ABSTRACT

Dysregulated cortisol is a risk factor for poor health outcomes. Children of distressed mothers exhibit dysregulated cortisol, yet it is unclear whether maternal distress predicts cortisol activity in later developmental stages. This longitudinal study examined the prospective relation between maternal distress during late childhood (9-12 years) and adolescence (15-19 years) and cortisol response in offspring in young adulthood (24-28 years). Data were collected from 51 recently divorced mothers and their children across 15 years. Higher maternal distress during late childhood was associated with lower total cortisol independent of levels of maternal distress in adolescence or young adulthood. Maternal distress during adolescence marginally predicted blunted cortisol when distress in childhood was low. Findings suggest that blunted cortisol activity in young adulthood may be a long-term consequence of exposure to maternal distress earlier in development.

Keywords: Maternal depression; Cortisol; Young Adults; Stress

INTRODUCTION

Exposure to clinical and subclinical levels of maternal distress has consistently been found to be a risk factor for mental health problems in offspring (Ashman, Dawson, & Panagiotides, 2008; Connell & Goodman, 2002; Goodman et al., 2011; Papp, Goeke-Morey, & Cummings, 2004), with effects that can persist into adulthood (e.g., Ensminger, Hanson, Riley, & Juon, 2003; Lieb, Isensee, Hofler, & Wittchen, 2002; Rohde, Lewinsohn, Klein, & Seely, 2005). Maternal depression, a commonly researched form of maternal distress, has also been linked to poor physical health in children (Timko, Cronkite, Berg, & Moos, 2002) and adult offspring (Timko et al., 2008). Further, child behavioral problems may increase maternal distress and depressive symptoms (e.g., Gross, Shaw, & Moilanen, 2008) potentially exacerbating the long-term risks for offspring.

Chronic stress early in life can disrupt regulatory processes involved in mounting adaptive responses to later stressors. Dysregulation of biological stress response systems is hypothesized to be a mechanism linking maternal distress to offspring mental and physical health problems (Luecken & Lemery, 2004). Hypothalamic pituitary adrenal (HPA) axis activity, a primary arm of the stress response system, is often measured by cortisol, the end product of the HPA axis' response to stress. Well-regulated HPA activity supports adaptive responses to challenging situations. Chronic stress during development, however, can promote dysregulation evidenced by exaggerated or blunted cortisol levels and reactivity (McEwen & Wingfield, 2003). It has been proposed that exaggerated cortisol activity may be the more immediate effect of environmental stressors, whereas attenuated activity may be a longer-term effect developed to protect against bodily damage from elevated cortisol levels (DeBellis, 2002; Lovallo, Farag, Sorocco, Cohoon, & Vincent, 2012; Miller, Chen, & Zhou, 2007). Both exaggerated and

attenuated HPA activity increase risk for a variety of adverse physical and psychological consequences including hypertension, heart disease, gastrointestinal illnesses, chronic pain, and psychological disorders (Heim, Ehlert, & Hellhammer, 2000; McEwen, 2002). Thus, exposure to maternal distress, a chronic environmental stressor, may put offspring at risk for poor mental and physical health outcomes through effects on offspring cortisol activity.

Most of the studies that have examined the link between young children's exposure to maternal distress and cortisol activity have focused on maternal depression. Preschool children of depressed mothers had higher concurrent diurnal cortisol relative to those of non-depressed mothers (e.g., Dougherty, Klein, Olino, Dyson, & Rose, 2009). A similar pattern of findings was found for preschool children of distressed mothers, such that preschooler exposed to maternal stress beginning in infancy exhibited higher diurnal cortisol levels relative to preschoolers with no exposure to maternal stress (Essex et al., 2002). . Dougherty et al. (2011) found that preschoolers with hostile depressed mothers had higher cortisol reactivity to a stress task relative to preschoolers of non-depressed or depressed non-hostile mothers (Dougherty, Klein, Rose, & Laptook, 2011). Among young children (age 2.5-6 years) from low income families, Fernald et al. (2008) found that higher maternal depressive symptoms were related to lower baseline cortisol and blunted cortisol response to cognitive testing (Fernald, Burke, & Gunnar, 2008).

