HHS Public Access

Author manuscript

Infant Behav Dev. Author manuscript; available in PMC 2018 May 01.

Published in final edited form as:

Infant Behav Dev. 2017 May ; 47: 92–102. doi:10.1016/j.infbeh.2017.03.007.

Chronic Stress in the Mother-Infant Dyad: Maternal Hair Cortisol, Infant Salivary Cortisol and Interactional Synchrony

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Abstract

Stress physiology is shaped by early experience, with enduring effects on health. The relation of chronic maternal physiological stress, as indexed by hair cortisol, to infants' stress systems and to mother-infant interaction quality has not been established. We examined maternal hair and salivary cortisol, six-month-old infants' salivary cortisol, and mother-infant interaction in 121 mother-infant dyads. High maternal hair cortisol was related to higher infant average salivary cortisol concentration. Maternal hair cortisol and bedtime salivary cortisol were both uniquely related to infant bedtime salivary cortisol. Mothers with higher hair cortisol were more intrusive and had lower positive engagement synchrony with their infants. Maternal intrusiveness moderated the association of maternal hair cortisol and infant salivary cortisol, such that maternal hair and infant average salivary cortisol were related only when mothers were more intrusive. Maternal chronic physiological stress may upregulate infants' developing stress systems, particularly in the context of lower mother-infant interaction quality.

Keywords

hair cortisol; salivary cortisol; infancy; stress; mother-infant interaction; parent-child relations

1. Introduction

Chronic early life stress has enduring consequences, including risk for poorer physical and mental health in adulthood (Anda et al., 2010). One of the most critical factors in infants' early experience is the dyadic mother-infant interaction. Early caregiving quality predicts language development and cognitive and social functioning (Gunnar and Stone, 1984; Saint-

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Georges et al., 2013). Caregiving quality also is related to infant cortisol function, including reactivity to stressors (Albers et al., 2008; Jahromi, Putnam, and Stifter, 2004). Children who experience early life stress show concurrent and enduring abnormalities in cortisol function (Goldman-Mellor et al., 2012: Lupien et al., 2000). Cortisol dysregulation is a key physiological mechanism through which early life stress leads to adverse long-term health outcomes (McEwen, 2006). The hypothalamic-pituitary-adrenal (HPA) axis, of which the end product is cortisol, is immature at birth. Infants and young children depend on sensitive, responsive caregiving; in the absence of sensitive care, their cortisol function may become dysregulated, exposing the developing brain to excessive cortisol (Gunnar & Talge, 2008).

Thus, it is critical to understand the proximal factors that shape early mother-infant interaction and infant cortisol function. The psychobiological theory of mothering (Barrett & Fleming, 2011) postulates that appropriate mothering requires coordination of multiple systems of perception, cognition, and action to read infant cues and respond sensitively. In mothers who are experiencing high levels of stress themselves due to psychosocial adversity, these systems may be compromised, and thus these mothers are less likely to be sensitive, responsive caregivers (Barrett & Fleming, 2011). Indeed, in a high poverty sample, mothers with higher salivary cortisol levels showed lower sensitivity to their infants (Finegood et al., 2016). Because chronic stress in the early years of life has enduring consequences for infants' later health and well being (Anda et al., 2010), and because early caregiving is critical to buffering infants from stress, chronic *maternal* stress is particularly important to characterize. Yet the role of chronic maternal physiological stress in infant cortisol function and mother-infant interaction quality has not been determined, in part because widely used salivary cortisol measures index acute rather than chronic stress (Russell et al., 2012).

1.1 Comparison of hair cortisol and salivary cortisol

Cortisol is deposited in the hair shaft as the hair grows, such that, taking into account the rate of hair growth, a hair sample provides a timeline of cumulative cortisol exposure over several months (Meyer & Novak, 2012). In contrast, salivary cortisol indexes cortisol concentration at a single point in time. Both these measures have their strengths and weaknesses. Due to diurnal variability in cortisol levels, approximately 70% of salivary cortisol variance is attributable to time-of-day effects (Adam, 2012). While repeated measures of salivary cortisol area under the curve from waking to bedtime (AUCg) have been used to estimate cumulative cortisol exposure, there are large fluctuations in salivary cortisol levels not only across the day but also from one day to the next (Ross et al., 2014). Salivary cortisol levels can be distorted by dairy or caffeine; illness; breastfeeding; and menstrual cycles (Kudielka et al., 2009) and are affected by time of waking and sleep duration (Edwards et al., 2001; Kumari et al., 2009). Thus, there are substantial methodological hurdles to assessing cumulative cortisol exposure with salivary cortisol, even with multiple measures across several days. Compared to salivary cortisol, hair cortisol concentration (HCC) is less versatile: Salivary cortisol can provide information about diurnal rhythm and acute response to challenge, which HCC cannot. However, as an index of chronic stress, HCC solves most of the methodological challenges associated with salivary cortisol, because HCC reflects cumulative exposure over the past several months and is not

unduly influenced by idiosyncrasies of the subject's experience or behavior on the day of sampling.

