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Basal and reactivity levels of cortisol in one-month-old infants born to overweight or obese mothers from an ethnically and racially diverse, low-income community sample

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Abstract

Establishing typical values of the steroid hormone cortisol at rest and after challenge is critical for understanding how environmental factors impact stress regulation and overall development, beginning at birth. Yet most extant samples are small or based upon low-risk populations, and few studies address the potential role of maternal weight during pregnancy in their study designs or sampling strategy. Here we report basal and reactivity levels of salivary cortisol within a racially and ethnically diverse sample of 132 infants approximately one month of age (Age in days: M= 37.61, SD=7.27) born to lower income overweight or obese mothers. Reactivity was assessed in response to a multi-domain infant stressor paradigm, which included assessment via the Newborn Behavioral Observation (NBO) system and extensive anthropometric measurements. Sample means for basal, post stressors, and reactivity to the NBO were significantly lower than those reported in reviews of low-risk samples. Parity was associated with cortisol levels such that first-born infants had lower resting cortisol and higher reactivity than infants born to multiparous women. Latino infants had lower basal cortisol. No other demographic characteristics significantly predicted cortisol. The variability in cortisol levels present in this sample suggests that considerable psychophysiological diversity may exist in samples of low-SES or high-risk participants. Findings provide useful ranges for samples of racially and ethnically diverse newborns from low-income families.

Keywords

cortisol; reactivity; infant; socioeconomic status; risk; obesity

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1. Introduction

Understanding the development of the hypothalamic-pituitary-adrenal (HPA) axis *in utero* and recruitment of this system during early life has become important for understanding how the stress-response system develops and functions across the life course, beginning at birth (Wadhwa, Buss, Entringer, & Swanson, 2009). Cortisol, a steroid hormone that is secreted when the HPA axis is activated, is a commonly-used proxy for estimating the functionality of the HPA axis (Jansen, Beijers, Riksen-Walraven, & de Weerth, 2010). As researchers increase efforts to understand development and fashion interventions designed to support healthy cortisol regulation (Slopen, McLaughlin, & Shonkoff, 2014), generalizable reference ranges for basal and reactivity are necessary for proper interpretation of cortisol data. Yet normative patterns of salivary cortisol reactivity, in particular, have not been adequately determined in diverse populations (Hunter, Minnis, & Wilson, 2011). This study contributes to the literature by reporting basal cortisol and reactivity profiles established in a sample of approximately one-month-old infants born to ethnically and racially diverse, low-SES, overweight or obese mothers.

Previous studies of infants tend to be based on samples with underlying clinical issues, such as colic (White, Gunnar, Larson, Donzella, & Barr, 2000) or prematurity (Grunau et al., 2007; Herrington, Olomu, & Geller, 2004; Mehler et al., 2015; Morelius, He, & Shorey, 2016), or include mothers with a history of maltreatment (Martinez-Torteya et al., 2015). In addition, the majority of studies are conducted predominantly with White Non-Hispanic samples (Davis, Glynn, Waffarn, & Sandman, 2011; Grunau et al., 2007; Herrington et al., 2004; Martinez-Torteya et al., 2015; White et al., 2000); older infants (Davis et al., 2011; Grunau et al., 2007; Hibel, Granger, Blair, Finegood, & Family Life Project Key, 2015; Martinez-Torteya et al., 2015; Provenzi, Giusti, & Montirosso, 2016; White et al., 2000) and/or middle class/low risk samples (Gunnar & Donzella, 2002; Haley & Stansbury, 2003). A review (Jansen et al. (2010) showed that a limited number of published studies have examined salivary cortisol basal and reactivity levels in infants under 3 months of age; of those studies, sample sizes are small, ranging between 10 and 60 participants, with only two studies including samples over 100. Moreover, all studies included in the review were low-risk. The few studies focused on infants from ethnic or racial minority families included smaller sample sizes and administered infant stressors within 24–48 hours after birth (Keenan, Gunthorpe, & Grace, 2007; Keenan, Gunthorpe, & Young, 2002); targeted the impact of prenatal stress (Luecken et al., 2013), or involved older infants, smaller samples and infants exposed to drugs in utero (Haley, Handmaker, & Lowe, 2006). Keenan et al. (2002; 2007) based upon the same sample, used the Neonatal Behavioral Assessment Scale (NBAS) (a measure similar to the one used in the present study, but more cumbersome and harder to administer) and a heel stick to stress infants within the first two days of life, but data collected from infants so close to the stressful experience of birth raises the possibility of confounded results (Mears, McAuliffe, Grimes, & Morrison, 2004). Although the majority of studies examined infant basal cortisol, since 2010 a number of other major studies or reviews about infant cortisol reactivity have been published focusing on a variety of variables including preterm infants (Morelius et al., 2016), sex differences in childhood (van der Voorn, Hollanders, Ket, Rotteveel, & Finken, 2017), adverse experiences (Hunter et

