Title
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Journal
Family Relations, 65(1)

ISSN
0197-6664

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Publication Date
2016-02-01

DOI
10.1111/fare.12184

Peer reviewed

HHS Public Access

Author manuscript

Fam Relat. Author manuscript; available in PMC 2018 May 21.

Published in final edited form as:

Fam Relat. 2016 February ; 65(1): 51–72. doi:10.1111/fare.12184.

Child Care and Cortisol Across Infancy and Toddlerhood: Poverty, Peers, and Developmental Timing

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Abstract

Evidence suggests that relations between child care and children's development—behaviorally and physiologically—likely differ between children from high-versus low-risk contexts. Using data from the Family Life Project (N = 1,155), the authors tested (a) whether within- and between-child differences in children's child care experiences (i.e., quantity, type, caregiver responsivity, and peer exposure) were predictive of their cortisol levels across infancy and toddlerhood and (b) whether these relations differed for children experiencing different levels of environmental risk. They found some evidence of such interactive effects. For children from high-risk contexts, within-child increases in child care hours were predictive of cortisol decreases. The inverse was evident for children from low-risk contexts. This relation grew across toddlerhood. Whereas a history of greater center-based child care was predictive of heightened cortisol levels for low-risk families, this was not the case for children from high-risk families. Irrespective of risk, greater peer exposure (between children) was associated with lower cortisol levels.

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Keywords

child care; cortisol; cumulative risk; poverty

The elegant coordination of our physiological stress systems—the fast-acting branches of the autonomic nervous system (ANS; parasympathetic and sympathetic) and the comparatively slower hypothalamic–pituitary–adrenal (HPA) axis—supports our abilities to negotiate the normative demands of everyday life as well as adjust to pronounced, real-time threats (Gunnar & Quevedo, 2007; McEwen, 2000; Sapolsky, Romero, & Munck, 2000). Ample evidence illustrates that the development of these systems is highly regulated by children's early experiences, ranging from more distal markers of adversity (e.g., poverty; Blair & Raver, 2012) to children's proximal experiences with meaningful adults (e.g., parental sensitivity; Gunnar & Vazquez, 2006; Hostinar & Gunnar, 2013). Notably, young children often spend their time across multiple ecological contexts. Reflecting this, a well-developed literature indicates that children's child care experiences can have similarly pronounced effects on HPA axis functioning (see Geoffroy, Côté, Parent, & Séguin, 2006; Phillips, Fox, & Gunnar, 2011; and Vermeer & van IJzendoorn, 2006).

With rare exception, however, the extant findings with respect to child care and children's adrenocortical functioning have been based on studies of middle- to upper income families (though see Rappolt-Schlichtmann et al., 2009). There is good reason to suspect that the relation between children's child care experiences and their subsequent development—behaviorally and physiologically—may be quite different for children growing up in the context of economic adversity (Berry et al., 2014, 2016; Côté et al., 2007; Côté, Borge, Geoffroy, Rutter, & Tremblay, 2008; Crosby, Dowsett, Gennetian, & Huston, 2010; Dearing, McCartney, & Taylor, 2009; Votruba-Drzal, Coley, & Chase-Lansdale, 2004). In the present study we build on this literature substantively and methodologically by considering (a) the degree to which such interactive effects between child care and environmental risk are evident in infancy and toddlerhood and (b) the extent to which these relations are robust to model specifications that explicitly disaggregate within- and between-child relations.

The HPA Axis Across Infancy and Early Childhood

The HPA axis maintains a diurnal rhythm marked by a daily peak after waking, a subsequent decline over the course of the day, and a nadir shortly after onset of continuous sleep (Edwards, Evans, Hucklebridge, & Clow, 2001). The diurnal pattern of HPA activity plays important roles in a variety of metabolic, immunological, and psychological processes that support our day-to-day functioning (McEwen, 2000; Miller, Chen, & Parker, 2011). In studies of children, the preferred assessment method of HPA axis activity is the collection of saliva and the measurement of cortisol (Granger et al., 2012). Cortisol is the "end-product" hormone released into the bloodstream from the adrenal glands—the final step in a biological cascade initiated by the hypothalamus and perpetuated by the pituitary gland. In addition to supporting the orchestration of several other processes (e.g., metabolic, immune, reproductive, and autonomic processes), moderate cortisol levels are thought to support

effective neural transmission and optimal learning and high-order cognition (de Kloet, Oitzl, & Joëls, 1999; Het, Ramlow, & Wolf, 2005).

In times of acute physiological or psychological stress, the HPA axis mounts a particularly pronounced response, culminating in high levels of cortisol that reach glucocorticoid receptors throughout the body and brain. Working with the ANS, these acute HPA stress responses coordinate the physiological (e.g., cardiovascular, immune, energy production/transportation) and psychological (e.g., attention, reflex) resources needed to overcome the stressor (Sapolsky et al., 2000). Yet, given negative feedback processes, high cortisol levels also play important regulatory roles in down-regulating HPA axis activation, allowing it and other systems (e.g., inflammatory response) to return to baseline (Johnson, Riley, Granger, & Riis, 2013). Collectively, these complex within- and cross-system dynamics support an organism's ability to both respond to and recover from the effects of environmental stressors (McEwen, 2000; Sapolsky et al., 2000).

HPA axis reactivity and regulation are evident very early in life. Newborn infants can mount an HPA axis response to environmental stimuli (e.g., doctor examination; see Gunnar & Quevedo, 2007; Hostinar, Sullivan, & Gunnar, 2014), and normative circadian rhythms tend to stabilize as infants begin to forego their afternoon naps (Watamura, Donzella, Kertes, & Gunnar, 2004; though see Bright, Granger, & Frick, 2012). However, the span from infancy through early childhood is also a time of meaningful developmental change. Indeed, a growing theoretical and empirical literature indicates that children's early experiences play a critical role in the organization of their emerging adrenocortical systems (Blair & Raver, 2012; Boyce & Ellis, 2005; Del Guidice, Ellis, & Shirtcliff, 2011; Gunnar & Vazquez, 2006; Hostinar et al., 2014).

Economic Adversity and the HPA Axis

Low-income ecologies present a confluence of distal and proximal risk factors thought to influence children's developing physiological stress systems (Berry, in press; Blair, Granger, et al., 2011; Blair, Raver, et al., 2011; Evans & English, 2002) and undermine optimal cognitive and social development (Dearing, Berry, & Zaslow, 2006; Evans, 2004). For example, children growing up in low-income contexts are more likely to face distal stressors, such as inhospitable and dangerous neighborhoods and inadequate access to services and social capital (Crosnoe & Ansari, 2016; Ellen, Mijanovich, & Dillman, 2001; Israel et al., 2006). Such distal risks are known to have trickle-down effects that undermine parents' abilities to effectively read, interpret, and respond to their children's needs (Blair & Raver, 2012; Conger, Conger, & Martin, 2010; Linver, Brooks-Gunn, & Kohen, 2002).

In turn, a convergent literature comprising experimental work with animals (i.e., rodents, nonhuman primates) as well as observational studies of young children indicates that sensitive and responsive caregiving can support adaptive HPA axis functioning (Gunnar & Donzella, 2002; Gunnar & Herrera, 2013; Hostinar & Gunnar, 2013). This is evident with respect to children's acute stress responses. For example, young children with secure attachment relationships and more sensitive caregivers tend to show better regulated HPA axis responses when faced with acute psychological stressors (e.g., strange adults, scary

toys, separation, social rejection; Feldman, Singer, & Zagoory, 2010; Gunnar, Broderson, Nachmias, Buss, & Rigatuso, 1996; Gunnar, Larson, Hertsgaard, Harris, & Brodersen, 1992; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). Changes in the quality of children's caregiving environments have also been linked with their baseline, or resting levels of HPA axis activity. For example, at the more extreme end, children who are moved from very high-risk households (i.e., households experiencing contact with child protective services) into foster care have been found to evince comparatively lower resting cortisol levels than their peers who remain in high-risk homes (Bernard, Butzin-Dozier, Rittenhouse, & Dozier, 2010). It is important to note that similar relations are evident with respect to parenting within the normal range. For instance, in prior work with the same sample as used in the present study, our group showed that higher levels of maternal sensitivity in infancy are predictive lower levels of resting cortisol, after adjusting for income and a number of potential confounds (Blair, Raver, et al., 2011).

Beyond psychosocial risks, children growing in the context of economic adversity are more apt be exposed to households that are more densely populated, noisy, disorganized, and unpredictable—aspects typically discussed under the umbrella term *chaos* (Evans, 2004; Evans & English, 2002; Evans & Wachs, 2010). A growing literature suggests that chaotic environments may alter children's ANS (Evans & English, 2002) and HPA axis functioning (Evans & English, 2002) in early and middle childhood. Recent work by researchers in our laboratory suggests similar effects with respect to infants and toddlers (Berry, Blair, Vernon-Feagans, Willoughby, & Granger, 2015), with within-child increases in chaos predictive of contemporaneous increases in resting salivary cortisol in later infancy and toddlerhood.

Child Care and HPA Axis Functioning

Notably, young children growing up in low-income contexts spend substantial amounts of time in settings outside of their homes—such as nonparental child care. Indeed, in the United States approximately 43% of children in poverty attend regular nonparental care by 9 months of age (Capizzano & Adams, 2003). A well-developed literature indicates that young children's early child care experiences also play a meaningful role in their HPA axis functioning. Meta-analytic findings indicate that—compared to their normal diurnal patterns experienced at home—children tend to show cortisol increases across the day on days when they attend child care (see Geoffroy et al., 2006; Vermeer & van IJzendoorn, 2006). Some work suggests these patterns are particularly strong in toddlerhood and the beginning of the early childhood years (Vermeer & van IJzendoorn, 2006; Watamura, Donzella, Alwin, & Gunnar, 2003) and for children who attend lower quality child care (Dettling, Parker, Lane, Sebanc, & Gunnar, 2000; Sims, Guilfoyle, & Parry, 2006). There is also some, albeit limited, evidence of long-term effects; for example, Roisman and colleagues (2009) found that spending greater proportions of time in center-based care in infancy and early childhood was predictive of children's subsequent cortisol awakening response in adolescence.

