blunted HPA-axis response indicated by hypocortisolism (Tarullo & Gunnar, 2006). However, other evidence suggests that chronic cumulative stressors—such as those that tend to be more prevalent in low-income contexts—are associated with elevated levels of basal cortisol (Evans & English, 2002) and allostatic load (comprised partially of elevated cortisol levels; Evans & Kim, 2012). Thus, although the long-term effects of children's child-care experiences on the developing HPA axis remain unclear, it seems possible that for those who face limited environmental risk, greater time spent in child care could represent a chronic stressor that up-regulates basal cortisol levels over time.

In contrast, for children facing high levels of environmental risk outside child care, attending greater hours in child care may be associated with comparatively more normative diurnal cortisol profiles that support the down-regulation of the HPA axis over time. The limited available work suggests that—unlike their more affluent peers—children at risk due to economic adversity may show a normative diurnal decline in their cortisol levels across the child-care day (Rappolt-Schlichtmann et al., 2009). This may be explained by the possibility that for children facing heightened environmental risk, child care may be comparatively less physiologically stressing than their experiences outside child care. Therefore, the finding for child-care quantity for these children may partially reflect a dose–response relation, such that child care is a mechanism through which these children spend less time in more highly stressful environments. In turn, HPA-axis functioning may adapt to the comparatively lower levels of stress in child care. This, of course, remains a question to be addressed directly. However, there is some indication that changing the environments of children at environmental risk may have effects on system-wide HPA-axis functioning. For example, although the findings are likely representative of comparatively greater environmental risks, as well as a more dramatic intervention compared with child care, Dozier et al. (Bernard, Butzin-Dozier, Rittenhouse, & Dozier, 2010) found that children from high-risk contexts (i.e., contact with child protective services) who were subsequently placed in foster care showed lower cortisol levels across the day than did those who remained in their homes. As such, the present findings and prior work are consistent with the idea that child care may play a buffering role for children facing heightened environmental risk.

Notably, our design differs from prior studies in ways that make it difficult to compare the findings across studies directly. For instance, few studies have directly tested the effects of child-care "dosage." Instead, most studies are based on samples of children attending relatively full-time care (i.e., 30+ hr). The limited extant findings with respect to child-care dosage have been somewhat mixed. For instance, contrary to typical findings for children attending full-day care, Gunnar, Tout, de Haan, Pierce, and Stansbury (1997) failed to find elevated afternoon cortisol levels in their study of children attending half-day care, possibly suggesting that higher levels of child-care exposure may be required to affect HPA-axis functioning. Other short-term studies have shown no relation between weekly child-care hours and changes in children's diurnal cortisol levels (Vermeer et al., 2010). In contrast, long-term studies have shown that spending greater amounts of time in center-based care in the first 3 years of life (but not weekly hours, per se) to be predictive of a *lower* awakening cortisol levels in adolescence (Roisman et al., 2009). In addition, these authors failed to find interactions between children's child-care experiences and parental sensitivity, suggesting that child-care effects were not particularly pronounced for those with less sensitive parents—one of the risk factors considered in the present study. It should be noted, however, that cortisol response is typically considered to be a distinct process from typical HPA-axis functioning throughout the day (Chida & Steptoe, 2009), and the sample considered by Roisman et al. was diverse, but not reflective of especially high-risk families. Thus, although our findings with respect to child-care quantity are consistent with theory, inconsistencies across the literature indicate that further replication is warranted.

Child-Care Quality and Type, Cumulative Risk, and HPA-Axis Functioning

Contrary to our hypotheses, child-care quality was not associated with children's 48-month cortisol levels. There were no main-effect relations, nor did quality interact with cumulative risk or moderate the two-way interaction between child-care hours and cumulative risk. These null relations were somewhat unexpected. Ample prior evidence has indicated that children who attend higher quality child care tend to show diurnal cortisol patterns that are comparatively closer to normative diurnal declines (Dettling et al., 2000; Sims et al., 2006; Vermeer et al., 2010). In addition, we reasoned that greater doses of high-quality care would serve as a comparatively stronger buffer against the effects of high environmental risk than equally high doses of lower quality care. Yet, similar to other longer term studies (e.g., Roisman et al., 2009), we found no relation between quality and children's cortisol levels.

These null relations may be explained in part by our proxy measure of child-care quality—nonparental caregiver responsivity measured using the HOME scale (Caldwell & Bradley, 1984).

Traditional measures of child-care quality, such as the ECERS (Harms & Clifford, 1980), which provide somewhat broader representations of structural, process, and physical quality of children's experiences in child care could account for the effects of child-care quantity on HPA-axis functioning. Indeed, there may be a dose–response relation between quality and quantity that emerges in the context of more comprehensive measures of quality (e.g., Votruba-Drzal et al., 2004). Notably, however, this was not evident in the present data.

There was also no evidence that attending center-based care was associated with children's cortisol levels. This relation was not moderated by cumulative risk, nor was the interaction between cumulative risk and child-care quantity mitigated by child-care type. We are hesitant to interpret null effects substantively; however, this aligns with some prior work, which has suggested that the prototypical diurnal cortisol increase seen on child-care days tends to occur irrespective of whether children attend center- or home-based child care (Groeneveld, Vermeer, van IJzenfoorn & Linting., 2010; however, see Roisman et al., 2009, with respect to links between center-based care and cortisol awakening response in adolescence). Although center-based care has been linked to potential stressors, such as noisier environments (Groeneveld et al., 2010), larger peer groups (Morrissey, 2010), and fewer teachers available per child (Groeneveld et al., 2010), center-based care has also been linked with potential supports against environmental stress, such as predictable schedules, better trained caregivers, and richer learning environments (Fuller et al., 2004). With respect to HPA-axis functioning, these differing supports and challenges across child-care types may essentially cancel each other out, negating any effect of child-care type.