It is essential to resolve the pattern of association between HCC and well-established salivary measures, so as to integrate the emerging HCC literature with the vast existing salivary cortisol literature. While HCC has rapidly gained popularity in recent years, there is little data addressing the association of HCC with salivary cortisol. In a small sample, van Holland et al. (2012) reported a moderate correlation with mean salivary cortisol concentration (SCC) when saliva was intensively sampled, six samples per day across three days. In a recent study, HCC for 1 cm of hair, corresponding to a one-month period, was highly correlated with mean AUCg from saliva samples collected three times every day during that period, but was unrelated to mean slope or cortisol awakening response (CAR; Short et al., 2016). In a study of pregnant women, HCC for 3 cm of hair, corresponding to a three-month period, correlated with AUCg when the AUCg was calculated from saliva collected three times a day for two different three-day periods during the three-month span (D'Anna-Hernandez et al., 2011). However, HCC and AUCg were unrelated when the AUCg was calculated from a single three-day sampling period. Based on these few studies, it appears that HCC may relate to average SCC and AUCg when these salivary measures are based on numerous samples or when the sampling days are spread out across time.

1.2 Interplay of maternal and infant cortisol and mother-infant interaction

Several studies report associations between mothers' and young children's SCC (Sethre-Hofstad et al., 2002; Stenius et al., 2008). Maternal HCC and child HCC have been concurrently related in older children (Ouellette et al., 2015). In Antarctic seals, fur cortisol concentrations were correlated in mothers and their newborn pups, and were higher in mothers breeding in high density colonies, suggesting possible fetal programming (Meise et al., 2016). In light of the long-term health implications of infant HPA dysregulation, it is critical to examine the role of maternal chronic physiological stress in infants' cortisol exposure.

A key mechanism through which mothers may indirectly shape infant HPA function is caregiving quality. The infant HPA axis is immature at birth, and cortisol function in young children varies depending on the quality of caregiving they receive (Gunnar & Talge, 2008). Low maternal sensitivity is associated with higher infant basal SCC across the day and diurnal AUCg calculated from three samples from waking to bedtime (Letourneau et al., 2011). Low maternal sensitivity has also been related to higher overnight AUCg based on three samples collected from late afternoon until the next morning (Philbrook et al, 2014). If mothers who are chronically physiologically stressed are less responsive to their infants, parenting quality may represent one pathway through which maternal chronic physiological stress could influence infant HPA function. However, the relation of maternal HCC to mother-infant interaction quality has not been established.

The degree of synchrony between maternal and child cortisol may vary depending on caregiving quality. In the salivary cortisol literature, it has been reported that the association of mother and child cortisol response to a stressor is moderated by quality of the mother-child interaction (Sethre-Hofstad, Stansbury, & Rice, 2002; van Bakel & Riksen-Walraven,

2008). Affective involvement has also been shown to moderate the synchrony between mother and child diurnal salivary cortisol (Williams et al., 2013). We are not aware of any studies considering whether the association of maternal HCC with infant cortisol varies by mother-infant interaction quality.

1.3 Hair cortisol and psychosocial stress

It is important to differentiate HCC, a biological index of chronic stress, from widely used indices of perceived stress, depression, and objective psychosocial stressors, in order to inform interpretation of maternal HCC and integrate this research with the vast existing literature on self-reported maternal stress. Mothers of infants are undergoing the stressful transition of adjusting to parenthood or to having an additional child, so parenting stress is particularly relevant to consider. Parenting stress is associated with less sensitive caregiving, insecure attachment, and risk for later socioemotional difficulties in the child (Castel et al., 2016). However, we know of no studies examining parenting stress in relation to HCC. There are several studies of HCC's association with general perceived stress, and findings are equivocal: HCC has been related to both higher (Kalra et al., 2007) and lower perceived stress (Gerber et al., 2013; Karlen et al., 2011), and also found to be unrelated to perceived stress (Braig et al., 2015; O'Brien et al., 2013; Stalder et al., 2012). Wells et al. (2014) reported a curvilinear association, such that HCC initially increased with higher perceived stress but then decreased at the highest perceived stress levels. Thus, the association of HCC and perceived stress may vary as a function of the perceived stress level of the sample studied. Further research is needed to determine whether the inconsistencies in the literature can be attributed to a consistent but non-linear relationship between HCC and perceived stress or instead are due to relative independence of perceived and chronic physiological stress indices.

