Maternal Stress Beginning in Infancy May Sensitize Children to Later Stress Exposure: Effects on Cortisol and Behavior*

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Background: Preclinical studies demonstrate that the neonatal environment can permanently alter an individual’s responses to stress. To demonstrate a similar phenomenon in humans, we prospectively examined the relationships of maternal stress beginning in infancy and concurrent stress on preschoolers’ hypothalamic-pituitary-adrenal activity and later mental health symptoms.

Methods: Salivary cortisol levels were assessed in 282 4.5-year-old children and 154 of their siblings. Maternal reports of stress were obtained when the children were ages 1, 4, and 12 months, and again at 4.5 years. Children’s mental health symptoms were assessed in first grade.

Results: A cross-sectional analysis revealed that preschoolers exposed to high levels of concurrent maternal stress had elevated cortisol levels; however, a longitudinal analysis revealed that concurrently stressed children with elevated cortisol also had a history of high maternal stress exposure in infancy. Importantly, children exposed only to high levels of concurrent or early stress had cortisol levels that did not significantly differ from those never exposed to stress. Further analysis of the components of stress indicated that maternal depression beginning in infancy was the most potent predictor of children’s cortisol. We also found that preschoolers with high cortisol levels exhibited greater mental health symptoms in first grade.

Conclusions: These results link the findings of preclinical studies to humans by showing that exposure to early maternal stress may sensitize children’s pituitary-adrenal responses to subsequent stress exposure.

Key Words: Children, cortisol, mental health symptoms, maternal stress, maternal depression, longitudinal

*See accompanying Editorial, in this issue.
Maternal stress result in elevated cortisol levels and dysfunctional behaviors in offspring that are evident later in life (Caldji et al 2000; Coplan et al 1996; Francis et al 1999a; Levine 1957; Liu et al 1997). Furthermore, these effects, which have been observed in rodents (Caldji et al 2000) and primates (Coplan et al 1996), appear to be mediated by disturbances in the quality of mother–infant interaction.

Despite convincing data from animal studies, little is known about the early environmental factors that contribute to individual differences in human HPA function. Numerous retrospective studies of adults and children suggest that increased reactivity of the HPA system is associated with early trauma (Cicchetti and Rogosch 2001; DeBellis et al 1999; Heim et al 2000; Kaufman et al 1997; Yehuda et al 2001) and severe deprivation (Gunnar et al 2001). Studies of less severe stress are predominantly cross-sectional or short-term longitudinal studies. Studies of infants and toddlers suggest that maternal postpartum depression and behaviors (Field 1994) and resulting insecure attachments (Spangler and Grossmann 1993) are positively associated with children’s cortisol levels. In addition, a longitudinal study of children of depressed mothers found that postpartum depression is positively associated with children’s cortisol at 3 years of age (Hessl et al 1998); however, a main effect of maternal depression on cortisol levels was no longer found when the children were age 7 (Ashman et al 2002). Other studies of preschoolers and older children suggest that children’s cortisol levels are positively correlated with numerous concurrent stresses ranging from maternal depression and other social stresses (Lupien et al 2000; Schmidt et al 1997; Tout et al 1998) to broader family characteristics known to be associated with higher stress levels, for example, low socioeconomic status (Lupien et al 2000, 2001). Two recent reviews, however, have noted the inconsistencies in many of the findings regarding early trauma (Bremner and Vermetten 2001) and concurrent stresses (Gunnar and Donzella 2002) and the need for long-term developmental studies. Indeed, what is missing are long-term prospective studies that track the nature and timing of early stress exposure and the linkages to children’s later stress exposure, HPA functioning, and behaviors.

In contrast to earlier studies, this study tracked maternal stress from infancy into childhood, allowing for a more reliable determination of the nature, magnitude, and temporal pattern of children’s exposure to maternal stress. These data allowed us to examine the hypothesis that exposure to high levels of maternal stress during infancy sensitizes children to later stress exposure. We specifically predicted that children exposed to concurrent maternal stress would show increased cortisol and later behavioral symptoms only if they had a history of early stress exposure. In addition to directly linking the preclinical findings to humans, we sought to establish the specific components of maternal stress that were the most potent predictors of increased HPA function. We also considered the possible influence of family socioeconomic status on the association of maternal stress and children’s HPA functioning.