Prospective relations between maternal depression and a variety of measures of offspring cortisol activity later in development have also been reported. Ashman and colleagues (2002) found that 7-8 year old children with depressed mothers had elevated baseline cortisol relative to children with non-depressed mothers, but only in children with high internalizing problems. Maternal depression during the child's first two years of life was the best predictor of elevated baseline cortisol. Gump and colleagues (2009) found that exposure to chronic maternal

depressive symptoms, from ages 3 months to 10 years, was related to lower baseline cortisol in 10 year-old offspring, regardless of child internalizing symptoms. Bouma et al. (2011) found blunted cortisol reactivity to a stress task in adolescent female offspring of parents with lifetime depressive problems relative to offspring of parents without depressive problems, independent of adolescent's current depressed mood (Bouma, Riese, Ormel, Verhulst, & Oldhinke, 2011). In summary, while a number of studies report significant associations between maternal distress and cortisol activity, it remains unclear the direction of effect, which indices of cortisol activity are most affected, whether effects occur later in development, and which developmental periods are most vulnerable. Even within similar developmental periods, it is difficult to draw conclusions given the diverse measures of cortisol activity reported. The current study adds to the literature by investigating the relations between maternal distress and cortisol activity in a group at risk for exposure to maternal distress, offspring of divorced parents. It also adds to this literature by investigating this relation in young adulthood, a later developmental period than previously examined.

Women who recently experienced divorce are at increased risk for psychological distress (Lorenz, Wickrama, Conger, & Elder, 2006) and depressive symptoms (LaPierre, 2009) compared to non-divorced women. Illustratively, an epidemiological study found that divorce increases the risk for major depression 2-4 fold in women in the United States (Weissman et al., 1996). A separate epidemiological study conducted in the Netherlands found that divorce also increased women's risk for dysthymia and social phobia, for those reporting high levels of martial quality prior to the divorce (Overbeek et al., 2006). These elevated risks may be due to stressful life changes including economic hardship (Amato, 1993; Fischer, 2007), role changes (Martinez & Forgatch, 2002), and conflict with ex-spouses (Buchanan, et al., 1991). It is

estimated that 30-50% of youth in the U.S. will experience parental divorce or separation (Kennedy & Bumpas, 2008), putting a sizeable number at risk for exposure to maternal distress. Notably, parental divorce has been found to relate to blunted cortisol in young adult offspring (Bloch, Peleg, Koren, Aner, & Klein, 2007; Kraft & Luecken, 2009). Given the increased risk for psychological distress in divorced women, exposure to maternal distress may also help explain the long-term effect of parental divorce on offspring dysregulated cortisol.

Children from divorced families are often exposed to additional risk factors including low socioeconomic status (e.g., Fischer, 2007) and inter-parental conflict (e.g., Kot & Shoemaker, 1999), which have both been linked to dysregulated cortisol (e.g., Lupien, King, Meaney, & McEwen, 2001; Saltzman, Holden, & Holahan, 2005). Further, exposure to maternal depressive symptoms increases risk of offspring mental health problems (e.g., Ashman, et al., 2008), and both internalizing (e.g., Lopez-Duran, Kovacs, & George, 2009) and externalizing problems (Alink et al., 2008) have been associated with dysregulated cortisol. It is therefore necessary to examine these factors as possible explanatory variables when seeking to understand the effect of maternal distress on cortisol activity in children from divorced families.