al., 2011), mother-infant adrenocortical attunement (Hibel et al., 2015), or SES and race/ethnicity in older children (Tackett, Herzhoff, Smack, Reardon, & Adam, 2017).

Although interest in infant cortisol regulation appears to be growing, there is sparse literature examining the association with maternal Body Mass Index (BMI), which may be important because prenatal factors such as maternal obesity may have a "programming" effect on endocrine and immune function in the developing infant (Elhassan, Miller, Vazquez, & Lumeng, 2015; Wadhwa et al., 2009). This area of research is vital given that approximately 70% of the adult population in the United States (66.2% of women, 73% of men) is either overweight (BMI=25 to <30) or obese (BMI=30 or higher), and rates are even higher among women of color (82% of African Americans, 77.1% of Latinas/Hispanics) (Centers for Disease Control and Prevention (CDC), 2016; Flegal, Kruszon-Moran, Carroll, Fryar, & Ogden, 2016). Among the few extant studies, the only one that examined maternal *pre-pregnancy* BMI (Elhassan et al., 2015) found no association between pre-pregnancy maternal BMI and diurnal cortisol or reactivity in pre-school children. Two other studies reported no association between maternal pregnancy weight gain and fasting plasma cortisol concentrations in children 8.5 years of age (Phillips et al., 2005), or between postpartum maternal BMI and infant cortisol (Luecken, MacKinnon, Jewell, Crnic, & Gonzales, 2015; Phillips et al., 2005). More research is needed in this important domain.

Finally, this research also focuses on low-SES families, a large, yet understudied, population. Approximately 30% of the U.S. population lives in "low income" households, with incomes less than 200% of the Federal Poverty Level (FPL) (Henry J. Kaiser Family Foundation, September 22, 2017), and three-quarters of the U.S. population live in homes with incomes less than 500% of the FPL (Doty, 2005). The overall poverty rates for African American or Latino/Hispanic children are 34% and 28%, respectively (The Annie E. Casey Foundation, 2017). Thus, the predominant focus of experimental research on higher income families precludes our ability to truly understand physiological development and functioning within this important and large population of infants.

The specific goals of this study were to provide ranges for newborn basal and reactivity cortisol that might be generalizable to a broader population than is typically assessed (particularly for reactivity) and to examine potential sociodemographic predictors of levels. To address limitations in the extant literature, we provide novel data from a relatively large community sample of healthy young infants born to predominantly low-income women from a range of racial/ethnic and educational backgrounds who were overweight or obese prior to becoming pregnant. We assessed cortisol in infants younger than those typically studied in order to add to the knowledge base about HPA-axis development early in infancy, but after the physiological stress associated with delivery is likely to have passed (Mears et al., 2004). Building upon evidence that physical stressors (e.g., inoculations (Thompson, Morgan, Jurado, & Gunnar, 2015)) provide robust responses, but with a desire to assess reactivity to more environmentally-normative experiences than a "heel-stick," we assessed reactivity using a prolonged protocol that combined a series of typical, developmentally-appropriate physical and social challenges. Though we did not have specific hypotheses, we expected that infants would exhibit a detectable increase in cortisol, in response to the complex

stressor protocol, with sufficient variability to discern whether sociodemographic factors were associated with differences in newborn cortisol values.

