Infant Behavior & Development 37 (2014) 298–304

Contents lists available at ScienceDirect

Infant Behavior and Development

The effects of SES on infant and maternal diurnal salivary cortisol output

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ARTICLE INFO

Article history:
Received 28 October 2013
Received in revised form 20 January 2014
Accepted 8 April 2014

Keywords:
Cortisol
HPA-axis
Stress
SES
Infancy
Circadian rhythm
Mother–child relations

ABSTRACT

The present study directly compared diurnal salivary cortisol output and maternal-infant synchrony in low and high socio-economic status (SES) mother–infant dyads. Saliva cortisol samples were collected from 32 6–12-month-old infants and their mothers on the same day in the morning, afternoon and evening, and assayed for free cortisol concentration. Low-SES infants and mothers exhibited higher average salivary cortisol output, without dysregulation, compared to high-SES infants. Low-SES infants and mothers also showed reduced synchrony in cortisol output compared to high-SES infants and mothers. Results are discussed with respect to maternal sensitivity and early stress reduction interventions.

Socio-economic status (SES) has a profound effect on physiological systems that are sensitive to the social environment. Many studies confirm that low SES and stress impacts the Hypothalamic-Pituitary-Adrenal (HPA) axis, also known as the stress axis, resulting in an increase in the production of cortisol (e.g., Chen, Cohen, & Miller, 2010; Dickerson & Kemeny, 2004; Kalman & Grahn, 2004). Normatively, cortisol production follows circadian rhythms, peaking early in the morning after awakening, declining over the course of the day, and reaching its lowest levels just before sleep (e.g., Sapolsky, Romero, & Munck, 2000). Exposure to a stressful event typically results in a marked increase in the production of cortisol, and the more quickly the body returns to the resting cortisol rate, the more adaptive the response (e.g., Kirschbaum & Hellhammer, 1989). Generally, cortisol production in response to stressors is a beneficial coping mechanism, but exposure to chronic stress is marked by an overall increase in cortisol production throughout the day (e.g., Fernald & Gunnar, 2009; Lupien, King, Meaney, & McEwen, 2000). That overall increase in cortisol output has been linked to a variety of physical and psychological problems, including anxiety disorders, depression, somatic complaints, aggression, attention problems, cardiovascular disease, respiratory disease, and some types of cancer (e.g., McEwan, 2000; Miller et al., 2009; Santiago, Wadsworth, & Stump, 2011).

Although the relationship between SES and cortisol in adults is complex, several studies have shown that lower SES is associated with higher cortisol output. For example, Cohen, Doyle, and Baum (2006) found decreasing cortisol and epinephrine concentrations with increasing levels of SES, regardless of age, race, gender, and body mass. Similarly, lower SES as indicated

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http://dx.doi.org/10.1016/j.infbeh.2014.04.008
0163-6383/

by education was associated with higher cortisol levels in the evening (Cohen, Schwartz, et al., 2006) and higher SES as measured by job grade was associated with lower average cortisol levels, but only in men (Steptoe et al., 2003; but Decker (2000) reported no relationship in Dominican men and Cohen, Doyle, et al. (2006) and Cohen, Schwartz, et al. (2006) reported a confound between race and SES).

Not only does exposure to chronic stress lead to elevated cortisol levels, but it can also alter cortisol regulation. Maintaining chronically high levels of cortisol output can be characterized by a dysregulated diurnal output pattern, with cortisol levels failing to decrease in the afternoon and evening (Cohen, Schwartz, et al., 2006; Kalman & Grahn, 2004). Indeed, low-SES adults exhibit flatter diurnal rhythms as a result of less of a decline in cortisol output in the evening (Cohen, Doyle, et al., 2006; Cohen, Schwartz, et al., 2006). Alternatively, if the chronic stress is severe and early in life, as in the case of chronic child abuse or neglect, the diurnal pattern is disrupted with blunted cortisol levels in the morning and a flat slope over the course of the day (e.g. Tarullo & Gunnar, 2006).