**Methods and Materials**

We recruited 570 women from prenatal clinics. Eligibility requirements included being over age 18, in the second trimester of pregnancy, and, because the original focus of the project was on issues of family and work, to be living with the father and either employed or homemakers (for details, see Hyde et al 1995). Of those who consented to participate, 560 had live births, and 453 provided data at 1, 4, and 12 months after delivery and when the child was 4.5 years old. Of these, 408 still lived within driving distance of the project offices and agreed to participate in a home-based assessment at age 4.5 years; 370 consented to participate in the cortisol collection at that time. Our study focused on the 282 families whose children provided afternoon/evening saliva samples, collected at home by the parents on at least 2 of 3 consecutive collection days. In addition, for the families with multiple children, saliva was similarly collected from the sibling closest in age to the study child. Collecting the saliva samples at home afforded the opportunity to assess basal cortisol levels naturally, and obtaining samples from a sibling afforded the opportunity to replicate select analyses. Parents’ informed consent (as well as permission for their children’s participation at age 4.5 years and in first grade) was obtained at each interview following University IRB-approved procedures. Parental permission to contact teachers was also obtained in first grade, after which the permission of school principals and, where required, district officials was obtained; teachers’ informed consent was obtained at the start of their interviews.

The sample of 282 4.5-year-olds consisted of 135 boys and 147 girls; there were 154 siblings (32 younger and 122 older; 86 boys and 68 girls; age range 2–10 years). At the time of the first interview (pregnancy), the 282 mothers ranged in age from 20–41 years (median age = 30); 95% were married, and 41% were first-time mothers. Two percent had less than a high school degree, 40% were high school graduates, 37% were college graduates, and 21% had some education beyond college. Eleven percent of the families represented ethnic minorities. Family income ranged from less than $10,000 to more than $200,000; median income was $48,500. The 282 families did not differ from the remainder of the original 560 families who had live births with respect to father’s education, family income, marital, or minority status; however, the 282 mothers had significantly more years of education (M = 15.25 years; SD = 2.19) than the mothers who were no longer participating (M = 14.74 years; SD = 2.08). In addition, the 282 families did not differ from the remaining 126 families who participated in the 4.5 year home assessment with respect to maternal stress during the infancy period or the 4.5 year assessment; however, the 282 families had...
significantly higher incomes at the 4.5 year assessment (M = $68,705, SD = $37,585 vs. M = $59,917, SD = $30,229), and the 282 mothers reported significantly less depression symptoms during the infancy period (M = 6.19, SD = 4.96 vs. M = 7.25, SD = 5.29).

Children’s Cortisol Levels

Cortisol was assessed in saliva because it can be noninvasively collected and reflects the plasma concentration of the nonprotein bound active fraction (Kirschbaum and Hellhammer 1994). The target collection time was between 3:00 PM and 7:00 PM (before dinner) because this is a quiescent period of the circadian cycle. Eighty-six percent of the samples were collected within the target period; the time range for all samples was from 12:00 noon to 10:00 PM. Salivary cortisol was assessed with the Pantex (Santa Monica, CA) RIA modified for saliva. The detection limit of the assay (ED₉₀) was .03 μg/dL. The mean interassay and intraassay variation was 7.4% and 3.8%, respectively (for additional details, see Smider et al 2002). To normalize the distribution, raw cortisol values were transformed as the log₁₀ of the median if variation was 7.4% and 3.8%, respectively (for additional details, see Smider et al 2002). To normalize the distribution, raw cortisol values were transformed as the log₁₀ of the median if three values were available or the mean if two values were available. Collection time was coded in 15-min increments; medication use was coded 1 if the child was currently on antibiotics or cold or allergy medications and 0 otherwise.