2. Method

2.1 Participants

The Stress, Eating, and Early Development study (SEED) is a longitudinal study designed to investigate the associations between prenatal stress and weight gain on child health and development in a cohort of 162 mother-infant dyads (see Bush et al., 2017 for details). Inclusion criteria were that women be 18–45 years of age, 8–23 weeks pregnant with a singleton gestation, have a BMI of 25 – 40, and incomes less than 500% of the FPL (75% of the participants lived at or less than 200% of the FPL). Medical conditions that may interfere with baseline body composition (e.g. diabetes, abnormal glucose.

As noted in Table 1, approximately 75% of the participants lived in households with incomes at or less than 200% of the FPL. Medical conditions that may interfere with baseline body composition (e.g. diabetes, abnormal glucose screen in early pregnancy, hypertension, and eating disorders) were exclusionary.

2.2 Procedures

To accommodate new mothers and improve enrollment within the first several weeks of birth, mothers and their babies were invited to either come to the university clinic or to be assessed in their home. Newborn assessments were completed with 147 mother-child pairs at roughly one month of age, and data for this study are derived from the 132 infants with viable cortisol values at that visit (see Table 1 for Descriptives).

2.3 Measures

2.31 Stress Paradigm

Developmental Challenge Protocol: The Newborn Behavioral Observation (NBO) is a structured set of 18 neurobiological observations designed for infants one to three months of age (Nugent, Keefer, Minear, Johnson, & Blanchard, 2007). The NBO includes two main components: 1) assessment of infant habituation, including sound and light, which is performed while the infant is sleeping (if the infant sleeps at the assessment), and 2) eleven behavioral tests, including assessments of infant rooting, sucking, grasp, crawl, and sit reflexes; orienting to the sounds of voice and rattle; and visual responses to a red ball, face, and voice. The NBO was administered by trained study personnel. NBO administration was completed in 105 (80%) infants, and was terminated early in 27 (20%) infants due to extreme discomfort/fussiness (n=23), drowsiness (n=4), or at maternal request, which resulted in 27 cases missing administration of 1 or more items. We surmised that it was possible, and perhaps likely, that infants who demonstrated sufficient distress to warrant early termination of the measure had reached a threshold stress level that would adequately contribute to the reactivity response. Accordingly, their data was included in our analyses.

The time duration for NBO administration varied because such variation is typical when measures are administered in the home (Luecken et al., 2015), and because some infants

were permitted to feed during a break in the middle of administration if it was required to calm the newborn sufficiently to continue administration. Ranges for NBO time duration and time of day of cortisol administration of the measures are provided in Table 1. Preliminary analyses confirmed that feeding and the time of day of the administration were not related to basal cortisol or reactivity.

Anthropometric Assessment: After completion of the NBO, trained study personnel took anthropometric measurements of the infants, including recumbent length, weight, tricep and subscapular skin fold measurements, and waist circumference. Measurements were taken twice, and taken a third time if values were more than 10% discrepant.