Low SES adolescents and children also show markedly higher cortisol levels compared to middle and high SES children. For example, Chen et al. (2010) took saliva samples over a two-day period at 6-month intervals over the course of two years in 9–18-year-olds and found that low-SES teens exhibited significantly higher levels of cortisol across the two-year period, producing nearly twice as much cortisol as high-SES children (Chen et al., 2010). Similar results were reported for children at 6 and 8 years of age, where children's cortisol levels were also negatively correlated to family income (Lupien et al., 2000). Both Evans and English (2002) and Evans (2003) reported higher levels of cortisol in low SES 9-year-olds than their high-SES peers. These children also had higher levels of epinephrine and resting blood pressure, which are supplementary indicators of chronic stress exposure.

Living in poverty during childhood has also been linked to long-term elevated cortisol output, even if individuals are no longer in poverty. More time spent living in poverty since birth resulted in higher cortisol levels at age 13 (Evans & Kim, 2007). Miller et al. (2009) also found similar effects in an older age group (25–40 years). Regardless of SES status at time of testing, participants who had lived in poverty at any point during the first five years of life exhibited higher cortisol levels compared to participants who had not (Miller et al., 2009). Miller et al. (2009) suggested that this finding could be evidence for a sensitive period in which basal cortisol output is determined by environmental stressors. The high cortisol output during this period is then "programmed" into the HPA axis and maintained throughout the lifespan (Miller et al., 2009).

One way to explore a sensitive period for basal cortisol output is to study cortisol output in early infancy. Several lines of research have approached this question with respect to SES from different angles. One approach has been to study cortisol responses to a stressful task in either high or low SES babies, without a direct comparison. For example, Haley and Stansbury (2003) collected cortisol from high SES 5- and 6-month-olds before and after a still face procedure, and found a modest increase in cortisol levels in response to the stress. Similar results were reported for exclusively low SES newborns in response to a heel stick procedure (Keenan, Gunthorpe, & Grace, 2007). These studies suggest that cortisol levels are an accurate reflection of immediate stressors in infancy, although they do not speak to whether there are SES differences in output.

A second approach has been to explore the links between an average cortisol level (tested twice a year over several years) and executive function. In a series of studies, Blair and colleagues longitudinally followed a group of predominantly low SES infants, testing cortisol at 7, 15, and 24 months, and also testing parenting, maternal engagement and children's executive function (Blair et al., 2008, 2011; Berry, Blair, Willoughby, & Granger, 2012). They report higher average cortisol levels in low SES children over the first two years, with cortisol mediated by maternal engagement. While these data suggest the importance of studying low SES samples and the long-term stability of cortisol output, they do not test the diurnal pattern or directly compare low and high SES infants.

A third approach has been to study the diurnal pattern in infancy. Indeed, two recent studies indicate that infants as young as 6 months of age do show the diurnal pattern seen in adults (Stenius et al., 2008, 2010). Stenius et al. (2008) tested salivary cortisol levels in middle-to-high income 6-month-old infants three times over the course of a day, and found highest levels in the morning, with levels dropping in the afternoon and then lowest at bedtime. This diurnal pattern is disrupted in older children experiencing significant adversity (with blunted morning response and no change throughout the day, e.g., Tarullo & Gunnar, 2006) but under the adequate-but-not-optimal conditions of lower SES, the disruption has not been found in preschoolers (e.g., Lupien et al., 2000). This has not yet been tested in infants.