To our knowledge, research has not assessed relations between depression and HCC of mothers of infants. Studies including a variety of sample populations have shown mixed results (Dettenborn et al., 2012; Dowlati et al., 2010; Faresjö, Jullander, Götmalm, & Theodorsson, 2014; Steudte-Schmiedgen et al., 2017; Stalder et al., 2014; Wei et al., 2015). Compared to matched controls, clinically depressed individuals have been reported to have higher HCC (Dettenborn et al., 2012), lower HCC (Steudte-Schmiedgen et al., 2017) andno difference in HCC (Dowlati et al., 2010). Wei et al., (2015) found that HCC was higher in patients who recently had a first depressive episode compared to recurrent depressive patients and controls, but found no differences between recurrent depressive patients and controls. Again, more research is needed to determine whether the association of depression with HCC may vary by demographics and clinical characteristics of the sample. In sum, the relation of chronic biological stress to perceptions of stress is uncertain and inconsistent: Both perceived stress and depression vary from study to study in whether they are related to HCC and, if so, the direction of association.

HCC does appear to be consistently related to objective psychosocial stressors in human adults, though this has not previously been specifically reported for mothers of infants. HCC increases in relation to objective psychosocial stressors such as unemployment (Dettenborn et al., 2010) and negative life events (Staufenbiel et al., 2014). For example, a recent study in

Kenya reported higher HCC in those who made less than minimum wage and those who reported feeling physically unsafe when collecting water (Henley et al., 2014).

1.7 Current study

We assessed HCC and diurnal salivary cortisol in mothers six months postpartum, and we collected diurnal salivary cortisol from their infants. Mother-infant interaction quality during a home visit was scored using a free play paradigm, yielding indices of maternal intrusiveness and positive engagement synchrony. We also measured parenting stress, depressive symptoms and socioeconomic status. Our objectives were (1) to determine the association of maternal HCC with maternal SCC, and examine how these two maternal cortisol measures relate to infant SCC (2) to investigate mother-infant dyadic interaction in relation to maternal and infant physiological stress, and (3) to describe the association of chronic biological stress, as indexed by maternal HCC, with psychosocial indices of stress. We expected that maternal HCC would be correlated with both maternal and infant SCC, and would be related to lower mother-infant interaction quality. We further anticipated that mother-infant interaction quality would moderate the association of maternal HCC with infant SCC variables, such that the association of maternal HCC with infant SCC would be more pronounced in the context of poorer mother-infant interaction quality. Finally, we expected that maternal HCC would be independent of parenting stress but would be related to socioeconomic status, given prior literature in which HCC is more consistently associated with objective stressors than with self-reported stress.

2. Method

2.1. Participants

Home visits were conducted with 131 mother-infant dyads. Mothers had to provide a usable hair cortisol sample to be included in current analyses, resulting in a final sample size of N = 121 (infant age range 5.86-7.53 months; 62 male). Mothers were primarily Caucasian (80.5%) and most had a college degree or higher (87.4%; see Table 1). Infants had no known hearing, visual, neurological, or developmental disorders and were not from multiple births; mothers were fluent in English. Participants were recruited from a department-maintained database; from publicly available state birth records; from online advertising; and through recruitment events.

2.2. Procedure

This study was approved by the university institutional review board and parents gave informed consent prior to participation. Dyads participated in a home visit when infants were approximately 6 months old (M = 6.67, SD = .43) lasting about one hour, which included infant behavioral assessments, a mother-infant free play interaction, maternal questionnaires, and collection of a maternal hair sample. Additionally, mothers were trained on home salivary collection procedures. After mothers had completed three days of home saliva collection from themselves and their infants, research staff returned to collect the samples.

2.3. Measures

2.3.1. Hair cortisol—Hair cortisol collection procedures followed our validated methods (Davenport, Tiefenbacher, Lutz, Novak, & Meyer, 2006; Meyer et al., 2014). Maternal hair cortisol was measured by sampling a small amount (15-30 mg) of hair, the 3 cm closest to the scalp. Because washing, hair straightening or dying, and styling products may affect HCC (Hoffman et al., 2014), mothers were asked about their hair history including the frequency that the hair got wet. Human scalp hair grows at approximately 1 cm per month (LeBeau et al., 2011), so the 3 cm sample indexed cortisol output over the past 3 months. Hair samples were stored in plastic tubes labeled with subject ID, and were frozen at -20° C until cortisol analysis. Hair samples were weighed, washed twice with isopropanol to remove contaminants, dried, and ground into a fine powder. Cortisol was extracted into methanol, which was then evaporated, and the residue was reconstituted in assay buffer. Reconstituted extracts were analyzed for cortisol using a sensitive and selective commercially available enzyme immunoassay (Salimetrics, LLC; Carlsbad, CA). Assay readout was converted to pg cortisol per mg of dry hair weight. Intra- and inter-assay CVs were less than 2.0% and 5.0% respectively. Raw HCC values were log transformed because the data were not normally distributed. Most mothers (128/131) consented to hair collection. Seven maternal hair samples were excluded, two due to use of steroid medications and five because they had statistically extreme values and were missing information about medication use. This resulted in a final sample of 121 mothers with usable hair cortisol. HCC was unrelated to frequency of washing, Spearman's rho = - .07, $p = .43$, or to color treatment of hair, $t(106) = .39$, $p = .70$. Therefore it was not necessary to control for variation in hair care habits.