2.32 Cortisol—Consistent with other studies, (Montirosso et al., 2016), salivary cortisol was collected at 3 time points during the visit. A baseline sample (A) was collected after mothers were consented and the mother-child dyad had time to acclimate to the assessment context. A second sample (B) was collected after the NBO and anthropometric measurements were completed (mean time between A and B = 22.2 minutes, SD=4.9) to capture reactivity to the NBO challenges. The third sample (C) was collected approximately 15 minutes after sample B (Mean=15.1 minutes, SD=1.2) to capture additional reactivity after bodily measures and/or potential recovery to baseline. Saliva was collected by trained study personnel using the Salimetrics Infant's Swab (SIS), which was placed in infants' mouths for approximately 30 seconds until saturated, placed in a Swab Storage Tube, and temporarily stored in a −20°C freezer until transported to a −80°C freezer. Samples were then shipped to the University of Dresden (Kirschbaum lab) for assay. After thawing, samples were mixed and centrifuged, and cortisol was assayed using a commercial immunoassay with chemiluminesence detection (Cortisol Luminescence Immunoassy: IBL-Hamburg, Hamburg, Germany; detection limit of 0.179 nmol/l). The intra-assay coefficients of variation were 4.0% – 6.7%, and the corresponding inter-assay coefficients of variation were 7.1% – 9.0%.

Fifteen participants were excluded from analysis involving basal salivary cortisol. Data for cortisol timepoint A were completely missing for 12 participants (1 refusal, 1 undetected, 10 had no sample). One participant with cortisol concentration values exceeding 100 nmol/L was removed due to biological implausibility. There were three participants who had cortisol values that exceeded 40 nmol/L at collection point A, a value reported as a conservative cut-point for cortisol levels (Tollenaar, Jansen, Beijers, Riksen-Walraven, & de Weerth, 2010). Values exceeding 40 nmol/L at baseline were investigated for possible artificial inflation; medical record data was examined for indications that the child was taking a steroid-based medication, such as hydrocortisone cream, which could explain higher cortisol values. After review and consideration of steroid medication half-life, skin absorption rates, and timing of prescription in the medical records, we decided to exclude two participants with cortisol values of 68.57 nmol/L and 58.81 nmol/L at collection point A. If cortisol values at collection point A appeared valid but participants' corresponding point B or C value exceeded the aforementioned cut-point of 40 nmol/L, the values were retained as there was no reason to believe a confound had entered during the protocol. Five additional cases were missing data for timepoint B (2 had no sample, 1 implausible value, 2 for protocol

deviation); and 14 cases were missing data from timepoint C (10 had no sample, 1 implausible value, 3 for protocol deviation). Finally, we also excluded values that were more than 3 SD above the mean.

2.32 Analyses—Cortisol values were normalized using natural log transformation prior to conducting data analyses (McCarthy et al., 2009). Reactivity was calculated twice: as the difference from time point A-to-B ("A-to-B reactivity") and as the difference from time point A-to-C ("A-to-C reactivity"). We did not capture recovery of peak responses with this protocol.

Given that our data is non-normally distributed, and Spearman correlations are more robust to outliers than Pearson correlations, Spearman correlations were used for analyses examining associations between cortisol and demographic variables. Associations are presented as partial correlations adjusted for potential confounds: time of day for all analyses, and time between samples for analyses with reactivity measures.

Finally, in an effort to provide some context to these findings, we averaged the means and standard deviations of data from 29 studies involving infants birth to nine weeks of age included in the Jansen et al. (2010) review. We then conducted a one sample t-test to compare our study findings to the Jensen et al. "population" mean derived from sample size-weighted averages.

3. Results

Study descriptives and raw value ranges for A, B, and C cortisol measurements are provided in Table 1 and Table 2. Table 3 presents correlations with selected variables. Paired t-test analyses showed significant increases on average in cortisol from A-to-C (t (117) =3.90, p<0.001), but not from A-to-B (t (126) =1.44, p=0.15). Over half the infants (59%) showed an increase in cortisol across the protocol and (55%) had a "meaningful" increase in cortisol larger than 10% (Granger et al., 2006) from A-to-C. Infants who terminated the NBO early (see Table 2) showed more A-to-B reactivity (spearman r (125) =.20, p=0.03) than infants who completed the NBO, but otherwise were not different. Difference scores (A-to-B and A-to-C) were used to calculate correlations. Basal and reactivity levels were not significantly correlated with gestational age, birth weight, vaginal-versus-cesarean birth, 5-minute APGAR scores, age at assessment, maternal education, income, maternal marital status or maternal BMI (see Table 3). Similarly, breastfeeding occurring between saliva samples (basal and completion of challenge tasks) was unrelated to basal and post stressor cortisol, and A-to-B and A-to-C reactivity (r's = −0.09 to −.00, p's =0.32 to 0.99). Parity was related to cortisol such that first-born infants had lower basal levels (r (129) =−.34; p=<0.001) and higher A-to-B (r (123) =0.24; p=0.01), and A-to-C reactivity (r (114) =0.21; p=0.02) than infants from multiparous mothers. Females had lower A-to-B reactivity at the trend level (r (123) =−0.17, p = 0.06).