The current study extends prior research by examining relations between exposure to maternal distress during childhood and adolescence and cortisol response in young adults who experienced parental divorce. We examined the independent effects of maternal distress during late childhood (9-12 years) and adolescence (15-19 years) on cortisol produced across a stress task (total cortisol) and cortisol reactivity to the task. Blunted (as opposed to elevated) cortisol levels were hypothesized to result from exposure to maternal distress because chronic childhood stressors have typically been linked with attenuated cortisol responses (Lovallo et al., 2012; Miller et al., 2007). Because younger children are more dependent on their mothers, we

hypothesized that exposure to maternal distress during late childhood would be more strongly related to lower offspring cortisol response than exposure to maternal distress in adolescence. In addition, we examined the interaction between exposure to maternal distress at the two time points, hypothesizing that higher maternal distress at both time points would be associated with lower total cortisol and reactivity across the task. Finally, we evaluated whether the relation between exposure to maternal distress and cortisol activity was explained by divorce-related factors (i.e., time since divorce, inter-parental conflict, and income) or current offspring mental health problems.

METHOD

Sample

Participants included 51 young adults (YA) in the control group in a randomized trial examining the efficacy of an intervention for divorced mothers and their children (Wolchik et al., 2000). The group assigned to the intervention was not included due to potential intervention effects on cortisol activity. Participants were primarily identified via court records in the Phoenix, Arizona metropolitan area. Eligible mothers had a child between ages 9-12 and had experienced divorce within the past 2 years (see Wolchik et al., 2000 for eligibility criteria).

Assessments were conducted in participant's homes at 6 time points across 15 years. Trained interviewers administered questionnaires and conducted cortisol assessments at the final time point. Families received compensation for completing interviews. Children signed informed assent and mothers and offspring above 18 signed consent forms. All research procedures were approved by the Institutional Review Board at Arizona State University.

The analyses used data collected at 4 time points: baseline (Time 1), 3-month (Time 2), 6-year (Time 3) and 15-year (Time 4) follow-ups. Seventy-six families completed assessments

at Time 1 and 2, 68 completed Time 3, and 60 completed Time 4. Nine cases were removed from analyses: one YA refused the cortisol task, one had cortisol levels 4.8 SD above the mean, 2 were taking hypothyroid medication, and 5 were pregnant. The final sample included 51 YAs. T-tests and chi-squared tests of Time 1 demographic variables (age, sex, income) and maternal distress did not differ significantly between the 51 cases included in the analyses and the 9 excluded cases (all p's>.15) or the 16 cases who did not participate at Time 4 (all p's>.28).

Mean age at Time 1 was 10.3 years (SD=1.1, range 9-12) and at Time 4 was 25.5 years (SD=1.2, range 24-28). Mean age at time of divorce was 9.2 years (SD=1.2, range 7.25–11.67). Fifty-one percent (n=26) of YAs were female; 94% were Caucasian. Mean age of mothers was 35.9 (SD=4.8, range 27-50) years at Time 1. The majority of mothers (54%) reported gross family income at Time 1 ranging from $20,000-$35,000, 28% reported income $20,000 or lower, and 18% above $35,000; 80% of mothers completed at least some college. Time 1 demographic variables were not significantly related to maternal distress or cortisol variables (all p's>.19).

Measures

Maternal Distress. The 27-item Psychiatric Epidemiology Research Interview (PERI; Dohrenwend, Shrout, Egri, & Mendelsohn, 1980) was reported by mothers at all 4 time points. This measure assesses non-specific psychological distress during the past month and asks about psychological symptoms found across a range of psychiatric disorders. Sample items include, how often in the past month you "felt hopeless", "felt anxious", and "were confused or had trouble thinking." Although not specifically a measure of depression (Coyne, 1994) the PERI has been used to detect depression in a community sample (Roberts & Vernon, 1981) and its latent structure was found to load on the construct of depression (Tanaka & Huba, 1984). More recently, the measure has been used as a screening measure for psychiatric disorders, including

affective disorder (Werbeloff et al., 2012). Mean scores above 1.55 are considered clinically significant (Shrout, Dohrenwend, & Levav, 1986). Dohrenwend et al. (1980) report acceptable reliability and construct validity. Three scores were computed: maternal distress during late childhood (average of Time 1 and 2; α=.96), maternal distress during adolescence (Time 3; α=.93), and current maternal distress (Time 4; α=.96). Sixteen percent of mothers scored above the clinical cut-off during childhood, 7% during adolescence, and 13% during young adulthood.