Maternal Stress

Maternal stress scores were composites of maternal reports of five domains: 1) maternal depression symptoms, 2) family expressed anger, 3) maternal parenting stress, 4) maternal role overload, and 5) financial stress. Except where noted, scores for the infancy period were averaged over the three assessments conducted at 1, 4, and 12 months after delivery; scores for the concurrent preschool period were based on the 4.5-year assessment. Maternal depression was assessed by the Center for Epidemiologic Studies—Depression scale (CES-D; Radloff 1977). Alpha coefficients were greater than .85 across all assessments. Family expressed anger in infancy was assessed with the average of three items from the Partner Role Quality scale tapping overt marital conflict (e.g., concerned about “arguing or fighting”; Barnett and Marshall 1989); at the 4.5-year assessment, the marital conflict scores were combined, using principal components analysis (PCA), with scores from the Anger Expression Inventory (Spielberger 1988) and the Negative subscale of the Family Expressiveness Questionnaire (Halberstadt 1986). Alphas were greater than .80 for the marital conflict scale and greater than .70 for the other two scales across all assessments. The first component of the PCA for the 4.5-year assessment accounted for more than 50% of the variance. Maternal parenting stress was calculated (PCA weights) from scores on the Competence and Child Reinforces Parent subscales of the Parenting Stress Inventory (PSI; Abidin 1986) and the average of three negative items (e.g., “I often feel angry with my child”) from the Childrearing Practices Report (CRPR; Block 1965); in the infancy period, the CRPR was assessed only at 12 months. Alphas were greater than .65 for parent–child negativity, greater than .75 for PSI Competence, and greater than .65 for PSI Child Reinforces Parent across all assessments. The first component of each PCA accounted for more than 50% of the variance. Maternal role overload was calculated (z scores, averaged; z = .48) from the Role Restriction subscale of the PSI (Abidin 1986) and the average of five items from the Role Overload scale (e.g., feeling “pulled apart by conflicting obligations”; Barnett and Marshall 1989); in the infancy period, the Role Overload scale was assessed only at 12 months. Alphas for the two scales were greater than .75 across assessments. Financial stress was the average of four items (e.g., “how much difficulty making monthly payments”). Alphas were greater than .70 across all assessments.

Separately for the infancy and preschool periods, scores for the maternal reports of the five stress domains were combined using PCA. The first component of each PCA accounted for more than 50% of the variance, and all stress domains had factor loadings greater than .50 for each period. Stress groups were then defined according to whether the composite scores were in the lower 25% (low), middle 50% (moderate), or upper 25% (high) of the stress distributions for each period. In addition, longitudinal stress scores were computed to define whether the mothers had been in the high stress group never, in the infancy period only, at the 4.5-year assessment only, or in both periods. Mothers in the high stress group at the 4.5-year assessment were characterized as follows: average CES-D score 14 (consistent with moderate depression; normal ranges 0–8), 60% reported marital conflict, 62% reported difficulties controlling anger, 90% reported negative family expressiveness, 68% reported parenting stress, 53% reported feeling anger toward their children, 93% felt overloaded “occasionally” to “very often,” and 72% felt financially stressed.

Socioeconomic Status

PCA was used to compute family socioeconomic status (SES) indexed by family income during the infancy and preschool periods and by father’s and mother’s education. The first component of the PCA accounted for 56% of the variance. High and low SES were defined as the upper and lower quartiles of the distribution of the resulting composite. Low SES families had median incomes of $35,000 (infancy) and $48,000 (preschool); average parental education was technical or vocational training. Middle SES families had median incomes of $48,000 (infancy) and $60,000 (preschool); average parental education was a college degree. High SES families had median incomes of $70,000 (infancy) and $81,000 (preschool); average parental education was more than a college degree.

Children’s Mental Health Symptoms

Mothers, teachers, and children were interviewed during the spring of the first-grade year about the children’s mental health symptoms during the prior 6 months. Adults completed the Health and Behavior Questionnaire (HBQ; Boyce et al 2002; Essex et al 2002); children were administered the Berkeley Puppet Interview (BPI; Ablow et al 1999; Measelle et al 1998). The mental health symptom items used in our study were derived primarily from the Ontario Child Health Study scales, well-established measures based on DSM-III criteria and with known
reliability and validity (OCHS-R; Boyle et al 1993). For the BPI, the symptom items were reworded to be appropriate for 4- to 8-year-olds (for details of the puppet interview method, see Ablow et al 1999). For our study, the scales for Internalizing Symptoms and Externalizing Symptoms were used. The Internalizing Symptoms scale includes subscales of depression, overanxious, and separation anxiety symptoms (the teacher form excludes separation anxiety). The Externalizing Symptoms scale includes subscales of overt aggression, relational aggression, oppositional defiant behaviors, conduct problems, inattention, and impulsivity. For each symptom scale, the scores of the mothers, teachers, and children were combined using PCA, where the first component represented what the three reports shared in common. For each PCA, the first component accounted for more than 50% of the variance and the factor loadings of each informant were greater than .50. Alpha coefficients for both scales exceeded .80.