To examine the association between cortisol and ethnicity and race we conducted a series of ANCOVAs, controlling for sampling time-of-day and time-between-samples, as appropriate. Latino infants had lower basal cortisol (F (1, 129) = 5.15, p=0.03), but no differences were

observed in reactivity (F's=0.19, 0.55; *p*'s=0.66, 0.46, for A-to-B and A-to-C, respectively). Three ANCOVAs examining potential differences in cortisol by the three infant race categories were all insignificant (F's=0.37–1.89; *p*'s=0.16–0.69).

One sample t-tests were performed comparing "population" means from Jansen et al. (2010), which were derived from pooling mean estimates across the 29 studies included in that review (see Table 2). Since the current study included 2 post-stressor measurements and 2 reactivity measurements these were each in turn compared with the Jansen pooled values. Basal and post-stressor cortisol, and A-to-B reactivity, from the current study were significantly lower than Jansen et al.'s pooled estimates. A-to-C reactivity was also lower in the current study at a trend level.

4. Discussion

These results contribute to a limited literature base by providing additional information on raw basal and reactive cortisol ranges in a relatively large, predominately high-risk/low-SES sample of young infants born to overweight or obese mothers. In this sample, over half the infants showed a greater than 10% increase in cortisol in response to administration of multi-level, complex challenging stimuli. Overall, despite applying rigorous exclusionary criteria, the ranges and SDs found in our data were fairly large. Given our large, diverse sample with primarily high-risk/low-SES participants, these findings might reflect wider variability in infant responses than typically found in low risk samples, consistent with greater risk of hyper- or hypocortisolism in high-stress environments (Gunnar & Donzella, 2002; Provenzi, Giusti, Fumagalli, et al., 2016; Thompson et al., 2015). Further longitudinal study is necessary to inform our interpretations of these results.

Results showed significant increases on average in cortisol from A-to-C but not from A-to-B. This result makes some sense given that sample B was taken after the completion of the NBO and anthropometric measurements, and sample C was taken 15 minutes later. Given that it takes approximately 20–25 minutes for children's cortisol levels to peak in saliva after a stress exposure (Ramsay & Lewis, 2003), it is likely that the value at timepoint C better captures peak HPA axis response to the NBO challenges.

That infants who terminated early had higher cortisol reactivity from point A-to-B is not surprising, as they usually terminated early because of distress. This variability in exposure to the stress paradigm is a potential limitation, yet perfectly standardized assessment of newborns is not feasible, especially in a community setting using lengthy developmental assessments rather than acute medical procedures (e.g., vaccinations or heel sticks).

Consistent with the sparse literature in this realm (Elhassan et al., 2015), we did not find any association between pre-pregnancy BMI and infant cortisol. Although the restricted range of BMI values in our only overweight and obese sample may have contributed to the null findings in our study (and possibly in Elhassan et al.), we had a broad range of BMI (25 to 44) within our sample and good variability. Nevertheless, additional studies are needed that also include normal weight women. It should also be noted that Elhassan et al. did find that *pregnancy weight gain* was associated with higher morning cortisol and higher cortisol at

recovery from a stressor (girls only) in preschoolers, although another study failed to find such an association in older children (Phillips et al., 2005). Accordingly, it may not be BMI that is relevant for infant cortisol, but rather increases in weight during pregnancy. Much more research is needed in this area to identify whether specific properties of BMI or weight gain influence offspring HPA axis function in infancy.