2.3.2. Salivary cortisol—Mothers were instructed to collect samples from themselves and their infants immediately upon the infant's waking and at the infant's bedtime (just before the last feeding) on three days when they spent most of the day together and when neither they nor the infant was sick. This diurnal sampling was repeated across 3 days because there are marked day-to-day fluctuations in cortisol levels (Ross et al., 2014). Mothers were instructed to avoid dairy and caffeine prior to sampling, and to sample when it had been at least an hour since the infant last was fed. The bedtime sample was to be collected at least an hour since the infant last napped. Compliance with these instructions was assessed via home diaries, in which mothers also reported the infant's and their own wake times and the times at which each sample was taken.

An infant-safe synthetic swab (Salimetrics, State College, PA) was placed in the infant's mouth for 60 seconds. Mothers also placed a synthetic swab in their own mouths for 60 seconds. Mothers were instructed to keep samples at the back of their freezers until the 3 days of sampling were completed, after which samples were collected by research staff. Once collected, salivary samples were frozen at -20° C until they were sent to Trier Laboratories in Germany for assay.

The majority of participants returned saliva kits (105 out of 131 participants; 80.15%). There were 88 mothers and 85 infants included in the salivary cortisol analyses after sample exclusion. Reasons for sample exclusion included steroid medication use; biologically

implausible values; or collecting waking cortisol more than 60 minutes after waking. Among the included samples, the mean time since wake when the waking sample was taken was 13.01 minutes (SD 9.93) for infants and 18.48 minutes (SD 12.72) for mothers. Because there was variability in the time waking samples were taken relative to the cortisol awakening response (CAR), which peaks approximately 30 minutes after waking, we tested for potential linear or quadratic associations of waking SCC with time since wake, both within each sampling day and averaged across sampling days. For both mothers and infants, time since wake was unrelated to waking SCC, and therefore was not included in subsequent analyses. Wake-up time also was unrelated to waking SCC values. Three variables were calculated to assess diurnal cortisol for both mother and infant, averaged across the 2 or 3 days of sampling: waking SCC, bedtime SCC, and average SCC. Waking SCC and bedtime SCC were calculated if the participant had at least 2 usable samples at that time of day. Average SCC was calculated if the participant had at least 3 usable samples (at least one waking and one bedtime). For mothers, the mean number of samples contributing to the average SCC was 5.34, SD = .97; for infants, the mean number of samples was 5.43, SD = .86. Because cortisol levels typically drop across the day, waking and bedtime values were weighted equally in computing average SCC. Statistically extreme values (> 3 SD from the mean) which were not explained by illness or medication use were winsorized. Variables were then log transformed using a log10 transformation to meet the assumptions of normality (Brummelte et al., 2011; Grunau et al., 2007; Hostinar, Johnson, & Gunnar, 2015).

2.3.3. Mother-Infant Interaction—Mothers were provided with six age-appropriate standardized toys and videotaped during a six-minute free play interaction in the home. Interactions were micro-coded using the methods of Feldman and colleagues (Feldman & Eidelman, 2004; Feldman, Gordon, & Zagoory-Sharon, 2011). Mother and infant direction of gaze, affect, vocalizations and interaction with toys were coded every 30^{th} of a second, frame by frame using Noldus Observer 11.0 (The Vaggenigen, Netherlands). Coders were trained to a kappa of 0.80, and one minute of each interaction was double coded for reliability (kappas .84 - .97). Dyadic variables for analysis - Positive Engagement Synchrony, Maternal Intrusiveness, and Maternal Withdrawal - were then computed from algorithms using Noldus Observer 11.0 software to determine temporal synchronies between infant behavior and maternal behavior. Positive Engagement Synchrony was defined as the proportion of time during the interaction that both mother and infant were simultaneously exhibiting any positive behaviors (Feldman et al., 2011). Maternal positive behaviors included positive affect; infant-directed speech; and gaze to infant. Infant positive behaviors included positive affect; positive vocalizations; and gaze to mother. Thus, the mother smiling while the infant gazed at the mother would be an example of Positive Engagement Synchrony. Maternal Intrusiveness was defined as the proportion of time that the mother was taking away a toy with which the infant was actively engaged. Maternal Withdrawal indexed the proportion of time that the mother averted her gaze from both the infant and the toys while the infant was engaged in the interaction.