Although such effects with regard to child care type (i.e., center care, family-based child care) have been rather mixed, there is some reason to suspect that heightened exposure to peers—such as often found in center-based child care—may be a salient stressor for young children (Fabes, Hanish, & Martin, 2003; Gunnar & Donzella, 2002). For instance, at a

behavioral level, the replicated link between greater exposure to center-based child care and subsequently heightened levels of aggression in childhood has been shown to be at least partially explained by the typically higher levels of peer exposure faced by these children (McCartney et al., 2010).

That said, the potential effects of peer exposure on HPA axis functioning in infancy and toddlerhood are largely unknown. Indeed, some work has suggested that heightened peer exposure may be associated with lower levels of HPA activity. For instance, in their generative study of infants and toddlers, Watamura and colleagues (2003) found that toddlers who spent more time playing with their peers tended to show lower cortisol levels than their less social classmates. Of course, the direction of this relation remains unclear; for example, perhaps less physiologically stressed children choose to play more with peers (Watamura et al., 2003; Phillips et al., 2011). However, it nonetheless raises the possibility that peer exposure may provide valuable opportunities for young children to learn to negotiate such complex social contexts.

Economic Adversity, Child Care, and Stress Physiology

With rare exception, the extant literature concerning child care and children's early adrenocortical functioning has been based on rather small, homogeneous samples of children from middle- to upper income families. Informed by findings from studies of children's behavioral outcomes, there is increasing evidence to suggest that child care effects for children growing up in poverty may be quite different those for children from more affluent home families (see McCartney & Berry, 2009).

Accumulating findings indicate that the beneficial effects of high-quality child care may be comparatively more pronounced for children from high-risk contexts. For instance, some work has shown that greater exposure to high-quality child care (Dearing et al., 2009), or even simply attending regular nonmaternal care (Geoffroy et al., 2007), may mitigate the detrimental effects of economic adversity or low levels of maternal education on children's subsequent academic achievement. Similar buffering effects have been noted with respect to children's language development, such that exposure to high-quality language environments in child care may mitigate the negative effects of low-quality language environments experienced at home (Vernon-Feagans, Bratsch-Hines, & The Family Life Project Key Investigators, 2013).

Related findings also extend to children's social development. For example, contrary to the replicated finding that more extensive hours in child care are predictive of heightened levels of aggression (Belsky et al., 2007), increasing evidence suggests that the opposite may be true for children from high-risk home environments. Greater hours in high-quality child care have been linked with lower levels of internalizing and externalizing behavior in samples of low-income children (Votruba-Drzal et al., 2004). Similarly, using data from a large Canadian sample, Côté and colleagues (2008) found that the prototypically positive relation between nonmaternal care and aggression in childhood was evident only for children from middle- to upper class families. Indeed, although it failed to reach statistical significance, there was a descriptive indication that nonmaternal care was associated with better social

outcomes for low-income children. We and our colleagues have recently shown similar findings with respect to child care exposure and several outcomes in pre-kindergarten (Berry et al., 2016). Specifically, we found that for children experiencing high levels of household chaos across early childhood, greater weekly hours in child care were predictive of comparatively fewer behavior problems. In particular, consistent with the idea of a buffering effect, greater child care exposure ameliorated the detrimental relation between household chaos and children's social problems.

We have also recently shown similar interactive relations with respect to children's HPA axis functioning at 48 months of age (Berry et al., 2014); specifically, using the same sample as in the present study, we found that the direction of the relation between child care exposure (averaged between 7 and 36 months) and children's cortisol levels at 48 months varied as a function children's broader environmental risk. For children from low-risk households, greater weekly hours of child care were predictive of higher cortisol levels. In contrast, for children facing substantial cumulative risks at home, greater hours of child care exposure were predictive of lower cortisol levels.

Of note, and contrary to our hypotheses, is that other aspects of children's experiences, such as caregiver responsivity and child care type, were unrelated to children's cortisol level, irrespective of children's home contexts. As such, the potential mechanisms underlying these relations remain unclear. Furthermore, this work was concerned with HPA axis functioning just prior to children's transition to school in relation to their average child care experiences across early childhood. Although this represents an important development span, we know little about the extent to which similar conditional relations are evident much earlier in development. Also, unlike the elegant within-person designs adopted by prior work that has considered links between child care and children's diurnal rhythms on child care versus non–child care days, our findings comprised only between-child analyses. Within-person (or *child-fixed effect*) designs afford important methodological advantages. Most notably, they strengthen the internal validity of one's inferences by essentially treating each individual as his or her own control group. In so doing, this holds all possible time-invariant confounds constant.

The Present Study

The aim of the present study was to consider the degree to which links between broad indices of cumulative environmental risk and children's cortisol levels across infancy and toddlerhood are moderated by children's child care experience across this period. To be specific, informed by our prior work considering strictly between-child differences at older ages, we addressed the following two questions:

1. Are within- and between-child differences in children's child care experiences—quantity, type, caregiver sensitivity, and peer exposure—across infancy and toddlerhood predictive of HPA axis functioning?
2. Are the respective magnitudes (or directions) of these associations contingent on children's levels of cumulative environmental risk at home?

Informed by our prior work, we hypothesized that increases in child care exposure would be associated with contemporaneous increases in children's cortisol levels for those facing low levels of environmental risk. In contrast, we hypothesized that this within-child relation of child care exposure and cortisol levels 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 increases in child care are predictive of contemporaneous decreases in children's cortisol levels. Given that our prior work showed evidence of a similar interaction effect at approximately 48 months of age (Berry et al., 2014), we expected this conditional relation to extend downwardly to children in toddlerhood. However, consistent with cross-sectional findings by others (e.g., Watamura et al., 2003), we allowed for the possibility that the relation might be comparatively weaker in early infancy. In our prior work we have found little evidence of effects of child care type (i.e., center- vs. non-center) or caregiver responsivity with respect to 48-month cortisol. Yet, informed by prior studies by others (Dettling et al., 2000; Roisman et al., 2009; Sims et al., 2006), we nonetheless conjectured that the high-quality and center-based care might play a particularly important role in children's cortisol levels for those from high-risk home environments. Finally, informed by that idea that peer relations may support (or reflect) optimal behavioral and physiological regulation for children in child care (Fabes et al., 2003; Watamura et al., 2003), we hypothesized that within-child increases in positive peer exposure would be associated with cotemporaneous decreases in children's cortisol levels—perhaps, particularly so for children experiencing heightened levels environmental risk. Our hypotheses with respect to between-child differences in children's child care experiences were substantively identical to the within-person representations above.

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 and central Pennsylvania; specifically, 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 was provided by Vernon-Feagans and colleagues (2013). The present analytic sample comprises the 1,155 children with child care data for at least one of the three points at which child care information was collected between the time children were 7 and 24 months of age. Those excluded from the analytic sample did not differ from the present sample with respect to race or family income or primary caregiver education level at 7 months.

Procedure

In the present study we focused on data collected across infancy and toddlerhood. Children's mothers were interviewed regarding demographic and personal information when their children were approximately 2 months of age. The demographic data used to calculate economic adversity, as well as information about child care usage, were collected from mothers during interviews when their children were 7, 15, and 24 months of age. At the ages

of 7, 15, and 24 months, saliva samples were collected from children during home visits to assess resting or "non-(intentionally) stimulated" cortisol levels. Saliva samples were collected after the data collectors had been in the home for at least 1 hour interviewing the primary caregiver and prior to conducting a number of assessments with children. The modal time of collection was approximately 10:00 a.m. at each wave of collection; however, there was some variability. Within-child differences in saliva sampling times across the three home observation points, as well as between-child differences in the (mean) saliva sampling times were adjusted statistically in all models. We use the term *resting* given that no active, purposeful stimulation was present, and children were given ample time (~ 1 hour) to return to baseline after the arrival of the research assistants (RAs). We cannot rule out, however, that our "resting" cortisol levels partly capture HPA axis stimulation due to the visit.

Unstimulated whole saliva was collected using either cotton or hydrocellulose absorbent material and expressing the sample into 2-ml cryogenic storage vials using a needleless syringe (cotton) or by centrifugation (hydrocellulose). Prior studies have indicated no differences in cortisol concentrations associated with the two collection techniques (Granger et al., 2007; Harmon et al., 2007). Children's saliva samples were immediately frozen at −20° C and subsequently stored at −80° C. Intra- and interassay coefficients of variability for cortisol (μg/dL) were, on average, less than 10% and 15%, respectively.

When children were in nonparental care for at least 10 hours per week, interactions between the child care caregiver and the child were rated for caregiver responsivity by independent raters.

Measures

Salivary cortisol—All samples from the 7-, 15-, and 24-month assessments were assayed for salivary cortisol using a highly sensitive enzyme immunoassay US FDA 510k cleared for use as an in vitro diagnostic measure of adrenal function (Salimetrics, State College, PA). The samples were assayed in duplicate, with the average of the duplicates was used in all analyses. The cortisol distributions were subject to log transformation to correct positive skew. We examined child temperature and use of medications (e.g., acetaminophen) as influences on child cortisol); no relations were evident, after adjusting for the time of day when the saliva sample was collected. Saliva collection times were included as both time-varying and time-invariant (i.e., mean time-of-day over time) control covariates.

Child care quantity—Children's primary caregivers provided the average hours per week that the child attended nonparental child care when the child was 7, 15, and 24 months old.

Nonparental caregiver responsivity—Traditional measures of child care quality, such as the Early Childhood Environment Rating Scale (Harms & Clifford, 1980) or the Observational Record of the Caregiving Environment (National Institute of Child Health and Human Development [NICHD] Early Child Care Research Network [ECCRN], 1996) were unavailable in these data. We thus used independent ratings of the nonmaternal caregivers' behavior toward the child, as scored with Home Observation Measure of the Environment scale (HOME; Caldwell & Bradley, 1984), as a proxy for process quality in child care. Indeed, although the HOME Responsivity scale is not a measure of child care quality, per se

—like the process quality measure used in the NICHD Study of Early Child Care and Youth Development (i.e., Observational Record of the Caregiving Environment)—it taps the sensitivity and responsivity of the nonparental caregiver to the child's needs in ways that are comparable across different types of child care.

