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Increased Waking Salivary Cortisol and Depression Risk in Preschoolers: The Role of Maternal History of Melancholic Depression and Early Child Temperament

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Abstract

Background: Elevated morning cortisol is a prospective predictor of major depression and may serve as a vulnerability marker. We examined the relation between morning cortisol and two prominent risk factors for depression in preschool-aged children: maternal depression and child temperament. We also explored whether maternal depression during the child's life, parental hostility and life stress explained these associations.

Methods: 94 children provided a morning salivary cortisol sample, and 92 children provided an evening sample. Child temperament and parenting were assessed using observational measures, and maternal depression and life stress were assessed with clinical interviews.

Results: Maternal history of melancholic depression and child temperamental low positive emotionality were significantly associated with higher morning cortisol. These relations persisted after controlling for children's negative emotionality and concurrent depressive symptoms, parental hostility, and life stress.

Conclusions: Our findings support the hypothesis that elevated morning cortisol may serve as an early-emerging vulnerability factor for depression, and highlight the importance of anhedonia in risk for depression.

Keywords

Depression risk; melancholia; HPA-axis; cortisol; temperament

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Key Points

- Research suggests that elevated waking cortisol is a prospective predictor of MDD and may serve as a potential vulnerability marker.
- This study found that elevated waking cortisol was associated with maternal melancholic depression and child temperamental low PE in preschoolers prior to any depressive disorder.
- Our findings underscore the relation between hypercortisolism and anhedonic traits.
- Future research should continue to investigate this potential vulnerability marker as our findings have implications for intervention and prevention with young children at risk for depression.

Depression has been consistently associated with abnormalities in the hypothalamic-pituitary-adrenal (HPA) axis, one of the body's major stress-response systems (Gold, Goodwin, & Chrousos, 1988). Hypercortisolemia is evident in approximately 50% of depressed patients and is particularly characteristic of the melancholic subtype (Thase, Jindal, & Howland, 2002). In addition, pre-existing differences in HPA-axis functioning may underlie the “stress sensitivity” observed in depression (Gotlib, Joormann, Minor, & Hallmayer, 2008; Holsboer, 2000), which render certain individuals more susceptible to the depressogenic effects of stress.

Evidence suggests that elevated morning cortisol, in particular, is a prospective predictor of major depressive disorder (MDD), and may serve as a trait vulnerability marker for depression (Adam, Sutton, Doane, & Mineka, 2008). Twin studies suggest that it is moderately heritable (Bartels, Geus, Kirschbaum, Sluyter, & Boomsma, 2003; Wust, Federenko, Hellhammer, & Kirschbaum, 2000). Elevated morning cortisol has been found not only in acutely depressed patients (Bhagwagar, Hafizi, & Cowen, 2005) and recovered depressed patients (Bhagwagar, Hafizi, & Cowen, 2003), but also in the offspring of depressed parents (Halligan, Herbert, Goodyer, & Murray, 2004; Mannie, Harmer, & Cowen, 2007; Young, Vazquez, Jiang, & Pfeffer, 2006). Moreover, prospective studies found that elevated morning cortisol predicts the onset of MDD in youths (Goodyer, Herbert, Tamplin, & Altham, 2000) and adults (Harris et al., 2000). However, little is known about what factors are associated with, or may account for, increased waking salivary cortisol or at how young an age it can be observed.

Demonstrating associations between better established vulnerability factors for depression and morning cortisol would strengthen the argument for elevated morning cortisol as an early-emerging vulnerability marker and could provide clues regarding possible mechanisms. This study examines the relation between morning cortisol and two vulnerability factors for depression in a community sample of preschool-aged children: maternal history of depression and early child temperament.

First, we examine whether maternal history of depression is associated with elevated morning cortisol in preschoolers. Both animal and human studies suggest that early perturbations of HPA-axis functioning can have enduring effects (Heim et al., 2000; Lehmann, Russig, Feldon, & Pryce, 2002). Previous studies have found associations between maternal depression and morning cortisol in adolescents and prepubertal children (Halligan et al., 2004; Mannie et al., 2007; Young et al., 2006); however, establishing an association in younger offspring has important implications for tracing the developmental pathway from HPA-axis abnormalities to later depression.