2.3.4. Parenting stress—Parenting stress was assessed using the Parenting Stress Index (PSI), 4^{th} Edition short form. Mothers rated 36 items on a 5-point Likert scale which were summed to yield a composite Total Stress scale (possible range 36-180; actual range

46-112). Sample items include “I feel trapped in my responsibilities as a parent” or “Since having a child, I feel that I am almost never able to do things that I like to do.” The 5-point Likert scale responses include: Strongly Agree; Agree; Not Sure; Disagree; Strongly Disagree. The PSI is validated for use with parents of children aged 1 month to 12 years (Cronbach's alpha = .85; Abidin, 1995).

2.3.5. Maternal depression—Maternal depression was measured with the Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff, 1977), a 20-item self-report questionnaire. The CES-D has internal, concurrent, and predictive validity and has been used widely with mothers of infants (Bureau et al., 2009). Possible scores range from 0 to 60, with a clinical cutoff of 16 (Cronbach's alpha = .86).

2.3.6. Socioeconomic status—Mothers reported maternal and paternal occupation; the highest educational level attained by mother and father; household composition; and household income. Occupational prestige was coded using the Job Zone coding scheme from the Occupational Information Network (O*NET, http://www.onetonline.org/help/online/zones), which ranks U. S. Census-based occupational categories on a 1-5 scale based on the education, experience, and training required. Because the sample was highly educated, maternal and paternal education variables were dichotomized based on whether or not parents were college graduates. Income and household composition were used to compute income-to-needs ratio based on the federal poverty level. Maternal and paternal occupational prestige were averaged to yield mean parent occupational prestige. Maternal and paternal education were averaged to yield a mean parent education variable. Finally, parent occupational prestige, parent education, and income-to-needs ratio were standardized and averaged to yield a composite SES variable.

2.4. Analysis plan

Relations between maternal HCC and SCC measures were examined using Pearson correlations. Maternal HCC and SCC measures were then correlated with infant SCC measures. Stepwise regression determined which maternal cortisol variables uniquely accounted for variance in infant SCC variables. To examine the interplay of cortisol and mother-infant interaction, we correlated maternal HCC and infant SCC measures with mother-infant Positive Engagement Synchrony, Maternal Intrusiveness, and Maternal Withdrawal. To test whether associations between maternal HCC and infant salivary cortisol measures were moderated by mother-infant dyadic interaction, ordinary least squares (OLS) regression analyses were conducted using the PROCESS macro in SPSS (Hayes, 2013), examining associations between Maternal HCC and those infant SCC measures which were related to maternal HCC. Positive Engagement Synchrony and Maternal Intrusiveness were tested as moderators. Bias corrected bootstrapped confidence intervals at the 95% level based on 5,000 samples were employed to test for interaction effects, and significant interactions were interpreted based on conditional effects at low and high values of the dyadic interaction variables. Finally, to test the relation of maternal HCC to psychosocial indices of stress, Pearson correlations and t-tests were conducted relating maternal HCC to SES, parenting stress, and depressive symptoms.

3. Results

Descriptive statistics for raw HCC and salivary cortisol measures are provided in Table 2. Correlations among all cortisol and behavioral variables are shown in Table 3.

3.1. Maternal HCC, maternal salivary cortisol, and infant salivary cortisol

Maternal HCC was not related to any measures of maternal salivary cortisol. We next examined how maternal HCC and SCC measures each related to infant SCC. Maternal HCC was related to infant average SCC, $r(83) = .30$, $p = .005$, and to infant bedtime SCC, $r(84) = .50$, $p < .001$. Maternal bedtime SCC also related to infant bedtime SCC, $r(84) = .30$, $p = .006$. Unique maternal cortisol predictors of infant bedtime SCC were identified by regressing infant bedtime SCC on its significant maternal cortisol correlates. Both maternal HCC, $\beta = .49$, $p < .001$, and maternal bedtime SCC, $\beta = .27$, $p = .005$, contributed to predicting infant bedtime SCC, $F(2, 80) = 19.05$, $p < .001$, together accounting for 32.3% of the variance.

3.2. Mother-infant interaction in relation to maternal HCC and infant salivary cortisol

Positive Engagement Synchrony was associated with lower maternal HCC, $r(112) = -.27$, $p = .004$; lower infant average SCC, $r(77) = -.29$, $p = .01$, and lower infant waking SCC, $r(77) = -.25$, $p < .05$. Maternal Intrusiveness was associated with higher maternal HCC, $r(114) = .22$, $p = .02$, but was not significantly related to infant SCC. Maternal Withdrawal was not related to any maternal or infant cortisol variables.