HOME scale observations were made when the child was 7, 15, and 24 months old. Informed by prior psychometric work (Caldwell & Bradley, 1984), we fitted a series of confirmatory factor analytic (CFA) models, based on 10 items tapping caregiver responsiveness and affection toward the child. (We dropped one item [i.e., caregiver permits messy play] typically used on the Responsivity scale because the CFA models indicated that it was unassociated with the latent construct at each point in time.)

We hypothesized that a single latent factor explained observed caregiver responsivity across the items within a given time point. Because items were scored dichotomously these models were fitted using a robust weighted least-squares estimator (Mplus; Muthén & Muthén, 2009). Descriptions of the CFA models are provided in the online supporting materials on the *Family Relations* website (http://onlinelibrary.wiley.com/journal/10.1111/(ISSN)1741-3729). In short, the results discussed below are based on factor scores estimated from a longitudinal CFA, constrained for partial measurement invariance. Higher values reflect higher levels of caregiver responsivity.

Child care type—Child care type was reported by independent observers during child care visits conducted when the target child was 7, 15, and 24 months of age (0 = non–center based, 1 = center based).

Child care peer exposure—The numbers of peers in the classroom were based on counts conducted by independent observers during child care visits at 7, 15, and 24 months of age.

Cumulative risk—Informed by extensive prior work with these data (see Vernon-Feagans et al., 2013), we created a cumulative-risk composite comprising seven variables—(a) family income, (b) maternal education, (c) constant spouse/partner living in the home, (d) hours of employment, (e) occupational prestige, (f) household density, and (g) neighborhood noise and safety—that were measured across infancy and toddlerhood. As described in detail in the online supporting 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 high (Cronbach's alpha = .92). Higher scores reflect higher levels of cumulative risk. We treated cumulative risk as a strictly between-child or time-invariant variable, given the limed within-person variability in cumulative risk over time (intraclass correlation coefficient = .86).

Control Covariates

Informed by prior studies (e.g., 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. As we describe in detail in the online supporting material, our control covariates included infant gender (male = 1), race (African American = 1), temperament, primary

caregiver reading ability, age at birth, marital status, maternal education, maternal hours worked per week, and research site (NC = 1). These control covariates were measured when the target child was 2 months of age, with the exception of temperament, which was measured at 7 months. Within- and between-person variation in saliva collection times were also controlled in all models.

Missing Data

As indicated in prior work (Blair, Granger, et al., 2011), there is little evidence of selective attrition in the sample through early childhood. Missing data were modest. For the cortisol measures missingness ranged between 13% and 26%, the latter representing children's cortisol levels at 24 months. There was no clear pattern to the missingness; missingnesss was not associated with maternal education, income, or partnered status. Missing data with regard to child care quantity ranged between 3% and 9%. Of those who had a spell of child care (i.e., 10 or more hours), missing data for estimated caregiver responsivity was 19%, and missing data for those reporting center-based versus non–center-based care ranged from 23% to 31%. To adjust for biases emerging due to missing data, we fitted all models using full information likelihood estimation (FIML). FIML estimation helps to reduce biases to the extent to which missingness is missing at random (MAR)—that is, conditionally random after adjusting for the observed variables included in the likelihood function (Enders, 2011). As a robustness check, we fitted an identical taxonomy of models in which only children with 10 or more hours of child care were included in the observed covariance matrix (i.e., excluding those who had never attended care, or child care hours = 0). As the findings were substantively identical and because zero hours represents a "real" value on the childcare-hours distribution, we report findings based on the less restricted sample.

Data-Analytic Plan

To address our questions, we fitted a taxonomy of multilevel models (Singer & Willett, 2003). We present a simplified exemplar model in the equations given below for parsimony and ease of explication. Here, the model tests the extent to which the respective within-person effects of child care hours, age, and a within-person interaction between child care hours and age varies as function of between-person cumulative risk. We, however, tested identical series of models for nonparental caregiver responsivity and type and peer exposure. In all models, we first tested the main effects of cumulative risk and the given child care predictor. We then added the respective two- and three-way interaction terms to the model in distinct steps. Benjamini-Hochberg (1995) adjustments were made for families of repeated tests (e.g., four 3-way interactions) to account for false-discovery rate. In a final step, we integrated all of the child care predictors into a final model to test their unique relations. Only interaction terms that survived Benjamini-Hochberg corrections were integrated into this final specification. (For substantive reasons, we left statistically nonsignificant between-person effects on cortisol growth in the model, even though they technically reflect Predictor × Time interactions.)

Time-varying Level 1 predictors (other than age), $\pi_{1i} - \pi_{4i}$ were group-mean (i.e., person) centered. As such, each carries only within-person variation and is orthogonal to between-child variation in Level 2 predictors. Let π_{1i} represent the conditional within-child effect of

child care hours, π_{2i} represent the conditional linear rate of change in cortisol between 7 and 24 months, π_{3i} represent the conditional within-person interaction of child care hours and age, and π_{4i} represent the respective effects of a vector of time-varying control covariates (e.g., time of saliva collection). Sometimes called a *hybrid model* (Allison, 2009), this multilevel specification is more efficient than typical child-fixed effect models common to the econometrics literature while still retaining the advantages of child-fixed effect models; specifically, the estimates for the Level-1 predictors are identical those obtained using a child-fixed effects specification, such that they account for all potential observed and unobserved time-invariant confounds. The hybrid approach also affords the ability to simultaneously model between-person relations at Level 2. Importantly, for our purposes, they also allow one to estimate cross-level interactions; specifically, as shown by parameter γ_{31}, we tested the three-way cross-level interaction addressing our questions: (1) Does the within-person effect of child care exposure moderate the association between cumulative risk and cortisol, and (2) does this interactive effect vary as a function of age? As displayed, the model for the residual variances includes only within-person, time-specific residual (ε_{ij}) and a random intercept (ζ_{0i}) —implicitly constraining the within-person effects (including linear growth) to be identical across children. This parsimonious specification is tested and relaxed, as appropriate.

Level 1:

$$Cort_{ij} = \pi_{0i} + \pi_{1i}\left(CCHrs_{ij} - \overline{CCHrs_i}\right) + \pi_{2i}\left(Age_{ij} - 7mo_i\right) + \pi_{3i} \left(\left(CCHrs_{ij} - \overline{CCHrs_i}\right) * \left(Age_{ij} - 7mo_i\right)\right) + \pi_{4i}\left(Covar_{ij} - \overline{Covar_i}\right) + \varepsilon_{ij}$$

Level 2:

$$\pi_{0i} = \gamma_{00} + \gamma_{01}\overline{CRisk_i} + \gamma_{02}\overline{CCHrs_i} + \gamma_{03}\overline{Covar_i} + \zeta_{0i}$$
$$\pi_{1i} = \gamma_{10} + \gamma_{11}\overline{CRisk_i} + \gamma_{12}\overline{Covar_i}$$
$$\pi_{2i} = \gamma_{20} + \gamma_{21}\overline{CRisk_i} + \gamma_{22}\overline{CCHrs_i} + \gamma_{23}\overline{Covar_i}$$
$$\pi_{3i} = \gamma_{30} + \gamma_{31}\overline{CRisk_i} + \gamma_{32}\overline{Covar_i}$$
$$\pi_{4i} = \gamma_{40}$$
$$\varepsilon_{ij} = \sim N\ (0, \sigma^2_{\varepsilon});\ \ \zeta_{0i} = \sim N\ (0, \sigma^2_0)$$

Model constraints were fitted to test the simple slopes for statistically significant interactions. For example, within-person effects of child care hours were tested at high (grand mean + 1 *SD*) and low (grand mean − 1) levels of cumulative environmental risk and age. All models were fitted using continuous variables—the simple slopes are merely conditional relations estimated from these models at field-typical “high” and “low” values. As specification checks, we fitted nested models in which the respective effects of the time-varying predictors were allowed to vary randomly across children. All comparisons of nested models were based on Satorra–Bentler (Satorra & Bentler, 2001) adjusted likelihood-ratio tests.

Results

Preliminary Analyses

Across the first 24 months of life, children spent approximately 20.23 hours in child care per week, on average. Just over one third of children (36%) attended 10 or fewer hours of care per week, on average, and approximately one third (33%) attended 30 or more hours of child care per week. The average hours per week spent in child care was rather stable over this period, and there was moderate to strong rank order stability in children's child care hours over this period ($r = .59$ $p < .001$, to $r = .61$, $p < .001$). Approximately 9% of the children attended at least two (of three) 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 child care (compared with non–center-based childcare) tended to experience slightly lower caregiver responsivity, on average (rs = −.08 to −.10, $p < .05$; and $rs = −.21$ to −.27, $p < .001$, respectively). Although there was some indication that less responsive nonmaternal caregiving was correlated with higher levels of salivary cortisol at 7 months ($r = −.13$, $p = .002$), no such relation emerged at any other points in time; neither was cortisol correlated with any other contemporaneous child care variables. Rank-order stability in children's cortisol levels over time was rather modest ($r = .04$, *ns*, to $r = .16$, $p < .001$).

On average, children with higher levels of cumulative risk tended to attend fewer hours in child care per week ($r = −.03$, *ns*, to $r = −.13$ $p = .001$) and, when they did attend, they tended to experience lower caregiver responsivity (rs = −.21 to −.23, $p < .001$) in non–center-based contexts ($r = −.04$, *ns*, to $r = −.08$, $p = .006$). Notably, these relations were rather modest. Cumulative risk showed modest positive zero-order correlations with children's contemporaneous cortisol levels at each point in time ($r = .11$, $p < .001$, to $r = .16$, $p < .001$).

Cumulative Risk, Child Care, and Salivary Cortisol

Child care quantity—As displayed in Column A of Table 2, preliminary main effect models indicated that, when considered in aggregate, within-child changes in child care hours were not associated with contemporaneous changes in children's cortisol levels. However, as evidenced by the statistically significant three-way interaction in Column B ($B = −0.011$, $p = .002$), this null main effect was explained by the fact that direction of the within-person child care effect was conditional on level of cumulative environmental risk experienced by the child, as well as child age. We display these conditional relations in Panels A through C in Figure 1; the values of all other predictors in the model were held at their respective means.