Finally, a fourth approach has been to study the synchrony in cortisol levels between infants and their mothers. Synchrony in this case refers to attunement of adreno-corticol function between mothers and their infants. In the above-described studies by Stenius and colleagues (2008, 2010), cortisol samples were also taken from mothers and fathers, along with the infants. Maternal cortisol levels were significantly correlated to infant levels at each collection period, but paternal levels were not (Stenius et al., 2008). And in studies that explore synchrony in response to a specific stressful event, higher maternal-infant synchrony is associated with maternal responsiveness in low-income 4-month-olds (Crockett, Holmes, Granger & Lyons-Ruth) and high-income 4-year-olds (Sethre-Hofstad, Stansbury, & Rice, 2002). But none of the studies exploring either the diurnal pattern or synchrony has compared low and high SES families. While the above studies, along with animal models (e.g., Meaney & Szyf, 2005) suggest that mothers do respond physiologically to their offspring's stress, in the context of a low SES sample, where an elevated cortisol response is predicted in both mothers and infants, it is an open question whether low and high SES mothers will show the same levels of synchrony.

The purpose of the present study was to directly compare low and high SES mother–infant dyads on cortisol sampled several times throughout the day. This comparison should shed light on whether SES affects the diurnal patters of cortisol output in the first year of life. We hypothesized that (a) low-SES infants would exhibit higher average salivary cortisol output,

without dysregulation, compared to high-SES infants and (b) low-SES mothers would also exhibit significantly higher salivary cortisol levels compared to their high-SES counterparts. A second purpose was to examine the synchrony between infant and maternal cortisol by SES. In the absence of any literature on SES and synchrony, we have no specific predictions; the goal was simply to test whether mother–infant salivary cortisol synchrony differed by SES.

1. Method

1.1. Participants

Thirty-two mother–infant dyads participated in this study. Infants were 6–12 months of age ($X = 9.24$ mos, range 6.3–12.2 mos). There were 16 high-SES mothers and infants (11 males, 5 females; 1 Hispanic, 1 Indian, 14 Caucasian) and 16 low-SES mothers and infants (10 males, 6 females; 1 Hispanic, 15 Caucasian). One additional high-SES infant–mother dyad was excluded from data analyses as an outlier. Participants were recruited through advertisements in the local newspaper, community list-servs, flyers posted in town, the Early Head Start program, and word of mouth. Participants were compensated with twenty dollars in gift cards and received a children's book.

Socioeconomic status was determined in two ways. Parents were asked to complete a needs assessment; those who indicated that they qualified for state assistance for housing or food (meaning at or below 185% of the FPL) were categorized as low SES. In addition, parents were also asked about maternal education, where "some college" or more qualified the infant as high SES. This measure of SES was used because parents generally report their education levels more accurately than income, and because maternal education is strongly correlated with both income and SES (e.g., Stevens, Lauinger, & Neville, 2009).

1.2. Materials

1.2.1. Salivary cortisol

This study replicated the methods used by Stenius et al. (2008). Infant salivary cortisol samples were collected using commercial Salimetrics® Infant Swab (SIS) collection kits, and maternal samples were collected using Salimetrics® Oral Swab (SOS) collection kits. Each participant kit included three SIS swabs, three SOS swabs, and 6 labeled storage tubes, and instructions for collection.

1.3. Procedure

1.3.1. Salivary cortisol

Saliva collection kits with written instructions were given to mothers upon a home ($n = 28$) or laboratory visit ($n = 4$) after detailed verbal instructions on when and how to collect the samples. Instructions included a time table for mothers to note exactly when samples were collected. Mothers collected saliva samples from themselves and their infants three times over the course of a single day, in the morning, afternoon, and evening. Morning and evening were defined as "a quarter after awakening and before first meal," and "before going to bed". Afternoon collection times varied between infant and mothers. For infants, it was defined as "after midday sleep" or "one hour after midday meal" if the child did not sleep. For mothers, afternoon was defined as "before dinner, or if dinner was later before 6 PM" (Stenius et al., 2008). During sample collection, mothers were asked to hold the adult oral swab in their mouth until it was soaked with saliva (approximately 90 s). In order to collect the infant sample, mothers were asked to hold the infant oral swab in their infants' mouth until soaked with saliva (approx. 30–60 s). Samples were frozen after collection at the mothers' homes until transported to the laboratory freezer by a researcher and stored at $-20\,^{\circ}$C.