To assess whether the observed associations of maternal HCC and infant SCC were moderated by the mother-infant interaction, OLS regression analyses were conducted relating maternal HCC to infant average SCC and infant bedtime SCC. In each model, Positive Engagement Synchrony and Maternal Intrusiveness were tested as moderators. For the infant average SCC model, the interaction effect of maternal HCC and Positive Engagement Synchrony was not significant, CI: -.75 - 1.08, indicating that Positive Engagement Synchrony did not moderate this association. However, results did support a significant interaction between maternal HCC and Maternal Intrusiveness in relation to infant average SCC, with the 95% confidence interval excluding zero, CI: 2.44 – 18.54. Inclusion of this interaction effect significantly improved the model, R^2 change = .08, $F(1, 70) = 6.75$, $p = .01$, indicating that Maternal Intrusiveness moderated the relation of infant average SCC to maternal HCC. Examination of conditional effects for values of Maternal Intrusiveness 1 SD below and above the mean revealed that maternal HCC and infant average SCC were associated among dyads with high Maternal Intrusiveness relative to this sample, but not for dyads with low Maternal Intrusiveness.

There were no significant interaction effects in the model relating infant bedtime SCC and maternal HCC, indicating that mother-infant dyadic interaction variables did not moderate this association.

3.3. Maternal HCC and psychosocial stress

Finally, we examined how maternal HCC, a physiological index of chronic stress, related to psychosocial indices of stress. Maternal HCC was unrelated to parenting stress or depressive symptoms. Higher maternal HCC was associated with lower SES, $r(119) = -.25$, $p = .007$.

4. Discussion

We examined the relation of maternal hair and salivary cortisol to six-month-old infants' salivary cortisol output, and the interplay of maternal hair and salivary cortisol with mother-infant interaction quality. Maternal HCC strongly and uniquely predicted infant average and bedtime SCC, suggesting the powerful implications of maternal chronic physiological stress for the infant's developing HPA function. Mothers with higher HCC were more intrusive with their infants and had lower positive engagement synchrony. This is, to our knowledge, the first study to demonstrate that maternal HCC is related to less sensitive parenting. Infants depend on their caregivers to help them regulate physiological stress, behavior, and emotion. Maternal chronic physiological stress, indexed by maternal HCC, may impede the mother's ability to provide social buffering of the infant's experience and cortisol exposure during this vulnerable developmental window. In the current study, maternal intrusiveness moderated the association between maternal HCC and infant average SCC, such that this association was present only at higher levels of maternal intrusiveness. Results highlight the complex interplay of maternal and infant cortisol and mother-infant dyadic interaction.

It is striking that maternal HCC, indexing cumulative cortisol output across the past three months, was more closely linked to infant salivary cortisol than were maternal salivary cortisol measures taken on the same days at the same times. Maternal HCC was the only unique predictor of infant average SCC, and both maternal HCC and maternal bedtime SCC independently explained variance in infant bedtime SCC. The HPA axis is immature at birth, and as it develops over the first years of life, the infant is dependent on sensitive, responsive caregiving to prevent excessive exposure to stress hormones (Gunnar & Talge, 2008). It may be that the experience of having a mother who is chronically physiologically stressed upregulates infants' HPA axes, leading to consistently higher cortisol exposure independent of the mother's level of acute stress on any given day.

Attunement of mother and infant salivary cortisol levels is well established in prior literature (e.g. Sethre-Hofstad et al., 2002; Stenius et al., 2008). Physiological attunement has been posited as having evolutionary roots, in that it is adaptive for mothers and infants to respond to each other's cues of external risk. Maternal and infant bedtime SCC were associated in the current study, consistent with the attunement literature. However, maternal and infant waking SCC were not related. This result diverges from other studies that do report attunement of mother-infant samples taken soon after waking (e.g. Bright et al., 2012; Stenius et al., 2008). On the other hand, our results are consistent with Benjamin Neelon et al. (2016), who found that mother and infant SCC were related only at bedtime, not in the morning. Further study is needed to fully characterize what factors account for variation in mother-infant salivary cortisol attunement across samples.

Current results underscore the utility of maternal HCC in explaining individual differences in infant salivary cortisol. However, the mechanisms underlying this association cannot be determined in this cross-sectional correlational study. Mother-infant interaction quality is one of several possible explanations for the association of maternal HCC with infant HPA function, and these mechanisms are not mutually exclusive. There is a genetic contribution to salivary cortisol levels (Kudielka et al., 2009; Wust et al., 2004), and heritability of HCC has been demonstrated in vervet monkeys (Fairbanks et al., 2011). It is possible that this heritable component of cortisol is more pronounced in hair cortisol, because large portions of the variance are not explained by time-of-day and day-to-day fluctuations, as is the case for salivary measures. Fetal programming may also shape infant HPA function. Developmental behavior genetics designs and prenatal measures of maternal HCC would be needed to tease apart the relative contributions of these mechanisms.

In contrast to the close association of maternal HCC and infant salivary cortisol, maternal HCC was unrelated to *maternal* SCC. While van Holland et al. (2012) reported a correlation between HCC and average SCC in adults, they collected six samples a day across three days, as compared to only two samples per day in the current study. Current results suggest that maternal hair and salivary measures provide independent information about different aspects of maternal HPA function. Importantly, maternal bedtime SCC did also uniquely explain variance in infant bedtime SCC. Thus, rather than adopting HCC as a convenient replacement for salivary cortisol measures, it would be ideal for future studies to collect *both* hair and salivary samples so as to obtain a more complete picture of maternal and infant HPA function.