Irrespective of cumulative environmental risk, no within-person child care effects were evident at 7 or 15 months of age; none of the displayed simple slopes for child care hours differed statistically from zero. Instead, the interactive effect grew over time, becoming more pronounced and statistically significant by 24 months of age. For children from high-risk (i.e., grand mean +1) contexts, increases in child care exposure at 24 months were associated with contemporaneous decreases in children's resting cortisol levels. In contrast, for children

from low-risk contexts, increases in children's child care exposure at 24 months were associated with contemporaneous increases in children's resting cortisol levels Tests of model constraints indicated that the *absolute* magnitude of the simple slopes displayed in Panel C of Figure 1 were statistically identical ($B_{\text{hirisk}} = -0.007$, $p = .001$ and $B_{\text{lorisk}} = 0.007$, $p = .001$, respectively). In absolute terms, these relations corresponded to absolute standardized simple slopes of approximately .14. (All standardized parameters are based on the variation existing at the given level of analysis, i.e., standardized within-person relations are calculated using only within-person variation in the X and Y. Standardized between-person difference are calculated using only the between-person variation in X and Y.) Albeit somewhat modest, it is worth noting that in relative terms these simple slopes are approximately two thirds the size of the standardized within-person effect for time of day (−0.22)—one of the most well-understood biological mechanisms underlying normative within-person variation in cortisol.

Between-child differences in children's average weekly child care hours were predictive of only children's cortisol growth rates (see Table 2, Column B), and neither this relation nor the relation between average child care hours and children's 7-month cortisol levels were moderated by children's level of cumulative environmental risk. Children who attended greater hours in child care on average (i.e., across the 7- to 24-month span of development) tended to show less negative declines in their cortisol levels over, irrespective level of cumulative environmental risk ($B = 0.005$, $p = .01$). We display this relation in Figure 2 for prototypical children experiencing low versus high levels of child care exposure per week on average (i.e., 10 and 40 hours, respectively) across infancy and toddlerhood; all other predictors are held constant at their respective means. Whereas children attending 40 hours of child care per week were estimated to maintain their 7-month cortisol levels over time ($B_{\text{hihours}} = -0.012$, *ns*), those attending 10 hours a week were predicted to show a statistically significant decline their cortisol levels between 7 and 24 months of age ($B_{\text{lohours}} = -0.20$, $p < .001$). Note that this decline was rather modest, approximating a 0.02-*SD* decrease, per month.

Child care type—There was no evidence of a within-person effect with respect to child care type ($B = -0.02$, *ns*; see Table 2, Column B); that is, time-specific shifts to center-based or non–center-based child care were not associated with contemporaneous changes in cortisol. There was, however, some evidence that the positive between-person relation between attending comparatively more center-based care (than non-center–based care) between 7 and 24 months and children's cortisol levels differed for children from higher versus lower levels of cumulative risk ($B = -0.17$, $p = .03$; see Table 2, Column B). This two-way interaction did not vary as a function of age—that is, it was not predictive of cortisol growth. There was, however, evidence that attending greater proportions of time in center-based child care was predictive of more rapid cortisol decreases over this period ($B = -0.22$, $p = .03$). In the former case of the interaction, it is also worth noting that the raw p-value for this estimate, .01, dropped to marginal levels of statistical significance (i.e., $p = .06$) after adjusting family-wise false discovery rate. We nonetheless display the simple slopes derived from this interaction in Figure 3, with this caveat in mind.

As shown in Figure 3, we found evidence that, for children experiencing low levels of environmental risk, those who attended comparatively greater proportions of their time in center-based child care (compared to non–center-based care) tended to have higher cortisol levels ($B_{\text{lorisk}} = 0.33$, $p = .001$). This is most clearly illustrated by considering the vertical distance between the dashed and nondashed gray trajectories at 7 months in Figure 3; it reflects the 7-month simple slope for those attending 100% of their time in child care in center-based care, compared to those who spent 100% of their time in non–center-based child care. This conditional relation is noteworthy, approximating a standardized association of .41. In contrast, child care type was unrelated to children's 7-month cortisol levels, for children from high-risk contexts ($B_{\text{hirisk}} = .13$, *ns*). As indicated by the divergence of the dashed and nondashed black trajectories over time, there was a descriptive indication that, for those from high-risk contexts, attending predominantly non–center-based care was predictive of comparatively less rapid cortisol declines between 7 and 24 months. However, the conditional effect of child care type at 24 months (mo) only approached statistical significance ($B_{\text{hirisk24mo}} = -.20$, $p = .12$). Considered another way, despite the descriptive differences in the estimated cortisol values plotted at 24 months of age in Figure 3, these values were statistically identical.

Nonparental caregiver responsivity—Within-person changes in caregiver responsivity between 7 and 24 months were not associated with contemporaneous changes in children's cortisol levels, irrespective of child age or level of cumulative risk. Similarly null relations were evident with respect to between-person differences in children's average levels of caregiver sensitivity across this period.

Peer exposure—Within-child changes in peer exposure (i.e., number of peers) were not associated with changes in children's cortisol levels over time. However, there was some indication that between-child differences in children's average levels peer exposure were predictive of children's overall cortisol levels. Specifically, on average, children who typically attended child care with greater numbers of peers tended to evince lower cortisol levels ($B = -0.03$, $p = .008$; see Table 2, Column B). This corresponded to a moderate standardized coefficient of approximately −.27. Adjusting for false-discovery rate, this relation did not vary over time or as a function of children's levels of cumulative risk.

Discussion

There is good evidence that children's child care experiences affect HPA axis functioning (see Geoffroy et al., 2006; Phillips et al., 2011; and Vermeer & van IJzendoorn, 2006). However, it is increasingly clear that these relations may be quite different for children growing up in high-risk environmental contexts. The aims of the present study were twofold. The first aim was to test the extent to which within- and between-child differences in children's child care experiences—quantity, type, caregiver responsivity, and peer exposure—across infancy and toddlerhood were associated with children's cortisol levels over this period. The second aim was to consider whether these relations differed for children experiencing higher levels cumulative contextual risk outside of child care.

Our results provide some support for such interactive effects. As hypothesized, for children from low-risk contexts, within-child increases in weekly child care hours were associated with contemporaneous increases in children's resting cortisol levels. The inverse was evident for children from high-risk contexts; increases in weekly child care hours were associated with contemporaneous cortisol decreases. The magnitude of this cross-level interaction strengthened over time, reaching statistical significant between the ages of 15 and 24 months. Interactive effects between cumulative risk and attending center-based care were also evident; however, they extended to only between-child differences in overall center-based exposure between 7 and 24 months of age (i.e., not within-person changes in center-based care). Controlling for child care hours, caregiver responsivity, and peer exposure, children from low-risk families who spent greater proportions of their time in center-based child care tended to have higher cortisol levels than their peers who attended more non–center-based childcare. In contrast, for children from high-risk contexts attending center-based care was unassociated with children's cortisol levels. With respect to children's exposure to peers in child care, there was evidence that children exposed to greater numbers of peers between 7 and 24 months (on average) tended to show comparatively lower cortisol levels. This relation did not vary as a function of cumulative risk or age. Within-person changes in peer exposure were unrelated to changes in cortisol, irrespective of age or level of cumulative risk.

Child Care, Cumulative Risk, and HPA Axis Functioning

Prior work conducted with lower risk samples has shown that children's cortisol levels tend to increase over the course of the day on days when they attend child care yet show normative diurnal declines on days in which they do not attend child care (Geoffroy et al., 2006; Vermeer & van IJzendoorn, 2006). Nontrivial differences between this prior literature and the present study make direct comparisons impossible. Prior work has typically concerned diurnal cortisol rhythms over the course of the day, within-child child care variation across a more limited span (i.e., ~1 week), and rather blunt indices of child care exposure (i.e., no care [at home] vs. full-time care; but see Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997). In contrast, we considered children's resting cortisol levels at a single point in time, within-child variation over the course of many months, and variation in the actual number of hours children spent in child care per week. Despite these differences (or perhaps because of them), the commonalities between this prior work and the present findings are noteworthy. Taken collectively, the findings suggest that within-child increases in child care exposure—on both short- and long-term developmental time scales—tend to be associated with within-child increases in salivary cortisol, for children from low-risk contexts. In particular, in the present study we found that—holding all other time-invariant confounds constant—fairly long-term shifts in child care exposure across spans of several months may underlie changes in HPA axis function, at least at 24 months of age.

This finding with respect to developmental timing is consistent with meta-analytic data suggesting that the link between child care and children's cortisol increases over the course of the child care day tends to be quite small in infancy yet pronounced toward early childhood (Geoffroy et al., 2006). The cause of these developmental differences remains largely unclear. On the one hand, it is reasonable to conjecture that the types of experiences

that occur in child care, such as exposure to peers, noise, and the divided attention of caregivers, might be particularly salient stressors during infancy. Developmentally, infants lack the internal self-regulatory control that is presumably needed to down-regulate their stress responses (Feldman, 2007; Fox & Calkins, 2003). On the other hand, children's experiences of the same child care context may also be quite different at different points in development (Geoffroy et al., 2006; Watamura et al., 2003). For instance, whereas an infant and early-childhood classroom may maintain the same number of peers, children tend to be more actively engaged with their peers in early childhood (positively and negatively; Watamura et al., 2003). Such normative social differences may begin to explain the emergence of the childcare effect in toddlerhood. This is an important question for future work.

Critically, our findings indicated an inversed within-child relation between child care hours and cortisol at 24 month of age, for children from high-risk contexts. For instance, for toddlers with cumulative risk levels that were 1 *SD* above the mean, increases in weekly child care hours were associated with contemporaneous decreases in children's cortisol levels. Although this conditional association was somewhat modest—corresponding to a standardized regression coefficient of approximately .14—it is approximately one quarter of the size of the effect of time of day, a known biological mechanism underlying within-person cortisol variation. Furthermore, the estimate is robust as a within-person effect, which (by design) adjusts for all possible time-invariant confounds.