The parity findings suggest that first-born infants have lower resting cortisol and higher reactivity. One possibility is that first-time birth mothers (who likely lack older children in the household) would have more time, and thus increased opportunities to respond to a newborn in a sensitive and timely manner, which has been found to predict this pattern of lower baseline cortisol and higher reactivity (Blair, Granger, Willoughby, & Kivlighan, 2006).

The finding that Latino infants had lower basal cortisol is consistent with two studies that reported lower levels among young Latino children with high levels of economic and/or psychosocial stress (Fernald, Burke, & Gunnar, 2008; Mendoza, Dmitrieva, Perreira, Hurwich-Reiss, & Watamura, 2017). The trend-level association suggesting that females had lower reactivity is consistent with at least one prior finding in female neonates (Davis & Emory, 1995) and findings reported in one review (Hollanders, van der Voorn, Rotteveel, & Finken, 2017).

There was a robust pattern of findings showing that infants in this study had statistically significant lower levels of cortisol at all three collection time points (basal and post-stress B and C), and lower A-to-B reactivity than the infants in the Jansen et al. (2010) review. The A-to-C reactivity was also lower, though at a trend level. A number of factors might explain the lower levels cortisol displayed by the SEED infants. First, given the higher level of stress often present in low-SES households, the infants in our study may simply have not been as aroused by a research assessment as those from lower-risk households, either due to variation in prenatal programming of the HPA-axis or postnatal environmental differences. Our results are also consistent with prior literature finding an association between low SES and/or parental conflict and low basal cortisol and/or blunted cortisol reactivity in toddlers and children (Sturge-Apple, Davies, Cicchetti, & Manning, 2012; Tackett et al., 2017). Alternatively, about half of the Jansen et al. review studies used a heel stick or an inoculation to stress infants which is thought to produce higher initial and peak cortisol levels than, for example, “learning event” studies (e.g., the Still Face Paradigm (SFP) (Thompson et al., 2015, p. 41), which may have inflated those means. Variation in assay methods might also impact the range and SD of cortisol values and explain differences found here (Jansen et al., 2010). This hypothesis is weakened, however, by fact that the mean levels from Jansen et al. were averaged across many different types of assays. Finally, differences in A–C reactivity may not have reached full significance because of the wide variance of our values.

We note that our data did not include information on napping. Future studies should include this potential confounder given the evidence suggesting an association between sleep and cortisol variability in infants (Egliston, McMahon, & Austin, 2007).

In summary, we report basal and reactivity cortisol levels for approximately one-month old infants born to lower income, ethnically diverse, overweight or obese women, providing much-needed information on levels of cortisol for this developmental period in this understudied population. Though infants presented with a broad range of basal and reactivity levels, very few demographic characteristics were significantly associated with cortisol, suggesting complexity in the prediction of cortisol in early infancy and the role of exogenous factors, such as maternal stress.

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Highlights

- *Infant basal cortisol and reactivity were measured in a low SES/high risk sample;
- *Infant basal cortisol and reactivity were lower than in reviews of low-risk samples;
- *Infant variability in cortisol levels suggests diversity in cortisol outcomes;