The finding that maternal HCC was related to higher maternal intrusiveness and lower mother-infant positive engagement synchrony supports the possibility that high levels of maternal physiological stress may interfere with sensitive parenting, consistent with a psychobiological theory of mothering (Barrett & Fleming, 2011). However, the opposite direction of causation would also be plausible: When the mother-infant dyad shows low behavioral synchrony, that mismatch may contribute to chronic physiological stress in the mother. We found that maternal intrusiveness moderated the association between maternal HCC and infant average SCC, such that this association was only present at higher levels of maternal intrusiveness. Maternal intrusiveness was defined as taking away toys with which the infant was actively engaged. This could be seen as a lack of dyadic interactional synchrony, in that the mother not accurately reading or attending to the infant's cues. Yet these dyads were actually *more* physiologically in synchrony, with maternal HCC relating to infant average SCC. It will be important for future research to take a nuanced approach to characterizing the interplay of maternal and infant cortisol and behavioral interactions.

Maternal HCC related both to infant average SCC and to infant bedtime SCC, but maternal intrusiveness only moderated the association with infant average SCC. In considering why maternal intrusiveness did not also moderate the maternal HCC – infant bedtime SCC association, it is important to keep in mind that the association of maternal HCC with infant SCC likely reflects shared genetics as well as caregiving. Shared genetics can be conceptualized as a stable, trait-like component to cortisol, whereas maternal intrusiveness and daily shared experiences comprise a situationally specific, state-like component. In

middle childhood, latent state-trait modeling shows that cortisol samples at morning and bedtime reflect both situationally-specific state components and stable trait components (Kertes & van Dulmen, 2012). To our knowledge, this type of analysis hasn't been done with infant cortisol, so the relative contribution of state and trait factors to infant cortisol at different times of day are unknown. Results underscore the need for studies that disentangle genetic, prenatal, and environmental influences of maternal HCC on infant SCC, and suggests that the relative contributions of genetic and environmental factors could vary by time of day.

Maternal HCC was higher in the presence of low socioeconomic status, an objective stressor. It is noteworthy that this association was observed even in a highly educated and relatively affluent sample, underscoring the sensitivity of HCC to this objective stressor. In contrast, maternal HCC was unrelated to parenting stress and depressive symptoms. To our knowledge this was the first study to examine the relation of HCC to parenting stress, a domain of perceived stress that is highly relevant to mothers of infants and has implications for attachment security and infant development (Castel et al., 2016). Prior research on HCC and general perceived stress also has often reported null results (Braig et al., 2015; O'Brien et al., 2013; Stalder et al., 2012). However, future studies should examine whether an association of parenting stress and HCC might emerge among mothers with less education or fewer economic resources at their disposal. While there was no association of depression with HCC, most prior studies compared a clinical sample to matched controls (e.g. Dettenborn et al., 2012) whereas the current study examined depressive symptoms within a community sample. Overall, results are in line with prior research, which consistently reports HCC elevations associated with objective stressors such as unemployment, crowded living conditions, and shift work (Dettenborn et al., 2010; Dettmer et al., 2014; Manenschijn et al., 2011) but is very mixed as to whether there is an association with perceived stress or depression.

The current study is one of very few to compare hair and salivary cortisol indices. An additional strength is that we collected three days of salivary cortisol measures from mothers and infants to obtain more stable salivary cortisol indices accounting for day-to-day fluctuations. This study is to our knowledge the first to examine the relative contributions of both hair and salivary maternal cortisol indices to infant cortisol output, and to assess the implications of elevated maternal HCC for mother-infant interaction quality.

Several limitations must be noted. First, because most of the infants had very little hair, we were unable to examine infant HCC in the current study. This shortcoming could be addressed in future studies by testing older infants or by using fingernail cortisol measurements as an alternative to hair (Warnock et al., 2010). Additionally, as hair cortisol assays become more sensitive, it will be increasingly possible to measure HCC from very small amounts of hair. Second, while mothers were instructed to collect saliva samples from themselves and their infants immediately after waking, the time of sampling varied somewhat, reflecting the challenges of sampling from this population. Mothers often reported that they changed diapers or soothed infants prior to sampling. Given that waking samples were collected on average 15 minutes after waking, most of our waking samples likely reflect the cortisol awakening response to some extent, and may overestimate what

levels would be immediately upon waking. Time since wake did not relate to waking SCC, but it would be ideal for future studies to more tightly control the time of sampling. An additional limitation is that we collected only two samples per day to minimize subject burden. For these reasons, we did not employ salivary measures that capture change across the day, such as slope and AUCg, and instead were limited to SCC at each sampling time and averaged across all samples. Collecting more samples per day would allow slope and AUCg to be examined in relation to HCC, but this would increase subject burden. A final limitation was that the current sample was highly educated, though it is noteworthy that even in this relatively privileged sample, maternal HCC was sensitive to SES. It will be important for future studies to include more diverse samples, so as to examine possible variations in the pattern of mother-infant cortisol associations as a function of SES and culture.