These findings align with our prior work with respect to testing between-child interaction effect as predictive of children's resting cortisol levels at 48 months. In that work we found similarly that the direction of the association between child care hours and children's cortisol levels differed for children from low- versus high-risk contexts (Berry et al., 2014). For children experiencing low levels of cumulative risk, attending a greater number of child care hours—on average, between 7 and 35 months—was predictive of comparatively higher cortisol levels, whereas for high-risk children a negative relation was evident. Indeed, the consistency between our prior and present findings is rather remarkable, given the different substantive interpretations of between- versus within-person effects. The former represents one's average child care exposure compared to other children's average exposures; the latter represents time-specific increases in child care hours at 24 months, compared to one's own prior child care hours at 7 and 15 months—irrespective of one's average level. It is important to note that our prior interactive findings at 48 months and the present interactive findings between 7 and 24 months almost certainly tap distinct effects. As above, the interpretations differ. In addition, 24-month and 48-month cortisol levels are virtually orthogonal ($r = .09$).

The present findings also align with prior studies that have considered interactive effects between environmental risk and child care exposure in the context of children's behavioral outcomes. For example, using the same sample as the present study, in prior work we have found that, for children experiencing high levels of household chaos across early childhood, greater weekly hours in child care were predictive comparatively fewer behavior problems. In particular, consistent with the idea of a buffering effect, greater child care exposure ameliorated the detrimental relation between household chaos and children's social

problems. Similar interactive effect have been noted by others (Côté et al., 2007, 2008), and work conducted with predominantly lower income samples has increasingly shown that the "beneficial" relations between child care and children's social and academic outcomes may be stronger and more consistent than those from studies of children from more affluent families (Crosby et al., 2010).

Despite some commonalities emerging across this literature, the mechanisms underlying these interactions remain largely unclear. Our results provide some indication that center-based care may play a role. We found that—for children from low-risk contexts—attending greater proportions of one's time in child care in center-based care was predictive of higher cortisol levels than spending more time on other types of nonparental care. In contrast, no such relation was evident for children from high-risk contexts. To some degree, this is may be inconsistent with prior work. For instance, on the basis of data from their heterogeneous (though not terribly high-risk) sample, Roisman and colleagues (2009) found that greater exposure to center-based care in early childhood was predictive of a lower awakening cortisol levels in adolescence. Of note, though, is that the cortisol awakening response is typically considered to be a process that is distinct from typical HPA axis functioning throughout the day (Chida & Steptoe, 2009). There is also, however, some reason to suspect that our findings align with some behavioral findings. For instance, there is a good indication that greater exposure to center-based care may be linked to heightened levels of aggression in early childhood (Belsky et al., 2007)—perhaps particularly so for children from more affluent families (Magnuson, Ruhm, & Waldfogel, 2007). Meta-analytic findings indicate a positive correlation between cortisol and aggression levels in early childhood (Alink et al., 2008). Indeed, considering the potential secondary effects of cortisol on behaviors and cognition is an important next step we intend to take with these data.

However, there is also reason to suspect that the translation of children's cortisol levels into behavioral effects will not be a simple one. For example, in the present study we also found that children who were exposed to greater numbers of peers, on average, between 7 and 24 months, tended to have lower cortisol levels. This is consistent with the (limited) extant data (e.g., Watamura et al., 2003), yet it is somewhat inconsistent with the idea that the link between center-based care and heightened levels of aggression is explained by its effects on heightened HPA axis activity: Peer problems may well underlie the effects of center-based care on aggression (McCartney et al., 2010). Suffice it to say, there is much left to clarify. Interestingly, prior work has been suggested that the negative relation between greater peer play and lower daily cortisol levels may reflect the fact children who are less physically aroused may "select into" greater amounts of peer play (Watamura et al., 2003). Of course, this may well be the case, yet it is noteworthy that our finding with regard to the number of children in child care—presumably reflecting peer exposure that is considerably less child driven—shows similar negative relation. Although it remains a conjecture to be tested directly, ongoing practice negotiating the social world of one's peers may, in fact, play a "steeling" or "inoculating" role in the way that children respond physiologically to subsequent peer interactions. Evidence from nonhuman primates suggests that such steeling effects may be common and play an important role in resiliency (Lyons, Parker, & Schatzberg, 2010).

As such, our present findings suggest that child care type and peer exposure—at least when considered cumulatively between ages 7 and 24 months—are linked with HPA axis functioning across this period. However, it is important to note that they do not appear to explain the effects of childcare *quantity*. Child care quantity, type, and peer exposure were additively and independently predictive of children's cortisol levels, and no high-order interactions were evident. Like our prior work with children's 48-month cortisol levels, caregiver responsivity was not related with children's cortisol levels, between or within persons. Thus, as noted by others in prior work (e.g., NICHD ECCRN, 2003) the mechanism underlying the (conditional) child care quantity effect remains largely unclear.

One possibility is that, for children facing the confluence of environmental challenges of poverty, child care may be comparatively less physiologically stressing than their experiences outside of child care. Indeed, the limited available evidence suggests that—unlike their more affluent peers—children from higher risk contexts tend to show a normative diurnal cortisol decline in their cortisol levels across the child care day (Rappolt-Schlichtmann et al., 2009). If this is the case, then the "beneficial" relation between greater child care exposure and cortisol observed for children from high-risk families may partially reflect a dose–response relation, whereby greater child care exposure leads to less exposure to the pronounced environmental stressors outside of child care. This, of course, remains a question to be addressed directly. Indeed, there are several potentially important mechanisms that were not addressed in the present study—such as the quality of children's peer relations (Gunnar, Sebanc, Tout, Donzella, & van Dulmen, 2003), teacher–child relationship quality (Lisonbee, Mize, Payne, & Granger, 2008), and degree of classroom chaos (Groeneveld, Vermeer, van IJzendoorn, & Linting, 2010)—that remain critical areas inquiry, with regard to clarifying the relation between child care exposure and HPA axis functioning.

Limitations and Future Directions

Several of our findings are robust to within-child analyses that accounted for all time-invariant confound or between-child analyses that adjusted for several observed control covariates. In fact, with regard to within-child effects, the conservative nature of these estimates may underlie the fact that there were several null within-person relations (i.e., type, caregiver responsivity, peer exposure). On the one hand, these specifications may strengthen the internal validity of our inferences. On the other hand, to the extent that the causal relation between these variables is contingent on *cumulative* exposure they would be missed by our within-person specification. Our between-person analyses largely represent the flip side of the same methodological coin. They allow us to pick up cumulative, traitlike variability in these child care measures—variability that is likely quite meaningful. However, our inferences are only causally unbiased to the extent to which we have accounted for all sources of potential endogeneity (e.g., confounds, simultaneity, measurement error, etc.). This assumption is typically dubious. As such, we make no causal claims. Nontrivially, though, these potential endogeneity problems 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 vary as a function of cumulative risk.

As is common with large, comprehensive epidemiological deigns, trade-offs must often be made between breadth and depth. For instance, we were unable to collect multiple saliva samples over the course of a day or across several consecutive days. This would have strengthened the reliability of our cortisol measures and made our findings more directly informative to the extant literature concerning children's diurnal cortisol patterns (Hellhammer et al., 2007). Our saliva samples were collected modally around 10:00 a.m., when the child was at rest, after he or she had had time acclimate to the RAs in the home. There was little evidence that children were particularly stressed by the RAs' visits. The vast majority of mothers rated their children's behavior during the visit as being typical. Of the minority who indicated that their children were behaving differently, it was typically due to external reasons (i.e., being sick, sleepy). For the few who ascribed differences as being due to the RAs' visit, they were evenly split between those showing emotions that were comparatively more negative and comparatively more positive than usual. This is consistent with the extant laboratory-based literature that suggests that exposure to novel adults typically fails to evoke an acute stress response—even when it is intended to do so (Gunnar, Talge, & Herrera, 2009). That being said, we cannot rule out the possibility that our cortisol measures may partially tap reactivity to the RAs' presence.

Finally, by using terms like *beneficial* to describe, for instance, the negative relation between child care hours and cortisol, we imply that lower cortisol levels may be substantively "better" than higher cortisol levels. Other interpretations are possible. For instance, high levels of cumulative risk could make these children especially vulnerable to child care effects that cause the HPA axis to be underactive (i.e., hypocortisolism). Underactive HPA axis functioning is also a known risk factor for less optimal cognitive functioning (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007) and behavior problems (at least, later in childhood; Alink et al., 2008; Gunnar & Vazquez, 2006). We cannot rule this interpretation out; however, we find it less plausible. Using data from the same sample as the present study, prior work has indicated that environmental risks such as high-quality parenting and household stability (Blair, Granger, et al, 2011; Blair, Raver, et al., 2011) are associated with lower cortisol levels. We have also found that lower cortisol levels are predictive of more effective executive functioning and better academic achievement (Berry, Blair, Willoughby, Granger, & The Family Life Project Key Investigators, 2012; Blair, Granger, et al., 2011). If it is the case that the lower cortisol levels we report presently are actually "less optimal," this would seemingly contradict the conclusions from this prior work.

Summary

With these limitations in mind, our findings suggest that the magnitude (and even the direction) of the relations between children's child care experiences and their developing HPA axis functioning may be quite different for children from high versus low levels of environmental risk. For children from high-risk contexts, within-child increases in weekly child care hours were predictive of contemporaneous cortisol *de*creases. The inverse was evident for children from low-risk contexts: Greater child care exposure was predictive cortisol *in*creases. This interactive effect grew over time and was pronounced at 24 months. On a related note, whereas a history of greater exposure to center-based child care (i.e., between children) was predictive of heightened cortisol levels for low-risk families, this was

not the case for children from high-risk families. Finally, there was an indication that—irrespective of cumulative risk—between-child differences in peer exposure were associated with lower cortisol levels. Collectively, these findings add to an emerging literature indicating that links between child care and children's development should be considered in the context of the multiple ecologies they inhabit.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by grants from the National Institute of Child Health and Human Development (1PO1HD39667 and 2PO1HD039667). Cofunding was provided by the National Institute of Drug Abuse, National Institutes of Health Office of Minority Health, National Institutes of Health Office of the Director, National Center on Minority Health and Health Disparities, and the Office of Behavioral and Social Sciences Research. We thank Peg Burchinal and Lynne Vernon-Feagans for their thoughtful comments on a prior draft of this article and express our sincere gratitude to all of the families, children, and teachers who participated in this research and to the Family Life Project research assistants for their hard work and dedication.