We also explore whether children of mothers with histories of melancholic depression are particularly likely to exhibit elevated morning cortisol, as melancholia is the subtype of depression that is most frequently associated with hypercortisolemia (e.g., Gold et al., 1986; Kupfer, 1991; Rush & Weissenburger, 1994; Wong et al., 2000). These analyses are exploratory given the small number of mothers with melancholia in our sample; however, to our knowledge no previous studies have examined the association between specific subtypes of maternal depression and offspring neuroendocrine function.

In addition, we examine whether children's elevated morning cortisol levels are due to the direct effects of a familial, possibly genetic, vulnerability for depression or to exposure to maternal depression and associated disruptions in parenting and life stress (e.g., Ashman, Dawson, Panagiotides, Yamada, & Wilkinson, 2002; Essex, Klein, Cho & Kalin, 2002), as these factors are often confounded. Particularly, we are interested in whether the timing of maternal depression (i.e., during prenatal or postnatal developmental periods), impacts

children's neuroendocrine functioning, as prior studies have suggested that such an association may exist, although methods and specific findings have varied (Ashman et al., 2002; Essex et al., 2002; Halligan et al., 2004; Young et al., 2006).

Second, theorists have long posited that the predisposition for depressive disorders is rooted in individual differences in temperament (e.g., Akiskal, 1989). Clark, Watson, and Mineka (1994) hypothesized that low positive emotionality (PE) and high negative emotionality (NE) are precursors to depressive disorders. In support of these theories, a few prospective studies have found that low levels of PE-related behaviors and high NE predict the development of depressive disorders (Caspi, Moffitt, Newman, & Silva, 1996; Hirschfeld et al., 1989; Kendler, Gatz, Gardner, & Pederson, 2006; van Os, Jones, Lewis, Wadsworth, & Murray, 1997).

There is evidence that both low PE and high NE are associated with increased morning cortisol in adults (e.g., Portella, Harmer, Flint, Cowen, & Goodwin, 2005; Steptoe, Gibson, Hamer, & Wardle, 2007), and NE-related constructs have been associated with increased morning cortisol in children (Kagan, Reznick, & Snidman, 1987). However, no studies to our knowledge have examined low PE and elevated morning cortisol in children. This is an important gap given the central role of anhedonia (which is characterized by low PE) in melancholic depression, and the association between melancholia and cortisol dysregulation, which has been observed even in young children (Luby et al., 2003).

In the current study, children's morning and evening cortisol were assessed on one occasion. We recognize that cortisol levels vary, and sampling over several days is preferred (Gunnar & Talge, 2007). However, given the demands of the larger project from which this sample was obtained, we felt that one occasion of sampling would be acceptable for several reasons. Studies have shown that peak cortisol secretion 30 minutes after awakening is a reliable index of adrenocortical activity in children and adults (Pruessner et al., 1997) and has greater test-retest stability than sampling based on fixed times (Wust et al., 2000). Furthermore, moderate stability ($r = 0.53$) has been demonstrated for morning cortisol, particularly when the state of the child is controlled prior to sampling (Goldberg et al., 2003).

In sum, we examine the relation between two prominent risk factors for depression and waking cortisol. We hypothesize that maternal depression history, particularly the melancholic subtype, and child temperamental low PE and/or high NE are associated with children's elevated morning cortisol even when several psychosocial factors are controlled. In addition, we hypothesize that the relation between elevated cortisol and depression risk is specific to morning cortisol and will not be observed for evening cortisol.

Method

Participants

A consecutive series of 166 children were recruited from a larger community sample participating in a study of temperament and risk for depression ($N = 559$). Families of young children were identified using a commercial mailing list. Children between the ages of 3 and 4 years, with no significant medical or developmental disabilities, and who lived with at least one biological parent were eligible.