Infant and maternal saliva samples were analyzed for levels of cortisol using commercial Salimetrics® 1-3002 Salivary Assay Kits and a 450 nm single plate reader (Salimetrics®, LLC). In order to prepare frozen saliva for cortisol analysis, samples were thawed and centrifuged. To increase consistency, cortisol controls and participant saliva samples were assayed in duplicate subsamples, and the average was used in all analyses. Saliva samples were then assayed for free cortisol levels according to the manufacturer's instructions using the commercially available Salimetrics® 1-3002 Enzyme Immunoassay Kit (Salimetrics®, LLC). Parent and infant samples from the same family were always analyzed in the same assay. The intra-assay coefficient of variation was less then 4% and the inter-assay coefficient of variation was less than 7%.

2. Results

Cortisol output was analyzed in a mixed model Analysis of Variance (ANOVA), with SES as the between-subjects variable and time of day as the within-subjects variable. Post hoc t-tests (differences between groups at each time point) were performed as needed on the basis of these ANOVA tests. The corresponding analyses were also performed on the diurnal slope and dyad output differences.

Infant cortisol levels are presented in Fig. 1. A 2 (SES: low vs. high) $\times$ 3 (time of day: morning, afternoon and evening) mixed ANOVA revealed a statistically significant effect of time of day, $F(2,30) = 14.688$, $p < .0001$, $\eta^2 = .23$, thus demonstrating the typical circadian rhythm. Post hoc tests show significant differences between morning and noon ($t(31) = 2.88$, $p = .007$), noon and evening ($t(31) = 2.75$, $p = .009$), and morning and evening ($t(31) = 5.22$, $p < .001$). Critically, there was also a main effect for SES, $F(1,30) = 6.542$, $p = .01$, $\eta^2 = .23$, with low SES infants exhibiting overall higher levels of cortisol than high SES infants. Post hoc t-tests comparing low and high SES infant output at each time point revealed a marginally significant difference in the

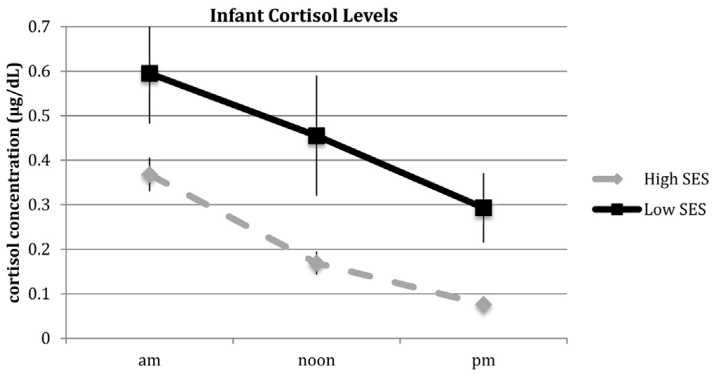

Fig. 1. Infant cortisol levels by SES and time of day.

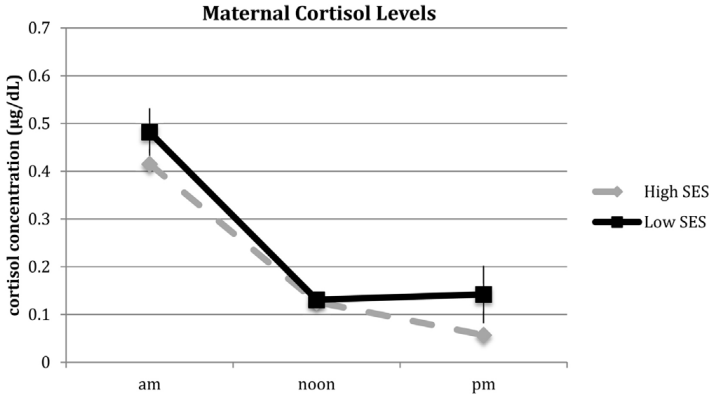

Fig. 2. Maternal cortisol levels by SES and time of day.

morning ($t(30) = 1.91$, $p = .06$), and a statistically significant difference in the afternoon ($t(30) = 2.08$, $p = .04$), and evening ($t(30) = 2.74$, $p = .01$). There was no interaction, $F(2,30) = .232$, $p > .1$, and no SES difference in the diurnal slop ($t(30) = 0.09$, $p > .1$).