References

Alink LRA, van IJzendoorn MH, Bakermans-Kranenburg MJ, Mesman J, Juffer F, Koot HM. Cortisol and externalizing behavior in children and adolescents: Mixed meta-analytic evidence for the inverse relation of basal cortisol and cortisol reactivity with externalizing behavior. Developmental Psychobiology. 2008; 50:427–450. DOI: 10.1002/dev.20300 [PubMed: 18551461]

Allison, PD. Fixed effects regression models. Vol. 160. Thousand Oaks, CA: Sage; 2009.

Belsky J, Vandell DL, Burchinal M, Clarke–Stewart KA, McCartney K, Owen MT. The NICHD Early Child Care Research Network. Are there long–term effects of early child care? Child Development. 2007; 78:681–701. DOI: 10.1111/j.1467-8624.2007.01021.x [PubMed: 17381797]

Benjamini Y, Hochberg Y. Controlling the false discovery rate: A practical and powerful approach to multiple testing. Journal of the Royal Statistical Society Series B. 1995; 57:289–300.

Bernard K, Butzin-Dozier Z, Rittenhouse J, Dozier M. Cortisol production patterns in young children living with birth parents vs. children placed in foster care following involvement of child protective services. Archives of Pediatrics & Adolescent Medicine. 2010; 164:438–443. DOI: 10.1001/archpediatrics.2010.54 [PubMed: 20439794]

Berry, D. Early childhood health disparities, biological embedding, and life-course health. In: Dearing, E., Votruba-Drzal, E., editors. The handbook of early childhood development programs, practices, and policies. Hoboken, NJ: Wiley; in press

Berry D, Blair C, Ursache A, Willoughby M, Garrett-Peters P, Vernon-Feagans L, … The Family Life Project Key Investigators. Child care and cortisol across early childhood: Context matters. Developmental Psychology. 2014; 50:514–525. [PubMed: 23772818]

Berry, D., Blair, C., Vernon-Feagans, E., Willoughby, M., Granger, D. Household chaos and child physiology across infancy and toddlerhood: A child-fixed effect approach. Paper presented at the biennial meeting of the Society for Research in Child Development; Philadelphia. 2015 Mar.

Berry D, Blair C, Willoughby M, Garrett-Peters P, Vernon-Feagans L, Mills-Koonce R. The Family Life Project Key Investigators. Household chaos and children's cognitive and socio-emotional development in early childhood: Does childcare play a buffering role? Early Childhood Research Quarterly. 2016; 34:115–127. [PubMed: 29720785]

Berry D, Blair C, Willoughby M, Granger DA. The Family Life Project Key Investigators. Salivary alpha-amylase and cortisol in infancy and toddlerhood: Direct and indirect relations with executive functioning and academic ability in childhood. Psychoneuroendocrinology. 2012; 37:1700–1711. DOI: 10.1016/j.psyneuen.2012.03.002 [PubMed: 22472478]

Blair C, Granger D, Willoughby M, Mills-Koonce R, Cox M, Greenberg MT. … The Family Life Project Key Investigators. Salivary cortisol mediates effects of poverty and parenting on executive functions in early childhood. Child Development. 2011; 82:1970–1984. DOI: 10.1111/j.1467-8624.2011.01643.x [PubMed: 22026915]

Blair C, Raver CC. Child development in the context of adversity: Experiential canalization of brain and behavior. American Psychologist. 2012; 67:309–318. [PubMed: 22390355]

Blair C, Raver CC, Granger D, Mills-Koonce R, Hibel L. The Family Life Project Key Investigators. Allostasis and allostatic load in the context of poverty in early childhood. Development and Psychopathology. 2011; 23:845–857. DOI: 10.1017/S0954579411000344 [PubMed: 21756436]

Boyce WT, Ellis BJ. Biological sensitivity to context: I. An evolutionary–developmental theory of the origins and functions of stress reactivity. Development and Psychopathology. 2005; 17:271–301. [PubMed: 16761546]

Bright MA, Granger DA, Frick JE. Do infants show a cortisol awakening response? Developmental Psychobiology. 2012; 54:736–743. [PubMed: 22006547]

Caldwell, BM., Bradley, RH. Home Observation for Measurement of the Environment. Little Rock: University of Arkansas Press; 1984.

Capizzano, J., Adams, G. Snapshot of America's Families III, No. 16: Children in low-income families are less likely to be in center-based child care. Washington, DC: The Urban Institute; 2003.

Chida Y, Steptoe A. Cortisol awakening response and psychosocial factors: A systematic review and meta-analysis. Biological Psychology. 2009; 80:265–278. DOI: 10.1016/j.biopsycho.2008.10.004 [PubMed: 19022335]

Conger RD, Conger KJ, Martin MJ. Socioeconomic status, family processes, and individual development. Journal of Marriage and Family. 2010; 72:685–704. [PubMed: 20676350]

Côté SM, Boivin M, Nagin DS, Japel C, Xu Q, Zoccolillo M, Tremblay RE. The role of maternal education and non-maternal care services in the prevention of children's physical aggression. Archives of General Psychiatry. 2007; 64:1305–1312. DOI: 10.1001/archpsyc.64.11.1305 [PubMed: 17984399]

Côté SM, Borge AI, Geoffroy MC, Rutter M, Tremblay RE. Nonmaternal care in infancy and emotional/behavioral difficulties at 4 years old: Moderation by family risk characteristics. Developmental Psychology. 2008; 44:155–168. DOI: 10.1037/0012-1649.44.1.155 [PubMed: 18194014]

Crosby DA, Dowsett CJ, Gennetian LA, Huston AC. A tale of two methods: Comparing regression and instrumental variables estimates of the effects of preschool child care type on the subsequent externalizing behavior of children in low-income families. Developmental Psychology. 2010; 46:1030–1048. DOI: 10.1037/a0020384 [PubMed: 20822221]

Crosnoe R, Ansari A. Family socioeconomic status, immigration, and children's transitions into school. Family Relations. 2016; 65 xx–xx.

Dearing, E., Berry, D., Zaslow, M. Poverty during early childhood. In: McCartney, K., Phillips, D., editors. Blackwell handbook of early childhood development. Boston: Blackwell; 2006. p. 399-423.

Dearing E, McCartney K, Taylor BA. Does higher quality early child care promote low-income children's math and reading achievement in middle childhood? Child Development. 2009; 80:1329–1349. DOI: 10.1111/j.1467-8624.2009.01336.x [PubMed: 19765003]

de Kloet ER, Oitzl MS, Joëls M. Stress and cognition: Are corticosteroids good or bad guys? Trends in Neurosciences. 1999; 22:422–426. [PubMed: 10481183]

Del Giudice M, Ellis BJ, Shirtcliff EA. The adaptive calibration model of stress responsivity. Neuroscience & Biobehavioral Reviews. 2011; 35:1562–1592. [PubMed: 21145350]

Dettling AC, Parker SW, Lane S, Sebanc A, Gunnar MR. Quality of care and temperament determine changes in cortisol concentrations over the day for young children in childcare. Psychoneuroendocrinology. 2000; 25:819–836. DOI: 10.1016/s0306-4530(00)00028-7 [PubMed: 10996476]

Dill, BT. Poverty in the rural US: Implications for children, families, and communities. New York: Annie E. Casey Foundation; 1999.

Edwards S, Evans P, Hucklebridge F, Clow A. Association between time of awakening and diurnal cortisol secretory activity. Psychoneuroendocrinology. 2001; 26:613–622. [PubMed: 11403981]

Enders, CK. Applied missing data analysis. New York: Guilford Press; 2011.

Evans GW. The environment of childhood poverty. American Psychologist. 2004; 59:77–92. DOI: 10.1037/0003-066x.59.2.77 [PubMed: 14992634]

Evans GW, English K. The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. Child Development. 2002; 73:1238–1248. DOI: 10.1111/1467-8624.00469 [PubMed: 12146745]

Evans, GW., Wachs, TD. Chaos and its influence on children's development. Washington, DC: American Psychological Association; 2010.

Fabes RA, Hanish LD, Martin CL. Children at play: The role of peers in understanding the effects of child care. Child Development. 2003; 74:1039–1043. [PubMed: 12938698]

Feldman R. Parent–infant synchrony biological foundations and developmental outcomes. Current Directions in Psychological Science. 2007; 16:340–345.

Feldman R, Singer M, Zagoory O. Touch attenuates infants' physiological reactivity to stress. Developmental Science. 2010; 13:271–278. [PubMed: 20136923]

Fox NA, Calkins SD. The development of self-control of emotion: Intrinsic and extrinsic influences. Motivation and Emotion. 2003; 27:7–26. DOI: 10.1023/A:1023622324898

Geoffroy MC, Côté SM, Borge AI, Larouche F, Séguin JR, Rutter M. Association between nonmaternal care in the first year of life and children's receptive language skills prior to school entry: the moderating role of socioeconomic status. Journal of Child Psychology and Psychiatry. 2007; 48:490–497. [PubMed: 17501730]

Geoffroy MC, Côté SM, Parent S, Séguin JR. Day care attendance, stress, and mental health. Canadian Journal of Psychiatry. 2006; 51:607–615. [PubMed: 17007228]

Granger DA, Kivlighan KT, Fortunato C, Harmon AG, Hibel LC, Schwartz EB, Whembolua GL. Integration of salivary biomarkers into developmental and behaviorally-oriented research: Problems and solutions for collecting specimens. Physiology and Behavior. 2007; 924:583–590. DOI: 10.1016/j.physbeh.2007.05.004

Granger DA, Fortunato CK, Beltzer EK, Virag M, Bright MA, Out D. Focus on methodology: salivary bioscience and research on adolescence: an integrated perspective. Journal of Adolescence. 2012; 35:1081–1095. [PubMed: 22401843]

Groeneveld MG, Vermeer HJ, van IJzendoorn MH, Linting M. Children's well-being and cortisol levels in home-based and center-based child care. Early Childhood Research Quarterly. 2010; 25:502–514. DOI: 10.1016/j.ecresq.2009.12.004

Gunnar MR, Brodersen L, Nachmias M, Buss K, Rigatuso J. Stress reactivity and attachment security. Developmental Psychobiology. 1996; 29:191–204. [PubMed: 8666128]

Gunnar MR, Donzella B. Social regulation of the cortisol levels in early human development. Psychoneuroendocrinology. 2002; 27:199–220. DOI: 10.1016/s0306-4530(01)00045-2 [PubMed: 11750779]

Gunnar, MR., Herrera, AM. The development of stress reactivity: A neurobiological perspective. In: Zelazo, PD., editor. The Oxford handbook of developmental psychology: Vol. 2. Self and other. New York: Oxford University Press; 2013. p. 45-80.