Of the 166 families asked to participate in the cortisol assessment, 92 children provided a morning and evening cortisol sample (87 of which were collected on the same day), and two additional children provided morning samples only, yielding a total of 94 children (41 females, 53 males). We compared parent and child characteristics of those who provided

cortisol assessments to those who did not using t-tests. We found that non-participating parents were rated as higher on parental hostility and non-participating children had experienced more stressful life events prior to the assessment compared to participating families. No children in the sample met criteria for a mood disorder as assessed using the Preschool-Age Psychiatric Assessment (PAPA; Egger, Ascher, & Angold, 1999). Children were of average cognitive ability as indexed by the Peabody Picture Vocabulary Test ($M = 104.42$, $SD = 13.62$; PPVT; Dunn & Dunn, 1997). Written informed consent was obtained by parents after the study procedures had been fully explained. The demographic characteristics of the sample are presented in Table 1.

Measures

Maternal Depression—The Structured Clinical Interview for DSM-IV (SCID; First, Gibbon, Spitzer, & Williams, 1996) was conducted with biological mothers to assess lifetime depressive disorders (MDD or dysthymic disorder (DD)). No mother met criteria for lifetime bipolar disorders. If lifetime depression was present, the onset and offset dates of all episodes were determined. Similar to Ashman et al. (2002), we created variables that indicated whether mothers were depressed during the child's gestation, the child's first two years, and/or the child's 3rd-4th years. Variables for each developmental period, reflecting whether or not the mother was depressed at any point during that period, were coded dichotomously (absent/present). Interrater reliability (indexed by the kappa) for lifetime depression was .93, based on 30 audiotaped interviews. Rates of lifetime depression are presented in Table 1. In addition, 82 mothers completed the Diagnostic Inventory for Depression (DID; Zimmerman, Sheeran, & Young, 2004; $\alpha = .89$; $M = 4.61$, $SD = 4.31$) to assess the influence of current maternal depressive symptoms on children's cortisol.

Laboratory Temperament Assessment—Children participated with a female experimenter in 12 standardized tasks selected from the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith, Reilly, Lemery, Longley, & Prescott, 1995), during which children were videotaped. The tasks were designed to elicit behavioral expressions of a range of temperamental traits. Mothers were present in the room for all episodes, with two exceptions noted. Episodes are described in the order in which they were conducted: (1) *Risk Room* – child played freely with novel and ambiguous stimuli (e.g., cloth tunnel, small staircase, mattress, Halloween mask, balance beam), followed by the experimenter asking the child to approach each object; (2) *Tower of Patience* – child and experimenter took turns building a tower of cardboard blocks according to a schedule of increasing delays, forcing the child to wait longer between turns; (3) *Arc of Toys* – child played freely in a room full of toys and then was asked to clean up; (4) *Stranger Approach* – a male research assistant entered the room where the child had been left alone, speaking to the child while gradually walking closer; (5) *Make That Car Go* – experimenter and child raced two remote-controlled racecars; (6) *Transparent Box* – child was given inoperable keys to open a transparent box that contained an appealing toy; (7) *Pop-Up Snakes* – experimenter and child played a practical joke on the mother, asking her to open what appeared to be a can of potato chips, but actually contained coiled spring snakes; (8) *Impossibly Perfect Green Circles* – experimenter asked the child to draw several circles, mildly criticizing each one, and asking the child to draw another; (9) *Popping Bubbles* – experimenter and child played together with a bubble-shooting toy; (10) *Exploring New Objects* – child was given an opportunity to explore new objects (tent, pet carrier, “goosey” toys, remote-controlled spider, plastic head covered with a red cloth); (11) *Snack Delay* – child was instructed to wait for the experimenter to ring a bell before eating a snack; (12) *Box Empty* – child was left alone with a wrapped empty box to open, under the pretense that an appealing toy was inside.