Results

Preliminary analyses were conducted to assess the influence of saliva collection time, current use of medications, and child gender on cortisol levels, as well as any gender differences in the associations of maternal stress and cortisol. The results showed that cortisol levels were related to sampling times ($r = -.299, p < .001$), children who were on antibiotics or cold/allergy medications ($n = 59$) had higher cortisol than children on minor or no medications ($F(1,278) = 13.759, p < .001$), and girls had higher cortisol levels than boys ($F(1,278) = 6.038, p = .015$). No other significant gender differences were found. When the 59 children on medications were excluded from the major analyses, no differences in results were found. Therefore, in all analyses collection time, medications, and child gender were included as covariates and adjusted means are reported. Except where noted, multivariate analysis of variance was used for the major analyses. Where significant overall effects are reported, group means are compared using a Bonferroni correction for multiple comparisons.

Initial analyses revealed a significant main effect of concurrent maternal stress [$F(2,276) = 5.508, p = .005$] on children’s cortisol levels at 4.5 years of age. Pairwise comparisons of means revealed that children exposed to high levels of concurrent maternal stress ($n = 69; M = .217, SE = .029$) had significantly higher cortisol levels than children exposed to moderate ($n = 143; M = .139, SE = .020; p = .02$) or low ($n = 70; M = .117, SE = .029; p < .01$) levels of stress (Figure 1). Because of the availability of saliva samples from siblings, it was of interest to establish whether these results would be replicated. Analyses revealed a similar association of exposure to concurrent maternal stress and cortisol levels in the siblings [$F(2,147) = 4.722, p = .010$].

![Figure 1. Afternoon basal cortisol levels (mean ± SEM of the raw cortisol values), adjusted for child gender, time of day, and medications, in 4.5-year-old children and exposure to low (lower 25%), moderate (middle 50%), or high (upper 25%) levels of concurrent maternal stress. *Significantly different from moderate or low levels of stress. No other significant pairwise comparisons.](image-url)

To address the primary hypothesis, we next examined the longitudinal data to determine whether the association of concurrent stress exposure with children’s cortisol levels was carried by children who were initially exposed earlier in life. These and the remaining analyses were conducted only for the preschoolers because longitudinal data beginning in infancy were not available for the siblings. Analyses revealed a significant main effect of the periods of exposure to maternal stress [$F(3,275) = 4.472, p = .004$] on children’s cortisol levels. Pairwise comparisons of means revealed that children who were exposed to high levels of concurrent maternal stress who were initially exposed to high maternal stress in infancy (i.e., exposure in both periods; $n = 40; M = .272, SE = .038$) were the only group that had significantly higher cortisol levels than children never exposed to maternal stress ($n = 182; M = .124, SE = .018; p < .01$; Figure 2). No pairwise comparisons were significant for children exposed in infancy only ($n = 31; M = .178, SE = .043$) or at age 4.5 only ($n = 29; M = .140, SE = .044$). These results suggest that exposure to concurrent maternal stress is associated with increased cortisol levels only for children whose exposure began in infancy and that exposure to maternal stress in only one period is not associated with increased cortisol.

The next analyses addressed the question of the association of cortisol and dysregulated behavior. The longitudinal data provided the opportunity to consider the association of preschool cortisol levels and the development of mental health symptoms after the normative challenge of
the transition into school. The results of partial correlations showed that the preschoolers with high cortisol levels evidenced greater internalizing ($r = .208, p = .004$) and externalizing ($r = .214, p = .003$) symptoms more than two years later at the end of first grade. Because the internalizing and externalizing symptom scores were moderately correlated ($r = .484, p < .001$), we also conducted two multiple regression analyses with each symptom score as the dependent variable controlling on the other. The association of cortisol and internalizing symptoms was reduced to marginal significance ($p = .056$), and the association of cortisol and externalizing symptoms was reduced to nonsignificance ($p = .107$). These results suggest that cortisol may be a little more closely associated with internalizing symptoms, especially as they co-occur with externalizing symptoms. Thus, we summed the separate symptom scores to form a total score where a high score reflects the co-occurrence of internalizing and externalizing symptoms. When children were categorized according to whether their cortisol levels were low (lower 25%, $n = 42$), moderate (middle 50%, $n = 110$), or high (upper 25%, $n = 46$), analyses revealed a significant main effect for the cortisol groups $[F(2,192) = 4.010, p = .020]$. Children with high cortisol levels at 4.5 years of age ($n = 46; M = .450, SE = .233$) had symptom levels in first grade that were significantly greater than those for children with moderate ($n = 110; M = -.250, SE = .148; p = .034$) or low ($n = 42; M = -.437, SE = .260; p = .046$) levels of cortisol (Figure 3). Furthermore, when the longitudinal maternal stress variable was included in the analysis, significant main effects were revealed for both cortisol $[F(2,186) = 3.374, p = .036]$ and maternal stress $[F(2,186) = 3.102, p = .047]$, and there was no significant interaction effect. This suggests that although they are correlated, children’s cortisol and exposure to maternal stress also make unique contributions to the prediction of children’s later mental health symptoms.