5. Conclusions

This study provides initial evidence that maternal chronic physiological stress, as indexed by hair cortisol, is strongly associated with infant day-to-day salivary cortisol output. Maternal hair and salivary cortisol measures independently contributed to infant HPA activity, highlighting the need to include both hair and salivary measures to fully characterize acute, regulatory, and chronic dimensions of stress physiology. The finding that maternal hair cortisol related to lower positive engagement synchrony and higher maternal intrusiveness suggests that maternal chronic physiological stress has implications not only for infant cortisol function but for also for caregiving quality in the mother-infant dyad.

Acknowledgments

This research was supported by a National Institutes of Health 1R03HD082550 grant to the first author. The authors thank Ryan Johnson and Elizabeth McCollom for assisting with data collection and Kendra Rosenberg for assaying the hair samples. We are grateful to the families who participated.

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Highlights

- Mothers' hair cortisol strongly relates to infants' salivary cortisol levels
- Mothers with high hair cortisol have less sensitive interactions with their infants
- Maternal chronic biological stress matters for infant stress and for caregiving

Table 1
Demographic Characteristics of Participating Families

Maternal age (years) M (SD)	33.41 (4.01)
Infant age (months) M (SD)	6.67 (.43)
Maternal race (%)	
Caucasian	80.5%
Asian	8.5%
Black	5.9%
Hispanic	2.5%
Native American	0.8%
Multiracial	1.7%
Infant race (%)	
Caucasian	70.6%
Asian	4.2%
Black	4.2%
Hispanic	5.9%
Multiracial	15.1%
Income-to-needs ratio M (SD)	5.90 (3.44)
Maternal education (% with at least a 4 year college degree)	87.4%
Paternal education (% with at least a 4 year college degree)	81.9%
Maternal occupational prestige (1-5 scale) M (SD)	3.92 (1.02)
Paternal occupational prestige (1-5 scale) M (SD)	3.87 (1.10)
Paternal co-habitation (% living with mother and infant)	92.6%
Parenting Stress Index Total Score M (SD)	70.98 (12.38)
Maternal depression (% above the clinical cutoff)	14.7%

Note: Possible range for the Parenting Stress Index is 36-180.

Table 2

Raw Hair and Salivary Cortisol Values

	M (SD)
Maternal Waking SCC (μg/dl)	0.26 (0.15)
Infant Waking SCC (μg/dl)	0.38 (0.27)
Maternal Bedtime SCC (μg/dl)	0.05 (0.08)
Infant Bedtime SCC (μg/dl)	0.10 (0.18)
Maternal HCC (pg/mg)	43.39 (102.92)

Table 3

Correlations among All Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Maternal Wake SCC	-	-	-	-	-	-	-	-	-	-	-	-	-
2. Infant Wake SCC	.03	-	-	-	-	-	-	-	-	-	-	-	-
3. Maternal Bed SCC	.21†	.03	-	-	-	-	-	-	-	-	-	-	-
4. Infant Bed SCC	.04	-.01	.30**	-	-	-	-	-	-	-	-	-	-
5. Maternal Average SCC	.92**	.17	.50**	.05	-	-	-	-	-	-	-	-	-
6. Infant Average SCC	03	.92**	.15	.32**	.13	-	-	-	-	-	-	-	-
7. Maternal HCC	.02	.15	.21†	.50***	.11	.30**	-	-	-	-	-	-	-
8. Positive Engagement Synchrony	.06	-.26*	.03	-.10	.05	-.29*	-.27**	-	-	-	-	-	-
9. Maternal Intrusivene ss	.20†	.17	-.04	-.06	-.19	.16	.22*	-.08	-	-	-	-	-
10.Maternal Withdrawa 1	.09	.05	.09	-.17	.11	.01	-.03	.04	.12	-	-	-	-
11.Maternal PSI	.05	.20†	-.11	-.17	-.04	.15	-.11	-.01	.03	-.06	-	-	-
12.Maternal CES-D	.04	-.06	.06	.14	-.04	-.01	.11	.06	.03	-.16	.19†	-	-
13. SES	.02	.17	-.16	-.16	.12	.02	-.22*	.00	-.12	.03	-.06	-.25**	-

†p < .10,

*p < .05,

**p < .01,

***p < .001

Note. SCC = salivary Cortisol concentration; HCC = hair Cortisol concentration; PSI = Total Parenting Stress; CES-D = Depression; SES = socioeconomic status