Coding of temperament data—Facial, bodily and vocal indicators of positive affect (PA), fear, sadness and anger, as well as several additional temperament-relevant behaviors such as interest and anticipatory PA, were coded during each episode, regardless of the emotion an episode was designed to elicit. This coding scheme captured atypical emotional behaviors, while also increasing scale reliability by generating affect ratings for all tasks. Each relevant display of facial, vocal, and bodily affect was coded on a three-point intensity scale. Ratings were summed within each episode, the totals for the 12 episodes were then totaled, and the three channels were aggregated to create scores for PA, sadness, fear, and anger reflecting facial, vocal, and bodily indicators.

Interest and anticipatory PA were also coded using a four-point intensity scale. A single rating was made per episode based on all relevant behaviors during that episode. Interest ratings were based on the child's comments about the activity and how engaged the child was in play. Anticipatory PA was defined as the expression of PA in anticipation of a positive event/reward.

Analyses used aggregated PE and NE scores. PE consisted of the average of z-scores for facial, bodily, and vocal PA, including anticipatory PA and interest across all episodes. NE consisted of the average of z-scores for facial, bodily, and vocal anger, sadness, and fear. Internal consistencies for PE and NE scales were .83 and .82, respectively. Interrater reliability, as indexed by the intraclass correlation (ICC, $N = 35$), was .89 and .74 for PE and NE, respectively. PE and NE scores were normally distributed across the sample.

Psychosocial Measures—We assessed several psychosocial factors that have been associated with HPA-axis functioning, including current emotional state, parental hostility, and life stress. The primary caregiver (“parent”) ($N = 94$; 93 mothers and 1 father) and the “co-parent” ($N = 73$; 72 fathers and 1 mother) completed the Child Behavior Checklist/1½-5 (CBCL/1½-5; Achenbach & Rescorla, 2000) Affective Problems scale ($\alpha = .62$ and .60, respectively) to assess children's current depressive symptoms. Parental hostility (91 mothers, 4 fathers) was assessed using the Teaching Task battery, an observational measure of parent-child interactions for preschool-aged children (Egeland et al., 1995). Videotapes were coded and ratings of parental hostility ($\alpha = .76$; interrater ICC = .83, $N = 53$) were based on parent's expression of anger, frustration, and annoyance directed towards the child. Total number of stressful life events involving the child in the 6 months prior to the assessment was assessed in the PAPA interview. Parents were asked whether 27 major life events occurred, and events were summed to yield a measure of life stress.

Salivary Cortisol Collection—Parents were given a cortisol collection kit during their laboratory visit. Saliva for cortisol determination was obtained by having the children dip a cotton dental roll into .025g of cherry Kool-Aid® mix. Children placed the cotton roll in their mouths until saturated. The wet cotton was collected and expressed into vials. Parents mailed the samples to our laboratory where they were stored at -20°C until assayed. These procedures are known to have little-to-no effect on cortisol concentrations (Talge, Donzella, Kryzer, Grierens, & Gunnar, 2005). Parents were instructed to collect their child's saliva 30 minutes after waking and 30 minutes before bedtime on a typical day, and to avoid feeding their child any food/drink before sampling. Average times of morning and evening samplings were 8:16 AM ($SD = 58$ min) and 8:15 PM ($SD = 49$ min), respectively, and sampling time was not significantly associated with morning ($r = -.13$, $p = .23$) or evening ($r = -.18$, $p = .08$) cortisol.

Cortisol samples were assayed in duplicate using a time-resolved fluorescence immunoassay with fluorometric end-point detection (DELFI). Samples yielding values above 44 nanomoles per liter (nmol/L) were excluded, which applied to one evening sample. The

inter- and intra-assay coefficients of variation were between 7.1%-9.0% and 4.0%-6.7%, respectively. Consistent with previous research, morning and evening cortisol values showed a positively skewed distribution (skewness = 1.31 and 3.03, respectively). We applied a log10 transformation to the data to yield unskewed values (skewness = .06 and .75, respectively). All analyses used transformed cortisol values. All other study variables were normally distributed and did not require transformations to the data.

Results

Means, standard deviations, and correlations among all major variables are presented in Table 2. A diurnal rhythm in cortisol was observed in the sample, $t(86) = 46.12$, $p < .001$, indicating that morning cortisol was significantly higher than evening cortisol for those who provided both samples on one day. Morning and evening cortisol were minimally correlated, suggesting they each capture distinct processes.