Additional analyses were conducted to investigate the role of SES and its influence on the association of maternal stress and children’s cortisol levels. Analyses revealed a significant main effect of SES $[F(2,276) = 4.161, p = .017]$ on cortisol levels. Pairwise comparisons revealed that the 4.5-year-old children living in low SES families ($n = 68; M = .223, SE = .029$) had significantly higher cortisol levels than those living in high SES families ($n = 62; M = .120, SE = .031; p = .013$); the cortisol levels of children living in middle SES families ($n = 152; M = .135, SE = .019$) were in between and not significantly different from either of the other two groups. Further analyses revealed that compared with the children living in middle or high SES families, children living in low SES families were exposed to higher levels of maternal stress both in infancy $[F(2,278) = 5.011, p = .007]$ and at age 4.5 $[F(2,278) = 4.184, p = .016]$; however, when both variables were considered together, analyses revealed significant main effects of both SES $[F(2,270) = 5.569, p < .01]$ and exposure to maternal stress $[F(2,270) = 3.901, p = .021]$, suggesting that the effects of early and concurrent high maternal stress on children’s cortisol levels are not due to its association with SES.
Figure 4. Afternoon basal cortisol levels (mean + SEM of the raw cortisol values), adjusted for child gender, time of day, and medications, in 4.5-year-old children and exposure to clinically significant maternal depression (Center for Epidemiologic Studies—Depression scale ≥ 16) never, in infancy only, at age 4.5 only, or in both periods. *Significantly different from exposure only at age 4.5 or never. No other significant pairwise comparisons.

The final set of analyses focused on identifying which components of maternal stress are the most potent in the development of individual differences in cortisol. The results of a stepwise multiple regression analysis of the five components of maternal stress showed that maternal depression was the only stress domain during infancy that was a significant independent predictor of later cortisol ($\beta = .123, p = .027$). Because the associations among the stress domains were sufficient to combine them into a composite stress score, we also considered the possibility of multicollinearity problems; however, the correlations were not so high (ranging from .10–.37) as to raise significant concerns about this issue.

We then identified 37 mothers who had clinically significant depressive symptoms (i.e., CES-D scores of 16 or higher; Radloff 1977) only when their children were infants, 4.5 years old, or in both periods and performed the same primary analysis as we did for maternal stress. Analyses revealed a significant main effect of the periods of exposure to maternal depression ($F(3,275) = 2.987, p = .032$). Pairwise comparisons of means revealed that children who were initially exposed to maternal depression in infancy who were also exposed concurrently (i.e., exposure in both periods) had marginally significantly higher cortisol levels ($n = 6; M = .534, SE = .097$) than children never exposed to maternal depression ($n = 245; M = .141, SE = .015; p = .09$) or exposed only concurrently ($n = 18; M = .112, SE = .056; p = .09$; Figure 4). There were no significant pairwise comparisons for children exposed only in infancy ($n = 13$). These results suggest that, similar to those for maternal stress, it is the combination of initial exposure to maternal depression in infancy and concurrent exposure that is most significantly associated with children’s elevated cortisol levels.