Table 1

Descriptive data for analytic sample

Variable	n	Mean (SD) or n (%)
Infant		
Age at assessment (days)	132	37.61 (17.27)
Gestational Age at birth (days)	132	277.59 (8.94)
Birthweight (kg.)	132	4.47 (0.72)
Sex (female)	132	68 (51.52%)
Race	132	
Caucasian		20 (15.15%)
African – American		40 (30.30%)
Multiracial/Other		65 (49.24%)
Not-Reported		7 (5.30%)
Ethnicity (Hispanic or Latino)		59 (44.70%)
Vaginal vs. Caesarian Birth	132	94 (71.21%)
Breast fed vs. non-breastfed	131	103 (78.63%)
Home Assessment vs. Clinic Visit	132	106 (80.30%)
Mothers		
Age (years)	132	28.24 (5.66)
Parity (First born)	132	78 (59.09%)
Marital Status (married or in a committed relationship)	131	88 (67.18%)
*Income	128	$24,418 ($20,301) Range: $0–$86,000
Education	132	
Some High School		12 (9.09%)
High School / GED		28 (21.21%)
Some College, Vocational training or Associate degree		66 (50.00%)
Baccalaureate or Graduate Degree		26 (19.70%)
Maternal Pre-pregnancy BMI	132	30.91 (5.14); Range=25–44
Study Protocol		
Cortisol Sampling (Time of Day)		12:00pm (SD=1.5 hrs.); range=9:00am – 4:30pm
Duration of NBO Testing (minutes)		10.81 (3.5)

*Note: 75% of the sample was at or below the “low income threshold of 200% FPL.

Table 2

Basal cortisol, post-stressor response and cortisol reactivity in response to NBO and anthropometric measures (nmol/L).

Point in time/Calculation	n	Range	Mean(SD) (n	One Sample *t*(df), *p*
A (basal; pre-stressor)	132	0.21 – 41.01	9.52 (8.447)	6.11 (131), <.0001
B (post-stressor paradigm)	129	0.94 – 37.47	10.71 (8.99)	16.78 (128), <.0001
C (2nd post-stressor collection)	121	0.75 – 82.05	15.87 (15.43)	5.79 (120), <.0001
A-to-B (reactivity)				
Overall *	127	−22.46 – 32.42	1.21 (9.47)	9.04 (126), <.0001
[Early Terminators only] *	[24]	[−13.03 to 23.91]	[3.92 (9.23)]	
A-to-C (reactivity)				
Overall *	118	−26.6 – 62.54	5.97 (16.64)	1.86 (117), 0.07
[Early Terminators only] *	[22]	[−17.24 to 62.54]	[13.08 (21.29)]	

* Note: “Overall” values are based upon the full analytic sample. “Early Terminators only” values are based upon the 27 infants who terminated the NBO early due to distress or fatigue. One-sample t-tests were conducted comparing pooled means from Jansen et al. (2010) (Basal (M=14.02), Post-Stressor (M=24.00), and Reactivity (M=8.81)) to SEED sample means described here; variance estimates were unavailable from the Post-Stressor point in time for 3 of those 29 Jansen et al. studies.

Table 3

Spearman Rank Partial Correlations between Sample Characteristics and Basal and Reactivity Cortisol Measures

	Adjusted for Time of Day	Adjusted for Time of Day and Time Between Samples[+]	
	A (basal)	A-to-B (reactivity)	A-to-C (reactivity)
Maternal Age	0.10	−0.03	0.04
Gestational Age	−0.00	−0.06	−0.08
Birthweight (Target Child)	−0.05	0.04	0.02
Sex (Target Child)	0.04	−0.17 ^{t}	−0.09
Vaginal Birth	0.05	−0.06	−0.02
Parity	0.34 ***	−0.24 **	−0.21 *
Married or with Partner	0.01	−0.08	0.03
HH Income	0.04	−0.04	0.02
% Poverty	0.01	−0.01	0.04
Home Assessment	−0.00	−0.06	−0.10

Note: N's range from 118 (for A–C reactivity) to 132 (for basal) due to missing data. For Sex, 1=girl, 0=boy. For Vaginal Birth, 1=Yes, 0=No. For Parity, 1=multiparous, 0=primiparous. For Home Assessment, 1=in family home, 0=in laboratory setting.

^{t} p <.10,

* p < .05,

** p < .01,

*** p < .001.

[+] Correlation between time between samples A and B and reactivity A-to-B = r=0.06, p=0.52, correlation between samples A and C and reactivity A-to-C = r=0.08, p=0.36, however to be conservative these potential confounders were adjusted in analyses.