Gunnar MR, Larson MC, Hertsgaard L, Harris ML, Brodersen L. The stressfulness of separation among nine-month-old infants: Effects of social context variables and infant temperament. Child Development. 1992; 63:290–303. [PubMed: 1611934]

Gunnar M, Quevedo K. The neurobiology of stress and development. Annual Review of Psychology. 2007; 58:145–173.

Gunnar MR, Sebanc AM, Tout K, Donzella B, van Dulmen MMH. Peer rejection, temperament, and cortisol activity in preschoolers. Developmental Psychobiology. 2003; 43:346–368. DOI: 10.1002/dev.10144 [PubMed: 15027418]

Gunnar MR, Talge NM, Herrera A. Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. Psychoneuroendocrinology. 2009; 34:953–967. [PubMed: 19321267]

Gunnar MR, Tout K, de Haan M, Pierce S, Stansbury K. Temperament, social competence, and adrenocortical activity in preschoolers. Developmental Psychobiology. 1997; 31:65–85. [PubMed: 9222117]

Gunnar, M., Vazquez, D. Stress neurobiology and developmental psychopathology. In: Cicchetti, D., Cohen, DJ., editors. Developmental Psychopathology: Vol. 2. Developmental neuroscience. 2. Hoboken, NJ: Wiley; 2006. p. 533-577.

Harmon AG, Hibel LC, Rumyantseva O, Granger DA. Measuring salivary cortisol in studies of child development: watch out—What goes in may not come out of saliva collection devices. Developmental Psychobiology. 2007; 49:495–500. [PubMed: 17577235]

Harms, T., Clifford, RM. Early Childhood Environment Rating Scale. New York: Teachers College Press; 1980.

Hellhammer J, Fries E, Schweisthal OW, Schlotz W, Stone AA, Hagemann D. Several daily measurements are necessary to reliably assess the cortisol rise after awakening: State and trait components. Psychoneuroendocrinology. 2007; 32:80–86. DOI: 10.1016/j.psyneuen.2006.10.005 [PubMed: 17127010]

Het S, Ramlow G, Wolf OT. A meta-analytic review of the effects of acute cortisol administration on human memory. Psychoneuroendocrinology. 2005; 30:771–784. [PubMed: 15919583]

Hostinar CE, Gunnar MR. Future directions in the study of social relationships as regulators of the HPA axis across development. Journal of Clinical Child & Adolescent Psychology. 2013; 42:564–575. [PubMed: 23746193]

Hostinar CE, Sullivan RM, Gunnar MR. Psychobiological mechanisms underlying the social buffering of the hypothalamic–pituitary–adrenocortical axis: A review of animal models and human studies across development. Psychological Bulletin. 2014; 140:256–282. DOI: 10.1037/a0032671 [PubMed: 23607429]

Israel B, Schulz A, Estrada-Martinez L, Zenk S, Viruell-Fuentes E, Villarruel A, Stokes C. Engaging urban residents in assessing neighborhood environments and their implications for health. Journal of Urban Health. 2006; 83:523–539. DOI: 10.1007/s11524-006-9053-6 [PubMed: 16739052]

Johnson SB, Riley AW, Granger DA, Riis J. The science of early life toxic stress for pediatric practice and advocacy. Pediatrics. 2013; 131:319–327. [PubMed: 23339224]

Linver MR, Brooks-Gunn J, Kohen DE. Family processes as pathways from income to young children's development. Developmental Psychology. 2002; 38:719–734. DOI: 10.1037/0012-1649.38.5.719 [PubMed: 12220050]

Lisonbee JA, Mize J, Payne AL, Granger DA. Children's cortisol and the quality of teacher–child relationships in child care. Child Development. 2008; 79:1818–1832. DOI: 10.1111/j.1467-8624.2008.01228.x [PubMed: 19037952]

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

Lyons DM, Parker KJ, Schatzberg AF. Animal models of early life stress: Implications for understanding resilience. Developmental Psychobiology. 2010; 52:616–624. [PubMed: 20957724]

Magnuson KA, Ruhm C, Waldfogel J. Does prekindergarten improve school preparation and performance? Economics of Education Review. 2007; 26:33–51.

McCartney, K., Berry, D. Whether the environment matters more for children in poverty. In: McCartney, K., Weinberg, R., editors. *Experience and development: A* festschrift *for Sandra Scarr*. London: Taylor & Francis; 2009. p. 99-124.

McCartney K, Burchinal M, Clarke–Stewart A, Bub K, Owen M, Belsky J. The NICHD Early Child Care Research Network. Testing a series of causal propositions relating time in child care to children's externalizing behavior. Developmental Psychology. 2010; 46:1–17. DOI: 10.1037/a0017886 [PubMed: 20053002]

McEwen BS. The neurobiology of stress: From serendipity to clinical relevance. Brain Research. 2000; 886:172–189. DOI: 10.1016/s0006-8993(00)02950-4 [PubMed: 11119695]

Miller GE, Chen E, Parker KJ. Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. Psychological Bulletin. 2011; 137:959–997. [PubMed: 21787044]

Muthén, LK., Muthén, BO. Mplus user's guide. Los Angeles: Muthén & Muthén; 2009.

Nachmias M, Gunnar M, Mangelsdorf S, Parritz RH, Buss K. Behavioral inhibition and stress reactivity: The moderating role of attachment security. Child Development. 1996; 67:508–522. [PubMed: 8625725]

National Institute of Child Health and Human Development Early Child Care Research Network. Characteristics of infant child care: Factors contributing to positive caregiving. Early Childhood Research Quarterly. 1996; 11:296–306. DOI: 10.1016/S0885-2006(96)90009-5

National Institute of Child Health and Human Development Early Child Care Research Network. Does amount of time spent in child care predict socioemotional adjustment during the transition to kindergarten? Child Development. 2003; 74:976–1005. [PubMed: 12938694]

Phillips DA, Fox NA, Gunnar MR. Same place, different experiences: Bringing individual differences to research in child care. Child Development Perspectives. 2011; 5:44–49. DOI: 10.1111/j.1750-8606.2010.00155.x

Rappolt-Schlichtmann G, Willett JB, Ayoub CC, Lindsley R, Hulette AC, Fischer KW. Poverty, relationship conflict, and the regulation of cortisol in small and large group contexts at child care. Mind, Brain, and Education. 2009; 3:131–142. DOI: 10.1111/j.1751-228X.2009.01063.x

Roisman GI, Susman E, Barnett-Walker K, Booth-LaForce C, Owen MT, Belsky J. … The NICHD Early Child Care Research Network. Early family and child-care antecedents of awakening cortisol levels in adolescence. Child Development. 2009; 80:907–920. DOI: 10.1111/j.1467-8624.2009.01305.x [PubMed: 19489911]

Sapolsky RM, Romero LM, Munck AU. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocrine Reviews. 2000; 211:55–89. DOI: 10.1210/er.21.1.55

Satorra A, Bentler PM. A scaled difference chi-square test statistic for moment structure analysis. Psychometrika. 2001; 66:507–514. DOI: 10.1007/BF02296192

Sims M, Guilfoyle A, Parry TS. Children's cortisol levels and quality of child care provision. Child: Care, Health and Development. 2006; 32:453–466. DOI: 10.1111/j.1365-2214.2006.00632.x

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

Vermeer HJ, van IJzendoorn MH. Children's elevated cortisol levels at daycare: A review and meta–analysis. Early Childhood Research Quarterly. 2006; 21:390–401. DOI: 10.1016/j.ecresq.2006.07.004

Vernon-Feagans L, Bratsch-Hines ME. The Family Life Project Key Investigators. Caregiver–child verbal interactions in child care: A buffer against poor language outcomes when maternal language input is less. Early Childhood Research Quarterly. 2013; 28:858–873. [PubMed: 24634566]

Vernon-Feagans L, Cox M. The Family Life Project: An epidemiological and developmental study of young children living in poor rural communities. Monographs of the Society for Research in Child Development. 2013; 78:1–150.

Votruba-Drzal E, Coley RL, Chase-Lansdale PL. Child care and low-income children's development: Direct and moderated effects. Child Development. 2004; 75:296–312. DOI: 10.1111/j.1467-8624.2004.00670.x [PubMed: 15015691]

Watamura SE, Donzella B, Alwin J, Gunnar MR. Morning-to-afternoon increases in cortisol concentrations for infants and toddlers at child care: Age differences and behavioral correlates. Child Development. 2003; 74:1006–1020. DOI: 10.1111/1467-8624.00583 [PubMed: 12938695]

Watamura SE, Donzella B, Kertes DA, Gunnar MR. Developmental changes in baseline cortisol activity in early childhood: Relations with napping and effortful control. Developmental Psychobiology. 2004; 45:125–133. [PubMed: 15505801]

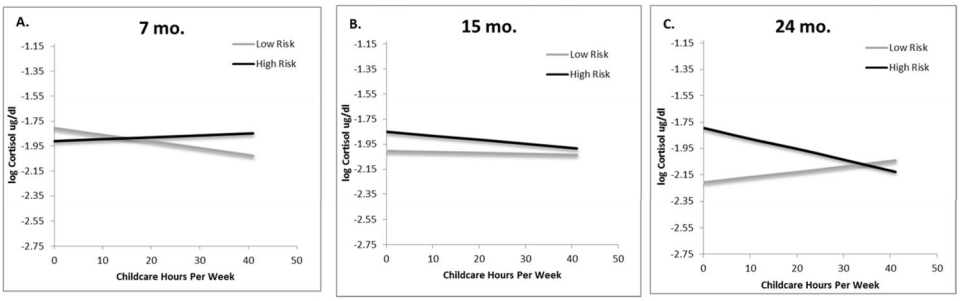

Figure 1.