YA Cortisol. Cortisol samples were collected from offspring at Time 4. Samples were taken between 2:00pm-9:00pm during a modified Trier Social Stress Task (Kirschbaum, Pirke, & Hellhammer, 1993) that included mental arithmetic and a videotaped speech task about personal strengths and weaknesses, which participants were told would be evaluated by psychologists. This task has been shown to induce significant cortisol responses in children and young adults (Yim, Quas, Cahill, & Hayakawa, 2010). YAs were instructed to avoid exercise, food, alcohol and caffeine two hours prior; compliance was queried and documented. Samples were collected with Salivette sampling devices (Sarstedt, Rommelsdorf, Germany)at 4 periods: before tasks (P1), immediately after tasks (P2), 20 minutes post-tasks (P3) and 40 minutes post-tasks (P4). Samples were assayed for cortisol using high-sensitive enzyme immunoassay (Salimetrics, Inc; sensitivity range .007-1.8; intra and inter-assay coefficients 4.13% and 8.89%). Cortisol values were log-transformed to correct for deviations from normality.

YA Mental Health Problems. Internalizing and externalizing symptoms at Time 4 (α=. 92 and .93, respectively) were measured using the Adult Self-Report (ASR) (Achenbach, Dumenci, & Rescorla, 2003), a 137-item measure for 18-59-year-olds that provides summary scores of internalizing and externalizing symptoms during the past six months. The ASR has shown good internal consistency, test-retest reliability and validity (Achenbach et al., 2003).

Divorce-related factors. At Time 1, mothers reported time since divorce and current income. They also reported on inter-parental conflict using the 10-item O'Leary-Porter Overt Hostility Scale (O'Leary & Porter, 1987). This scale has shown good internal consistency and test-re-test reliability (O'Leary & Porter, 1987). Time 1 (α=.88) and Time 2 (α=.75) scores were averaged.

Data analysis

Multivariate outlier analyses, using DFFITS, DFBETAS, Cook's distance as criterion (Neter, Wasserman, & Kutner, 1989) identified no influential cases. Skewness and kurtosis of all study variables were acceptable. Time of day of cortisol assessment, current maternal distress, and YA age, sex, body mass index (BMI), contraceptive use, medications, smoking status, and caffeine intake were examined as possible covariates. Bivariate correlations examined relations between potential covariates and individual cortisol samples, total cortisol and cortisol reactivity (see details below).

Both total cortisol output across the stress task and cortisol reactivity to the task were examined as outcomes, as both are theoretically meaningful aspects of cortisol response to challenge (Nicolson, 2008). First,multiple regression was used to predict total cortisol, calculated as area under the curve with respect to ground using the trapezoidal method (AUCg; see Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) from maternal distress. AUCg is often used to capture total hormonal output during a specified period of time (i.e., the challenge task). It correlates highly with other measures of cortisol secretion (e.g., intercept, area under the regression line; Fekedulegn et al., 2007). To examine unique effects of maternal distress at the two developmental periods, total cortisol was regressed on maternal distress in childhood and maternal distress in adolescence. To examine whether the effect of exposure to maternal distress

in adolescence on total cortisol depended on level of exposure in childhood, the interaction between maternal distress in childhood and maternal distress in adolescence was included in the model. The regression model controlled for identified covariates. For significant interaction terms, we graphed the interaction and examined simple slopes at one standard deviation above and below the mean of maternal distress in childhood (see Aiken & West, 1991).