We examined associations between children's cortisol and maternal depression and child temperament separately for the morning and evening samples. Given the lack of significant correlations between evening cortisol and study variables (see Table 2), we focus on associations with morning cortisol.

Maternal depression and morning cortisol

Morning cortisol (expressed in log10 transformed nmol/L) of children of mothers with lifetime depression ($M = .59$, $SD = .21$, $N = 32$) did not significantly differ from children of mothers with no lifetime depression ($M = .53$, $SD = .18$, $N = 62$), $t(92) = 1.26$, $p = .21$, *effect size correlation r (ES)*¹ = .15). In order to explore the effects of maternal melancholia on children's cortisol levels, a one-way ANOVA comparing children of mothers with a history of melancholia, mothers with lifetime non-melancholic depression, and mothers with no depression history yielded a main effect for group, $F(2, 91) = 3.38$, $p < .05$, and the assumption of homogeneity of variances was not violated, $F(2,91) = .75$, $p = .48$. Post-hoc tests revealed that children of mothers with melancholic depression ($M = .71$, $SD = .25$, $N = 8$) had significantly higher morning cortisol levels than children of mothers with no lifetime depression ($M = .52$, $SD = .19$, $N = 62$, $p < .05$, $ES = .40$), but did not significantly differ from children of mothers with non-melancholic depression ($M = .55$, $SD = .16$, $N = 24$, $p = .11$, $ES = .36$).

Timing of maternal depression and children's morning cortisol

As seen in Table 3, we examined the relations of timing of exposure to maternal depression and current maternal depressive symptoms with children's cortisol. No correlations were significant.

Child temperament and morning cortisol

Child low PE, but not high NE, was significantly associated with morning cortisol (Table 2). Moreover, low PE ($\beta = -.26$, $SE = .03$, $p = .02$) was associated with elevated morning cortisol levels, even after controlling for NE ($\beta = .01$, $SE = .04$) and parent-reported children's depressive symptoms ($\beta = -.08$, $SE = .01$).

¹We computed the effect size correlation as the point-biserial correlation, r , between the dichotomous independent variable and the continuous dependent variable so that it can be compared to the correlation coefficients for continuous measures presented in Table 2.

Psychosocial factors and morning cortisol

Children's morning cortisol was not associated with parent- or co-parent-reported ($r = .07$) children's current depressive symptoms, parental hostility, or life stress.

Overall regression model

We examined the unique effects of maternal melancholia and child temperamental PE in a stepwise linear multiple regression with morning cortisol as the dependent variable. As seen in Table 4, potential confounding variables were entered in Step 1, and maternal depression variables were dummy coded for melancholic and non-melancholic depression and entered along with child PE in Step 2. Maternal melancholia and child low PE were uniquely associated with children's elevated morning cortisol. For the participants who reported sampling times ($N=83$), results were similar when morning sampling time was included as a potential confound.

Discussion

We found that young children's elevated morning cortisol is associated with maternal history of melancholic depression and child temperamental low PE. Our findings are consistent with the hypothesis that elevated waking cortisol is a vulnerability marker for depression, given its relation to other prominent risk factors for depression in pre-school aged children. We recognize that the direction of the associations between familial and temperamental predispositions and neuroendocrine function may be difficult to discern. These factors may have reciprocal or transactional effects on one another and/or they may be tapping a common liability for depression.

Children of mothers with a history of melancholic depression, but not children of mothers with non-melancholic depression, exhibited elevated morning cortisol. To our knowledge, this is the first study to distinguish between parental melancholic and non-melancholic depression. This finding is noteworthy given that hypercortisolism is associated with the melancholic subtype of depression (Kupfer, 1991). Melancholia also appears to be more heritable (Kendler, 1997) and confers greater risk for intergenerational transmission of internalizing and externalizing disorders than other depression subtypes (Shannon et al., 2007). Our data raise the possibility that what is transmitted to the child is a sensitive stress-response system that confers risk for later depression.