Thus, taken together, the present findings observed for child-care quantity were evident irrespective of child-care quality or type. This is consistent with some behavioral studies, which have shown that greater hours in child care are associated with children's social development in early childhood even after adjusting for quality and type (Belsky et al., 2007; NICHD ECCRN, 2003; however, see McCartney et al., 2010). Notably, as raised by others (e.g., NICHD ECCRN, 2003), the fact that the conditional child-care quantity relations were not explained by quality or type highlights the fact that the mechanisms underlying the effects of child-care quantity often remain unclear. There are likely other aspects of children's experiences in child care that were not measured in the present study, such as home and classroom chaos (Groeneveld et al., 2010) and the quality of children's peer (Gunnar, Sebanc, Tout, Donzella, & van Dulmen, 2003) and teacher–child (Lisonbee, Mize, Payne, & Granger, 2008) relationships that may explain the relation between child-care quantity and HPA-axis functioning. These remain areas of future study. Notably, the present findings suggest that when possible, measuring contexts across home and child care may be critical to understanding their respective effects on development.

Limitations and Future Directions

Although the present findings were robust after adjusting for several covariates potentially linked to both children's child-care experiences, as well as their subsequent cortisol levels, the potential endogeneity inherent in our study design precludes us from making any causal inferences. There may be unobserved (or unobservable) variables that explain the interactive effects illustrated presently. However, these potential unobserved confounds would have to explain both selection into cumulative risk and selection into child care, as well as why the direction of the child-care effects varies as a function of cumulative risk.

Unlike most prior work considering links between children's child-care experiences and HPA-axis functioning, in which researchers have tended to measure children's diurnal cortisol rhythms across the day, we used measures of children's cortisol level when they were at rest—after they had been given time to become acquainted with the home visitor being in their home and prior to engaging the child in a broader battery of tasks. We did not obtain multiple measures across the day, which could strengthen the reliability of our cortisol measures (Hellhammer et al., 2007), as well as allow us to make more direct comparisons with the findings from prior work. Notably, this difference also raises a possible alternative interpretation of our findings.[2] For instance, given that saliva was collected in children's homes by research assistants (RAs), it is possible that children from higher risk environments tended to show more cortisol reactivity to the RAs' presence and that this effect was mitigated for children attending child care, who may be more regularly exposed to novel adults and social situations. That is, the interactive effects may, to some degree, tap cortisol reactivity to novel social events, rather than children's basal cortisol levels, per se. Ultimately, the correct interpretation remains unclear. Although we are unaware of work suggesting that children from higher risk environments tend to show heightened cortisol reactivity to novel adults, it is possible that this may be the case. It should be noted, however, that by 48 months, children in this sample had been receiving (typically, the same) RAs into their homes multiple times a year since infancy. Thus, although we cannot rule out the possibility that our cortisol measures may partially tap reactivity to the RAs, there is also reason to suspect that the RA visits to the home may not be interpreted by the child as being particularly novel.

Finally, it is worth noting that using terms like *buffering*, with respect to the negative relation between child-care quantity and children's cortisol levels for children with high levels of environmental risk, assumes implicitly that lower cortisol levels are substantively "better" than higher cortisol levels. Indeed, an alternative interpretation of the present findings could be that high levels of cumulative risk make these children particularly vulnerable to child-care effects that cause underactivation of the HPA-axis (i.e., hypocortsolism)—which is also a risk factor for less optimal cognitive functioning (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007) and behavior problems in later childhood (Alink et al., 2008; Gunnar & Vazquez, 2006). This remains an empirical question to be addressed directly; however, prior work gives us reason to doubt this second interpretation. First, with these data, environmental risks like low-quality parenting (Blair, Granger, et al., 2011) and household instability (Blair, Raver, et al., 2011) are associated with higher rather than lower levels of cortisol. Second, higher rather than lower levels of cortisol are predictive of worse executive functioning and lower levels of academic achievement (Berry, Blair, Willoughby, Granger, & the Family Life Project, 2012; Blair, Granger, et al., 2011). Thus, if the lower levels of

[2] We thank an anonymous reviewer for highlighting this interpretation.

cortisol predicted for high-risk children attending substantial hours of child care are a risk factor for less optimal development, it would seem to contradict prior findings with these data. Nonetheless, we intend to address these questions with respect to the potential secondary effects on children's behavior in future work.

Summary

Despite these limitations and areas in need of future study, the present findings suggest that links between children's child-care experiences and their subsequent HPA-axis functioning may differ markedly for children from high- and low-risk contexts. For children facing few environmental risks, greater hours in child care per week in the first 3 years of life were predictive of higher cortisol levels at 48 months of age. This relation was inversed for those experiencing several environmental risks—greater hours in child care were predictive of lower cortisol levels. There was no evidence of main or interactive effects for child-care quality or type. These results demonstrate the importance of considering the extent to which links between child care and development may vary for those from different home contexts.

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