Second, the unique and interactive effects of maternal distress on cortisol reactivity were examined using multiple regression analyses. Cortisol reactivity was calculated by subtracting each participant's cortisol value before the challenge task from their highest cortisol level after the task, based on the assumption that individual patterns of cortisol reactivity to a challenge task can differ. Approximately 40% of YAs showed reactivity (elevation from baseline) to the task. Cortisol reactivity was regressed on maternal distress in childhood and adolescence, and the interaction between the two time points, controlling for identified covariates.

Finally, to examine whether current internalizing and externalizing symptoms and/or divorce-related factors explained the results, bivariate correlations were used to assess whether these variables were related to cortisol measures, maternal distress or both. Given a significant relation between maternal distress and total cortisol or cortisol reactivity, variables found to relate to both maternal distress and cortisol were entered into the model to see if the observed relation remained significant (MacKinnon & Luecken, 2008).

MPlus (Muthén & Muthén, 1998-2011), with full maximum likelihood estimation, was used for analyses. Effect sizes were examined in addition to p-values. Partial-eta squared (η^2) values.01, .09, and .25 were interpreted as small, medium, and large effects, as recommended by Cohen (1988).

RESULTS

Preliminary Analysis

Covariates. Time of day was significantly related to P4 cortisol ($r=-.32$, $p<.05$) and marginally related to total cortisol ($r=-.28$, $p<.10$). Contraceptive use, coded using two dummy codes in order to control for sex (d1 = male vs. female no use) and contraceptive use among females (d2 = female use vs. no use), significantly related to total cortisol ($r=-.27$, $p<.05$). Other potential covariates were not significantly related to cortisol measures (all p's>.21). Current maternal distress was not significantly related to cortisol measures (all p's>.21). Based on these results, we included time of day and contraceptive use as covariates in the analyses.

Explanatory variables. Externalizing symptoms were significantly related to maternal distress in adolescence ($r=.31$, $p<.05$), P3 cortisol ($r=-.39$, $p<.05$), and total cortisol ($r=-.35$, $p<.05$). Internalizing symptoms were not significantly related to maternal distress or any cortisol measure. Inter-parental conflict was significantly related to maternal distress in childhood ($r=.42$, $p<.01$) and adolescence ($r=.34$, $p<.05$), but was not related to any cortisol measure. Time since divorce was significantly related to P1 cortisol ($r=.48$, $p<.01$), but was not significantly related to any measure of maternal distress. Income was not significantly related to cortisol or any measure of maternal distress (all p's>.18). Because current externalizing symptoms were significantly related to both predictor and outcome variables, this variable was added to significant models.

Primary Analyses

Total Cortisol. The multiple regression analysis (Table 1; Model 1; $R^2= .28$, $p=.02$) predicting total cortisol from maternal distress showed that higher maternal distress in childhood predicted lower total cortisol ($b=-.44$, SE=.16, $p=.005$; partial $\eta^2=.202$, a medium-to-large effect). Maternal distress in adolescence did not predict total cortisol ($p=.16$). The interaction

between maternal distress at the two time points was significant in predicting total cortisol (*b*=.22, SE=.10, p=.03; partial η^2=.112; a medium-sized effect). As shown in Figure 1, the relation between maternal distress in adolescence and total cortisol depended upon the level of distress in childhood. When distress during childhood was high, maternal distress in adolescence was not ssignificantly related to total cortisol. When distress in childhood was low, maternal distress in adolescence negatively related to total cortisol (small to medium-sized effect). However, neither simple slope reached significance (all p's > .34). Maternal distress in childhood was associated with lower total cortisol regardless of level of distress in adolescence.

Externalizing symptoms were then entered into the model (Table 1; Model 2; R^2=.311, p=.007). Externalizing symptoms marginally predicted lower total cortisol (p=.08), maternal distress in childhood remained a significant predictor (*b*=-.45, SE=.15, p=.003; partial η^2=.22, small-medium effect), and the interaction between maternal distress at the two time points became a marginal predictor of total cortisol (*b*=.19, SE=.10, p=.06; partial η^2=.092, medium-sized effect). The nature of the interaction was similar to that in the analysis that did not include externalizing symptoms. [INSERT TABLE 1 and FIGURE 1 HERE]

Cortisol Reactivity. For the analyses examining cortisol reactivity, multiple regression showed that no measures of maternal distress significantly related to cortisol reactivity (all p's>.12).