Our results appear to support findings that the relation between familial risk for depression and elevated morning cortisol is independent of parenting and depression during the child's life (Mannie et al., 2007), which is consistent with the heritability of waking cortisol. However, the nonsignificant findings concerning maternal depression during prenatal and postnatal development must be considered with caution due to the small number of mothers with depression during these periods. Contrasting the effects of maternal depression on children's basal cortisol and cortisol response to acute stressors, as well as assessing both parent and child cortisol levels, may shed further light on the environmental and biological influences in the intergenerational transmission of depression (Azar, Paquette, Zoccoliollo, Baltzer, & Tremblay, 2007; Brennan et al., 2008).

We also found that child temperamental low PE was uniquely associated with children's elevated morning cortisol, as the relation between low PE and morning cortisol persisted after controlling for temperamental NE, concurrent depressive symptoms, maternal depression, and psychosocial factors. This is the first study to demonstrate that temperamental low PE in children prior to any depressive illness is associated with elevated waking cortisol. This finding supports the specificity of the relation between low PE and depression risk (Clark et al., 1994), and is consistent with findings that low PE has

concurrent and predictive associations with other indices of depression risk (Durbin, Klein, Hayden, Buckley, & Moerk, 2005; Hayden, Klein, Durbin, & Olino, 2006). Furthermore, our finding suggests a possible neuroendocrine pathway through which PE may lead to depression, such that individuals low in temperamental PE may be less resistant, and biologically more sensitive, to the effects of stress (Lengua, Wolchick, Sandler, West, & 2000; Tugade & Fredrickson, 2004; Wichers et al., 2007).

Interestingly, both maternal melancholia and child low PE are characterized by anhedonia. This is noteworthy given that anhedonic features of depression have been specifically linked to elevated cortisol in depression (Luby et al., 2003; Turkcapar et al., 1999). Moreover, taxometric analyses suggest that melancholia, which is characterized by anhedonia, may be a discreet subtype of depression (e.g., Ambrosini, Bennett, Cleland, & Haslam, 2002). We found that maternal melancholic depression and child low PE were independently associated with child waking cortisol, suggesting that there may be multiple influences that converge on a common pathway between anhedonia and risk for depression.

Strengths and Limitations

This study had several strengths. We examined the relation between morning cortisol and a diverse range of risk factors for depression, including temperamental, familial, and environmental factors, in a sample of young children before the onset of depression in order to ensure that these factors preceded any depressive illness. We also used observational measures of child temperament and parenting, and maternal diagnoses were derived from semi-structured interviews.

This study also had several limitations. First, our sample was small, particularly with respect to mothers with melancholic depression and depression during the child's life, which limited our statistical power; therefore, our findings require replication. Second, the sample was recruited from a commercial mailing list, which may have been biased in unknown ways, and the sample lacked ethnic/racial diversity.

Third, we assessed children's morning cortisol levels 30 minutes after waking, but did not assess the *cortisol awakening response* (CAR), which is the degree of increase in cortisol during the first 30-40 minutes post-awakening. The CAR may be a more sensitive predictor of MDD in youth (Adam et al., 2008), although data are still limited. Fourth, we only assessed basal cortisol levels on one day. Additional samples would have increased the reliability of measurement, and probably yielded larger effects.

Fifth, the study was cross-sectional. Long-term follow-up is necessary to test the hypothesis that these factors contribute to the development of depression. Lastly, although we hypothesize that elevated morning cortisol levels confer risk for depression, it is unknown at what *level* of cortisol risk increases; therefore, future research should address this issue, which will also help establish the clinical utility of this vulnerability marker.

In closing, our findings demonstrate that elevated morning cortisol is related to other better-established risk factors for depression in very young children, suggesting that it may be an early-emerging vulnerability factor for depression. Thus, tracing the development and effects of morning cortisol may lead to a better understanding of the developmental pathways to depression and contribute to early intervention and prevention with young children at risk for depression.