Maternal cortisol levels are presented in Fig. 2 and a second 2×3 mixed ANOVA was conducted. Similar to the infants, the mothers showed a main effect for time of day, $F(2,30) = 62.005$, $p < .0001$, $\eta^2 = .57$. Post hoc tests show significant differences between morning and noon ($t(31) = 27.89$, $p < .001$), and morning and evening ($t(31) = 8.89$, $p < .001$) but not noon and evening ($t(31) = 1.4$, $p = .17$). There was also a marginally significant main effect for SES, $F(1,30) = 3.378$, $p = 06$, $\eta^2 = .01$, with low SES mothers exhibiting higher levels of cortisol than high-SES mothers. Follow-up t-tests showed a significant difference between high and low SES mothers in the evening only ($t(31) = 2.54$, $p = .01$). Again, there was no interaction, $F(2,30) = .730$, $p > .1$, and no SES difference in the diurnal slope ($t(31) = 0.22$, $p > .1$).

We explored synchrony in two ways. First, following Crockett, Holmes, Granger, and Lyns-Ruth (2013) and Griffin, Murray, and Gonzalez (1999), we computed the absolute value of the difference between mothers' and infants' cortisol levels, and used this as a dependent variable. Time of day (morning, noon and evening) was a within-subjects independent variable and SES (low or high) was the between-subjects independent variable in a mixed ANOVA. Results indicated a significant interaction between time of day and SES, $F(2,30) = 3.2$, $p = .04$, $\eta^2 = .06$. Post hoc tests show a significant difference by SES in divergence at noon ($t(30) = 1.97$, $p = .05$) but not in the morning ($t(30) = 1.05$, $p > .1$) nor evening ($t(30) = 1.38$, $p > .1$, see Fig. 3). There was also a main effect of time of day ($F(2) = 5.016$, $p < .01$, $\eta^2 = .10$), with significant differences between morning and noon ($t(31) = 2.43$, $p = .02$), noon and evening ($t(31) = 2.08$, $p = .04$), but not morning and evening ($t(31) = 0.83$, $p \geq .1$). Finally, there was also a marginally significant main effect of SES ($F(1) = 3.32$, $p = .07$, $\eta^2 = .11$).

We also ran correlations between maternal and infant cortisol levels separately for each time of day and by SES. High SES dyads were marginally correlated in the morning ($r = .438$, $p = .08$) and significantly correlated in the evening ($r = .585$, $p < .05$) but not correlated in the afternoon ($r = .16$, n.s.). In

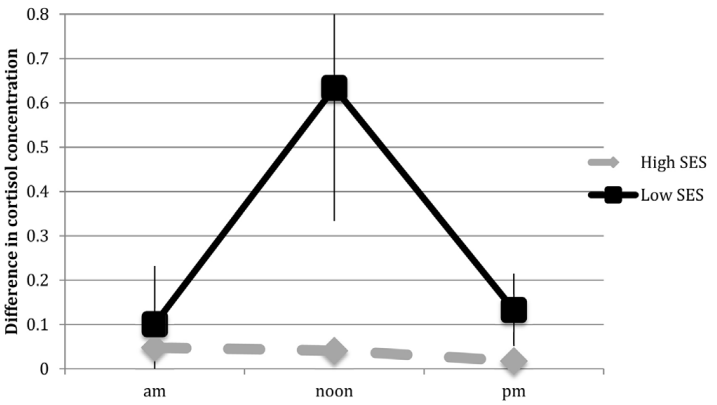

Fig. 3. Mother–infant difference in cortisol levels by SES and time of day.

contrast, the low SES dyads were not significantly correlated at any time of the day (morning: $r = -.12$; afternoon: $r = -.28$; evening: $r = -.31$) and all the correlations were negative.