DISCUSSION

The current study examined relations between exposure to maternal distress in childhood and adolescence and cortisol activity in young adults whose parents divorced in childhood. As hypothesized, exposure to higher maternal distress during late childhood (ages 9-12) predicted lower total cortisol output across a challenging task in young adulthood (ages 24-28). This effect

remained significant after considering income, inter-parental conflict, time since divorce, and current externalizing and internalizing symptoms. The relation between maternal distress in adolescence and total cortisol was conditional upon levels of earlier maternal distress. Specifically, maternal distress in adolescence was associated with lower cortisol only when maternal distress in late childhood was low. Tests of the simple slopes, however, did not reach significance. This interaction became marginal after accounting for current externalizing symptoms, although it remained a medium-sized effect. Neither exposure to maternal distress in childhood nor adolescence predicted cortisol reactivity to the task.

The results are consistent with studies finding associations between maternal depression/depressive symptoms and basal or diurnal cortisol in offspring in earlier developmental stages (Ashman et al., 2002; Fernald et al., 2008; Gump et al., 2009), as well as investigations that did not find relations with cortisol stress reactivity (Ashman et al., 2002; Gump et al., 2009). The current results are unique in two ways. First, they show longer-term relations between maternal psychological functioning in late childhood and cortisol activity in young adult offspring than previous studies that focused on developmental stages ranging from infancy to adolescence (e.g., Dougherty et al., 2009; Bouma et al., 2011). Second, the current study examined psychological distress rather than depressive symptoms or clinical depression.

The finding of blunted cortisol in young adults exposed to maternal distress is consistent with investigations that have examined cortisol levels in late adolescents exposed to early life adversity (DeBellis, 2002; Miller et al., 2007). This finding may help explain the long-term negative mental and physical health risk in children of depressed mothers (e.g., Ensminger et al., 2003; Timko et al., 2008). Though both hyper and hypo-responsiveness of the HPA axis have been linked with negative health outcomes (Heim et al., 2000; McEwen, 2002), blunted activity

is emerging as a separate but equally important risk factor for future physical and mental illness (Phillips, Ginty, & Hughes, 2013). Attenuated cortisol activity has been linked with major depressive disorder (Burke, Davs, Otte, & Mohr, 2005), chronic fatigue, and chronic pain (Fries, Hesse, Hellhammer, & Hellhammer, 2005). Future longitudinal research is needed to determine whether hypo-cortisol helps explain the long-term relations between exposure to maternal distress and offspring health outcomes.

It is important to consider why youth in late childhood may be particularly susceptible to exposure to maternal distress. Late childhood is a period characterized by stressful changes, including pubertal development and school transition (Simmons, Burgeson, Carlton-Ford, & Blyth, 1987). Brain development during this time limits youths' ability to regulate emotion and cope with stressors (Dahl, 2004). Mothers with higher levels of distress may be less able to provide adequate support for their children than those with less distress (Lovejoy, Graczyk, O'Hare, & Neuman, 2000), potentially increasing children's risk for developing dysregulated stress responses. Further, highly distressed mothers may model maladaptive coping strategies (Blandon, Calkins, Keane, & O'Brien, 2008; Thompson et al., 2010), which children may incorporate into their repertoire of dealing with stressors. These maladaptive coping strategies could lead to dysregulated cortisol activity (Repetti, Taylor, & Seeman, 2002).