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Abbreviations

HPA-axis	hypothalamic-pituitary-adrenal axis
PE	positive emotionality
NE	negative emotionality

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Table 1

Demographic and maternal clinical characteristics of the sample.

<i>Demographic characteristics</i>	<i>% (N)</i>	<i>M (SD)</i>
Child age (months)		43.42 (2.52)
Child female gender	43.6 (41)	
Child race		
Caucasian	91.5 (86)	
African American	2.1 (2)	
Asian American	2.1 (2)	
Other	5.3 (5)	
Hispanic	7.4 (7)	
Maternal age		37.1 (3.5)
Paternal age		39.8 (4.5)
Parental education		
Neither parent has a 4-year college degree	24.7 (22)	
One parent has a 4-year college degree	46.1 (41)	
Both parents have a 4-year college degree	29.2 (26)	
Maternal full-time or part-time employment	50.5 (46)	
<i>Maternal depression diagnosis</i>		
Lifetime depressive disorder	34.0 (32)	
Lifetime melancholic depression	8.5 (8)	
Current depressive disorder	5.3 (5)	

Note: N = 94.

Table 2

Correlations among study variables.

	1	2	3	4	5	6	7	8	9	10	11
1. Morning cortisol	--										
2. Evening cortisol	.07	--									
3. Child PE	-.25*	.13	--								
4. Child NE	-.03	-.04	.15	--							
5. Child depressive symptoms	-.08	.07	-.19 [†]	.06	--						
6. Maternal depressive disorder	.13	.10	-.08	.20 [†]	.25*	--					
7. Maternal melancholia	.25*	.02	-.03	.08	.03	.43**	--				
8. Parental hostility	-.03	-.05	-.13	-.13	.05	-.03	.01	--			
9. Child life stress	.18 [†]	-.17 [†]	-.13	-.09	-.03	-.08	.01	.09	--		
10. Age	.02	.06	.13	-.06	-.13	-.04	.04	-.03	.17	--	
11. Gender	-.07	-.11	-.09	.15	.15	.05	.04	-.13	-.03	-.01	--
Mean	9.36	2.24	.22	-.10	2.57	--	--	1.16	.49	43.42	--
SD	6.04	7.94	.65	.46	2.19	--	--	.24	.68	2.52	--

N = 94, except N = 91 for correlations involving evening cortisol; correlation analyses used log10 transformed cortisol values; however, means and standard deviations (SD) for cortisol levels are reported as raw cortisol levels in nmol/L; Child depressive symptoms = parent-reported CBCL Affective Problems scale; Maternal depressive disorder: 1 = lifetime major depressive disorder or dysthymic disorder (N = 32) and 0 = no lifetime depression (N = 62); Maternal melancholia: 1 = lifetime melancholic depression (N = 8) and 0 = no melancholic depression (N = 88); Gender: male = 0 and female = 1.

[†] $p < .10$

* $p < .05$

Table 3

Correlations between children's cortisol and timing of maternal depression.

Variable	Morning Cortisol N = 94	Evening Cortisol N = 91
Maternal depressive disorder during pregnancy (N=11)	-.03	.05
Maternal depressive disorder in child's first 2 years of life (N=13)	.06	.12
Maternal depressive disorder in child's 3 rd year-present (N=9)	.16	.15
Maternal current depressive symptoms (DID)	-.01	-.04

Note. DID = Diagnostic Inventory for Depression.

Table 4

Overall multiple regression model.

Morning Cortisol (N = 94)		
Variable	B (SE)	β
Step 1:		
Age	.00 (.01)	.02
Gender	-.03 (.04)	-.07
Child negative emotionality	-.01 (.05)	-.01
Child depressive symptoms	-.01 (.01)	-.06
Parental hostility	-.01 (.03)	-.05
Child life stress	.05 (.03)	.18
Step 2:		
Maternal non-melancholic depression	.02 (.05)	.14
Maternal melancholic depression	.15 (.08)	.23*
Child positive emotionality	-.06 (.03)	-.24*

N = 94.

*
 $p < .05$