3. Discussion

The present study compared diurnal salivary cortisol output in high and low SES mother–infant dyads. We hypothesized that both low-SES mothers and infants would exhibit higher average levels of salivary cortisol output compared to their high-SES counterparts. This hypothesis was supported. We further explored the synchrony between infants and mothers, comparing low and high SES mothers, and found more synchrony in the high-SES dyads then the low-SES dyads.

Low SES infants showed significantly higher levels of cortisol throughout the day compared to their high SES peers. Thus, young low-SES infants were responding physiologically to their home environments in similar ways to low SES older children and adults (e.g., Chen et al., 2010, Cohen, Doyle, et al., 2006; Cohen, Schwartz, et al., 2006). However, these infants also showed the typical diurnal pattern of higher levels in the morning, dropping throughout the day, thus showing no dysregulation. Indeed, there were no differences in the diurnal slope between the low- and high-SES infants. This implies that the stressors of living in a low-income home, while enough to increase cortisol output, were not so severe as to disrupt the pattern or blunt cortisol activation (Tarullo & Gunnar, 2006).

In children, chronically elevated levels of cortisol are associated with increased threat responses (Chen et al., 2010), difficulties in socioemotional adjustment (Evans & English, 2002), and poorer scores on attention continuity and speed of memory tests (Maldonado et al., 2008). While there are immediate negative effects of cortisol in childhood, permanently elevated levels of cortisol can also lead to an increased susceptibility to disease, including early mortality (Galobardes, Lynch, & Davey Smith, 2004), resistance to infectious diseases (Cohen, Doyle, Turner, Alper, & Skoner, 2004), cardiovascular disease (Galobardes, Smith, & Lynch, 2006), diabetes (Andrew, Gale, Waler, Secki, & Martyn, 2002), obesity (Power et al., 2005), and major depression (Anisman & Zacharko, 1992). Our results indicate that infants in their first year of life are as sensitive physiologically to these environmental stressors as children and adults.

It is important to note that high cortisol levels can serve an adaptive function for short-term stressors. High amounts of cortisol contribute to the fight or flight response and even in the context of poverty, having heightened cortisol can be adaptive as families face multiple stressors in their environments (Dickerson & Kemeny, 2004). However, high cortisol levels become problematic when the levels are chronically elevated (Kalman & Grahn, 2004). The finding that these differences emerge as early as 6 months of age, and are seen at three time points across one day (and therefore not in response to a specific stressor), could have serious implications for permanent alterations in HPA axis activity. Given the possibility of a sensitive period early in life during which cortisol output trajectories can be permanently altered (Miller et al., 2009), the present results suggest that our sample of infants may be at risk of permanently altered cortisol output in adulthood, regardless of their SES status in adulthood. This certainly warrants further study, specifically longitudinal studies that track the diurnal slope in low-SES infants over the course of the first few years of life.

We also found that low-SES mothers had higher levels of cortisol output, especially in the evening, but again, no SES differences in diurnal slope. This is consistent with previous findings that higher cortisol levels in the evening are associated with the chronic stress of living in poverty (Cohen, Doyle, et al., 2006; Cohen, Schwartz, et al., 2006). Low-SES families are regularly exposed to more cumulative stressors than families who do not live in poverty (Evans, 2004), and the elevated cortisol levels in low-SES mothers show the physiological reactivity that accompanies these stressors. This increased maternal stress can have negative implications for parenting. Prenatal psychological distress in mothers can have detrimental effects on cognitive, behavioral, and psychomotor development in their infants, while postpartum psychological distress in mothers contributes negatively to cognitive and socioemotional development (e.g., Kingston, Tough, & Whitfield, 2012). Chronically high cortisol levels are thought to contribute to or even cause depression, which has also been shown to have adverse effects on parenting in mothers (Field, 2010).