Fitted Within-Person Relations Between Child Care Hours and Children's Salivary Cortisol Levels, as a Function of Cumulative Environmental Risk and Child Age.

Note. N = 1,155. The y-axis represents the cortisol grand mean ± 2 SD (total cortisol variation). The x-axis represents the grand mean ± 2 SD (within-child variation only). The simple slopes for child care hours are statistically significant at 24 months of age (B_{hirisk} = 0.007, p = .002, β_{hirisk} = .14; B_{lorisk} = −0.007, p = .002, β_{lorisk} = .14). mo. = months.

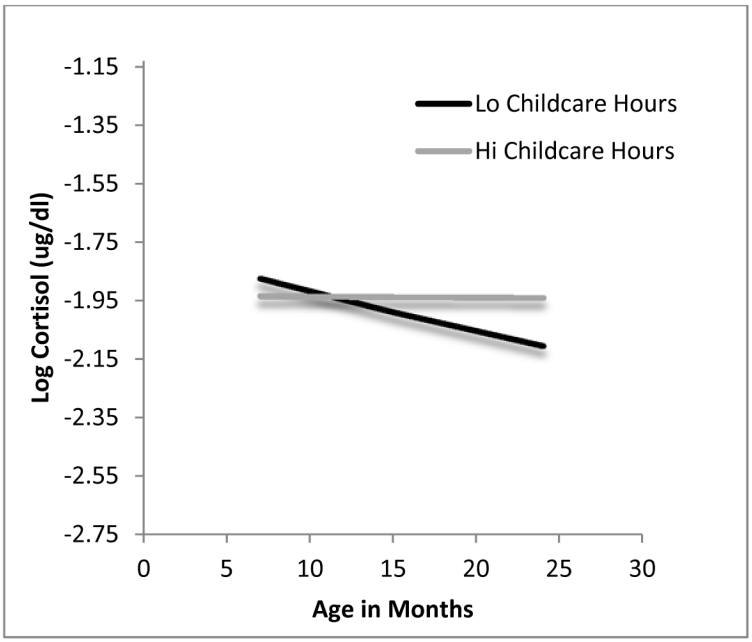

Figure 2.

Fitted Cortisol Growth Trajectories, as a Function of Low (Lo; 10 Hours) and High (Hi; 40 Hours) Levels of Weekly Child Care Hours (on Average) Between 7 and 24 Months.

Note. N = 1,155.

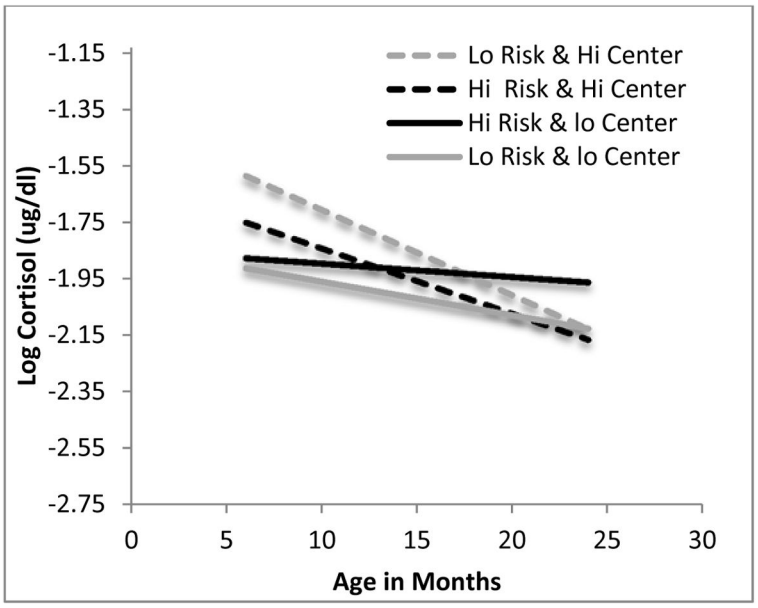

Figure 3.

Fitted Cortisol Growth Trajectories, as a Function of Levels of Cumulative Environmental Risk and Attending 100 % of One's Time in Child Care in Center-Based Care (Hi Center) or 100% of One's Time in Child Care in Non–Center-Based Nonparental Care (Lo Center). *Note.* $N = 1{,}155$. The y-axis represents the cortisol grand-mean ± 2SD (total cortisol variation).

Fam Relat. Author manuscript; available in PMC 2018 May 21.

Table 1

Zero-Order Correlations Between Children's Child Care Experiences, Cumulative Environmental Risk, and Cortisol Levels Between 7 and 24 Months Of Age

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	*M* (*SD*)
1. (ln) Cort 7 mo.	—																−1.84 (0.77)
2. (ln) Cort 15 mo.	.04	—															−1.95 (0.84)
3. (ln) Cort 24 mo.	.11**	.16**	—														−2.05 (0.80)
4. C Risk 6–24 mo.	.11**	.15**	.16**	—													0.02 (0.60)
5. CC CHours 7 mo.	−.01	.03	.06	−.03	—												20.18 (20.94)
6. CC CHours 15 mo.	.01	−.02	.08*	−.08**	.59**	—											20.58 (19.52)
7. CC CHours 24 mo.	.02	−.04	.03	−.13**	.46**	.61**	—										19.92 (20.20)
8. CC Qual 7 mo.	−.13**	−.05	.03	−.21**	−.10**	−.13**	−.10**	—									1.97 (0.52)
9. CC Qual15 mo.	−.12**	−.04	−01	−.23**	−.05	−.10**	−.09*	.75**	—								2.18 (0.45)
10. CC Qual 24 mo.	−.09*	−.04	−.00	−.23**	−.03	−.06	−.08*	.69**	.70**	—							2.06 (0.45)
11. CenterCare 7 mo.	.03	.01	−.06	−.04	.36**	.26**	.23**	−.25**	−.18**	−.20**	—						0.22
12. CenterCare 15 mo.	.02	.02	.00	−.06*	.26**	.46**	.33**	−.28**	−.21**	−.22**	.61**	—					0.26
13. CenterCare 24 mo.	.01	.03	−.04	−.08**	.24**	.35**	.49**	−.21**	−.14**	−.27**	.47**	.65**	—				0.27
14. CC PeerNum 7 mo.	.03	−.01	−.16**	.03	.08	.07	.11*	−.35**	−.27**	−.24**	.66**	.50**	.39**	—			3.23 (2.73)
15. CC PeerNum 15 mo.	−.03	.06	−.12*	−.01	.01	.11*	.10*	−.26**	−.28**	−.20**	.42**	.71**	.46**	.53**	—		4.35 (3.67)
16. CC PeerNum 24 mo.	−.04	.07	−.09	−.08	.01	.07	.08	−.28**	−.20**	−.34**	.39**	.45**	.64**	.43**	.55**	—	5.98 (4.92)

Note. ln = natural logarithm; Cort = cortisol; mo. = months; C Risk = cumulative risk; CC Hours = weekly child care hours, CC Qual = child care quality, CenterCare = attends center-based care = 1; CC PeerNum = number of peers in child care.

* $p < .05$.

** $p < .01$.

Table 2

"Hybrid" Multilevel Models Testing the Respective Within- and Between-Person Relations Between Children's Child Care Experiences, Cumulative Risk, and Cortisol Levels Between 7 and 24 Months of Age

Variable	A	B
Level 1		
CC Hours (WP)	0.00	−0.001
CC Qual (WP)	−0.058	−0.055
CC Type (WP)	−0.033	−0.023
CC PeerNum (WP)	0.013	0.012
Sample ToD (WP)	−0.06***	−0.060
Level 2		
Intercept	−1.89***	−1.90***
C Risk (BP)	0.002	0.03
CC Hours (BP)	−0.002	−0.002
CC Qual (BP)	−0.061	−0.074
CC Type (BP)	0.264**	0.227*
CC PeerNum (BP)	−0.025	−0.029**
CC Type(BP) × C Risk(BP)		−0.17*
CC Hours (WP) × C Risk		0.000
C Risk (BP) × CC Hours (WP)		0.006
Age	−0.10**	−0.10**
C Risk (BP) × age	0.076	0.072
CC Hours (BP) × age	0.005*	0.005*
CC Qual. (BP) × age	0.099	0.098
CC Type (BP) × age	−0.219*	−0.215*
CC PeerNum (BP) × age	0.014	0.014
C Risk (BP) × CC Hours (WP) × age		−0.011**
Control covariates		
Mean sample ToD	−0.013	−0.013
C. temperament	0.040***	0.004
C. African American	0.215***	0.227***
C. NC	−0.052	−0.054
C. male	0.052	0.042
M. literacy	−0.006	−0.005
M. age at child's birth	0.577*	0.591*
M. education	0.008	0.008
M. in school	−0.010	−0.004
M. work hours	0.001	0.000

Variable	A	B
Mean sample ToD × age	0.008	0.008
C. temperament × age	−0.348	−0.343
C. African American × age	−0.043	−0.045
C. NC × age	−0.030	−0.028
Male × age	−0.031	−0.031
M. literacy × age	0.002	0.002
M. age at child's birth × age	−0.764**	−0.786**
M. education × age	−0.055	−0.056
M. in school × age	−0.006	−0.007
M. work hours × age	−0.01	−0.019
Residual variances		
Level 1	0.522	0.522
Level 2	0.045	0.041
Model fit: Deviance	54,498.73	54,482.57

Note. N = 1,155. All covariates other than temperament (7 months) were measured at 2 months of age. CC Hours = weekly child care hours; WP = within-child variation, person-mean centered; CC Qual = child care quality; CC Type = attends center-based care = 1; CC PeerNum = number of peer in child care; C Risk = cumulative risk; BP = between-child variation, grand-mean centered; ToD = time of day of saliva sample; C. = child, M. mother; NC = North Carolina (0 = Pennsylvania); education = years of education; In school = in school (0–1); work hours = number of hours employed per week.

*$p < .05$.

**$p < .01$.

***$p < .001$.