Although the interaction became marginal after concurrent externalizing symptoms were included, the pattern is intriguing and highlights the importance of examining this interaction with larger samples. Rather than exposure to maternal distress at both time periods having an additive effect, the negative relation between maternal distress in late adolescence and total cortisol was stronger for young adults who were *not* exposed to higher distress in late childhood. It is possible that childhood exposure may contribute to a "floor" effect, such that additional

exposure in adolescence has limited impact on later cortisol activity. Alternatively, offspring may be more vulnerable to higher of maternal distress in adolescence if such exposure is novel. This effect should be interpreted with caution and further studies are needed to replicate the current findings.

Results should be considered in light of several limitations. First, although significant effects were medium to large in magnitude, the sample size may have prevented the detection of smaller effects. Second, we cannot rule out the possibility that the effect attributed to maternal distress in late childhood represents the continuation of maternal distress that occurred earlier in development. Third, the results may not be generalizable to offspring with other family structures, and the impact of maternal distress on offspring cortisol may be potentiated by stressors associated with parental divorce. Although we examined three divorce-related factors, other aspects of the divorce may be associated with offspring cortisol and may help contextualize the current results.. Fourth, the study examined the influence of maternal distress on offspring cortisol activity, but future studies may want to consider the potential bidirectional nature of this relation. Finally, the PERI assesses a general level of distress, and may not be generalizable to maternal depression.. Although previous studies have primarily focused on exposure to depressive disorders, the current results point to the importance of examining nonspecific distress.

The current study adds to the literature by examining prospective relations between exposure to maternal distress in late childhood and adolescence and cortisol activity in young adults who experienced parental divorce. The examination of HPA stress responses in young adulthood, when brain development is relatively stabilized, provides a picture of the long-term effects of maternal distress on offspring's physiological functioning. Findings of attenuated

cortisol activity in young adulthood can put these individuals at risk for poor physical and psychological health outcomes (Heim et al., 2000). Given the increased risk for psychological distress among divorced mothers and the large number of youths who experience parental divorce, understanding long-term effects of maternal distress on offspring mental and physical health outcomes has significant public health implications.

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Table 1

Regression of maternal distress on total cortisol (AUC_G)

	Model				
	b	*β*	*SE*	*t*	*p*
Model 1:					
Maternal Distress -Child	-.80	-.44	.16	-2.81	.005
Maternal Distress -Adolescent	-.50	-.28	.20	-1.41	.16
Maternal Distress - Child X Adolescent	1.0	.22	.10	2.14	.03
Time of day	-.18	-.04	.03	-1.32	.19
Sex	-.11	-.06	.11	-.53	.60
Contraceptive Use	-.18	-.15	.12	-1.32	.19
Model 2:					
Externalizing Symptoms	-.26	-.01	.004	-1.73	.08
Maternal Distress -Child	-.78	-.45	.15	-3.00	.003
Maternal Distress -Adolescent	-.36	-.15	.21	-.70	.48
Maternal Distress - Child X Adolescent	.87	.19	.10	1.85	.06
Time of day	-.16	-.03	.03	-1.03	.31
Sex	-.13	-.10	.09	-1.01	.31
Contraceptive Use	.06	.05	.09	.59	.56

Note. $n = 51$.

Table 1

Regression of maternal distress on total cortisol (AUC_G)

	Model				
	b	*β*	*SE*	*t*	*p*
Model 1:					
Maternal Distress -Child	-.80	-.44	.16	-2.81	.005
Maternal Distress -Adolescent	-.50	-.28	.20	-1.41	.16
Maternal Distress - Child X Adolescent	1.0	.22	.10	2.14	.03
Time of day	-.18	-.04	.03	-1.32	.19
Sex	-.11	-.06	.11	-.53	.60
Contraceptive Use	-.18	-.15	.12	-1.32	.19
Model 2:					
Externalizing Symptoms	-.26	-.01	.004	-1.73	.08
Maternal Distress -Child	-.78	-.45	.15	-3.00	.003
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Maternal Distress - Child X Adolescent	.87	.19	.10	1.85	.06
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Note. $n = 51$.