The synchrony data provides a new way to understand the cortisol output in mother–infant dyads. The high SES dyads showed very similar cortisol output at every time of day, and showed strong positive correlations in the morning and evening. Morning levels are thought to be adaptive and evening levels reflect daily stressors; the fact that both of those times are significantly correlated in high SES dyads suggests physiological synchrony. In contrast, the low SES dyads showed more divergence in both measures. Low SES dyads had significantly different cortisol levels in the afternoon compared to high SES dyads and mothers and infants' cortisol levels were negatively correlated at each time of day. In fact, they were increasingly negatively correlated in the afternoon and evening, suggesting that the build-up of daily stress is related to more divergence.

The implications of this reduced synchrony must be speculative for now, given such a small sample and a small number of statistical tests. However, in the context of the literature, these results warrant follow-up studies. One potential avenue for understanding the connection between SES and cortisol synchrony is maternal sensitivity. Previous studies on cortisol synchrony in middle-to-high income pairs (Sethre-Hofstad et al., 2002) or low-income pairs (Crockett et al., 2013) reported higher synchrony in pairs with more sensitive mothers. Moreover, Blair et al. (2008) reported that, in a low-income sample, the effect of what they called 'social advantage' (a way of assessing some aspects of SES) was totally accounted for by maternal engagement. In that study, infants whose mothers were highly engaged had lower cortisol levels in response to a stressful experience. And when those infants were followed longitudinally into toddlerhood (tested at 7 and 15 months), the toddlers' reactivity was associated with maternal engagement in infancy, not toddlerhood. In other words, the toddlers' cortisol response to a stressful event depended on how engaged their mothers were when they were infants, not how

engaged the mothers were at the time of testing (Blair et al., 2008). This finding supports Miller et al's (2009) proposal of a sensitive period in calibrating the HPA axis in infants.

These findings, together with the present results, hint at a potential role for maternal engagement or sensitivity. While maternal engagement wholly accounted for social advantage in Blair et al's (2008) study, that sample was predominantly low-income. Without a direct comparison of different income levels, the true association between SES and maternal engagement is still unclear. There are also questions remaining about directionality. Mothers' lack of attunement may increase or blunt cortisol output, or less sensitive mothers may begin with an impacted HPA system that then blunts their sensitivity. It is also unclear when infant cortisol becomes affected by maternal sensitivity and cortisol output. Are mothers less sensitive to infants who already have elevated cortisol output or is infants' cortisol output increased by less sensitive mothers? There is also a genetic component to cortisol response (Wust et al., 2004), which may impact all of these connections. But given this connection between HPA output and maternal sensitivity, the robust literature on SES and engagement, and the present findings of elevated cortisol and reduced synchrony in low SES mother–infant dyads, future research is needed to untangle these in order to identify a mechanism.

There are, of course, limitations to the study. The small sample size limited the statistical power of our results. Participants were in charge of obtaining their own saliva samples, and samples were assayed by trained researchers rather than by an outside laboratory. Nevertheless, each immunoassay kit included standards that indicated if any saliva samples deviated drastically from the expected range, and none did. Furthermore, the main effects for time of day in both mothers and infants replicated previous studies on cortisol output, suggesting that cortisol collection and analyses were reliable. Finally, inferences about mechanism and the role of maternal engagement are speculative.

Despite these limitations, the present findings suggest that low SES infants' HPA axes are already impacted by SES by 6–12 months of age. Future research should explore the connection among SES, maternal sensitivity and cortisol in order to identify a mechanism. At the same time, clinical research should focus on interventions that target maternal sensitivity, especially in low SES families, and those that target stress reduction, ideally beginning prenatally. Together, these research directions look promising for reducing the potentially devastating long-term impact of chronically elevated cortisol levels.

Acknowledgments

We thank Drs. Tim Machonkin and Tom Knight for invaluable assistance with the assays, and the parents and children who contributed to the study. Portions of these data were presented at the biannual meeting of the Society for the Study of Human Development, Ft Lauderdale, 2013.

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