**Discussion**

Using a longitudinal design with repeated assessments of maternal stress, our data suggest that maternal stress beginning in infancy predisposes children to increased HPA function during a period of concurrent stress. Importantly, the data show that children exposed to high levels of concurrent stress without a history of early stress exposure did not have elevated cortisol levels. Furthermore, cortisol levels were not elevated in children who were exposed only to high levels of early stress. Our results also highlight maternal depression in infancy as the facet of maternal stress that is the most potent in relation to children’s later cortisol levels. Although there was only a very small group of children who were exposed to clinically significant maternal depression in both periods, yielding marginally significant results when corrected for multiple testing, the results were similar to those found for stress: it was the combination of exposure to maternal depression in infancy and concurrently that yielded the highest levels of children’s cortisol. Previous studies have demonstrated positive associations between concurrent stress exposure and children’s cortisol levels (Gunnar and Donzella 2002; Lupien et al 2000, 2001; Schmidt et al 1997; Tout et al 1998). Numerous retrospective studies have demonstrated that early and severe trauma or deprivation are associated with elevated cortisol levels (Cicchetti and Rogosch 2001; Heim et al 2000; Yehuda et al 2001). Furthermore, it has recently been suggested that duration of abuse or deprivation may be important (De-Bellis et al 1999; Gunnar et al 2001). In addition, in a study of children with major depression, Kaufman et al (1997) found that early abuse combined with chronic adversity may result in increased stimulated corticotropin release, although cortisol levels were not elevated; however, prior studies have not been able to look prospectively at the combination of early and concurrent stress exposure to discern individual differences in children’s later cortisol levels.

We also found that children with high levels of cortisol at age 4.5 are at risk for mental health symptoms at the end of first grade when they have completed the normative challenge of the transition into school. This extends aspects of our previous findings in this same sample of children, which demonstrated reporter-specific (mother,
father, teacher) associations of children’s cortisol at age 4.5 with emotional and behavioral difficulties earlier in the school transition period when the children were in kindergarten (Smider et al 2002). Most relevant to the work described here are the previous findings demonstrating that preschoolers with elevated cortisol levels were reported by both mothers and teachers to have greater withdrawal-type problems (e.g., social withdrawal) in kindergarten. Together with our findings, which are also based on mother and teacher reports (fathers were not included in the current multiformal approach), this suggests that preschoolers with elevated cortisol levels are likely to experience greater emotional and behavioral difficulties throughout the school transition period.

Our findings regarding the relation between SES, maternal stress, and children’s cortisol confirm and extend the results of a recent study that showed that children as young as 6 years of age who are from low SES families have higher cortisol levels than children from high SES families and that SES partially accounts for the association of concurrent maternal depression and children’s cortisol levels (Lupien et al 2000). In our study, we found that the relation of SES and children’s cortisol levels extends to children as young as 4.5 years of age. Furthermore, our findings extend those regarding concurrent maternal depression to a longitudinal focus on children’s exposure to maternal stress beginning in infancy, showing that SES does not account for the effects on children’s cortisol levels of early and concurrent exposure to maternal stress.

Overall, our findings are consistent with animal studies demonstrating that neonatal stress and maternal behaviors and experience can strongly influence later HPA function and behavior in offspring (Anisman et al 1998; Bakshi and Kalin 2000; Caldi et al 2000; Coplan et al 1996; Francis et al 1999a; Kalin et al 1998; Liu et al 1997; Takahashi and Kalin 1991). For example, rat mothers that engage in high amounts of licking and grooming with their pups have offspring that, as adults, are less hormonally and behaviorally responsive to stress. The opposite is true for mothers that engage in low levels of licking and grooming. Furthermore, it has been demonstrated that this maternal behavior mechanistically underlies these differences in offspring (Frances et al 1999b). In humans, it has been shown that early maternal stress, and especially postpartum depression, also interferes with mother–infant interactions, and it is the lack of maternal sensitivity and responsiveness that are most closely associated with several concurrent parameters of the infants’ physiologic reactivity, including cortisol levels (Dawson and Ashman 2000; Field 1994). Therefore, it is likely that in our sample, altered mother–infant interactions secondary to mothers’ high levels of stress and depression mediated the effects on children’s later cortisol and behavior.

Although our study suggests that early exposure to stress sensitizes later HPA and behavioral responsivity, design limitations do not allow causal interpretations. It is also likely that genetics play a role (Young et al 2000); however, our data and the findings from other studies strongly suggest that early environmental conditions are especially important. Compelling evidence is provided by rodent cross-fostering studies documenting a clear mechanistic role for maternal behavior in the absence of genomic influences (Frances et al 1999b).

In summary, our findings suggest that exposure to maternal stress, and especially maternal depression, during infancy may increase the vulnerability of the developing child’s HPA system to later stress exposure. Furthermore, elevated cortisol levels appear to predict the later development of dysregulated behavior associated with psychiatric illness. Because cortisol has been linked to numerous pathophysiologic processes, our findings may have broad relevance to understanding factors that influence lifelong patterns of health and disease.

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