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Maternal Postpartum Depression Increases Vulnerability for Toddler Behavior Problems through Infant Cortisol Reactivity

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The current study examined the role of hypothalamic-pituitary-adrenal reactivity (a physiological indicator of stress) in early infancy as a mediator of the relationship between maternal postpartum depression and toddler behavior problems. Participants were 137 at-risk mothers and their children participating in a longitudinal study of intergenerational transmission of risk. Mothers' depression was measured five times during the infants' first 18 months. Infant cortisol was collected during a social stressor (the still-face paradigm) when infants were 6 months old, and mothers reported on toddlers' internalizing and externalizing symptoms at 18 months. Among this sample of high-risk mother–infant dyads, early postpartum depression predicted atypical infant cortisol reactivity at

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6 months, which mediated the effect of maternal depression on increased toddler behavior problems. Clinical implications are discussed.

As many as one in five mothers experience postpartum depression (19.2% mild or major depression during the first 12 months postpartum, Gavin et al., 2005). Postpartum depression impairs maternal functioning and can significantly affect infant development (Conroy, Marks, Schacht, Davies, & Moran, 2010; Feldman et al., 2009). Research robustly shows that maternal depression is associated with both internalizing and externalizing child behavior problems (see meta-analysis by Goodman et al., 2011). While this effect is well established, the *mediators* of maternal depression and child outcomes are less well understood. One proposed mechanism of risk transmission is through the child's stress response system. Maternal postpartum depression has been associated with altered cortisol regulation during childhood (Dougherty, Tolep, Smith, & Rose, 2013), which in turn has been linked to child behavior problems (Van den Bergh, Van Calster, Smits, Van Huffel, & Lagae, 2008). However, the full model of cortisol reactivity as a mediator has not been tested at early stages of child development or with a community sample of women at high risk for depression. Furthermore, research is sparse on specific *infant* patterns of cortisol reactivity that reflect risk for maladaptation. Finally, questions remain about the course of postpartum depression and how timing of maternal symptoms influences the effects of maternal depression on child internalizing and externalizing symptoms. To fill these gaps, the current study examined a longitudinal model of infant cortisol reactivity as mediator of the association between maternal postpartum depression and child behavior problems during toddlerhood, in a sample of high-risk community women.

Depression in the postpartum period

The postpartum period is typically defined as the first 12 months following delivery (Gavin et al., 2005; O'hara & Swain, 1996). The course of depression in the months following delivery has been studied extensively in groups of mothers identified as meeting criteria for postpartum depression (Vliegen, Casalin, & Luyten, 2014). Studies generally find the course varies between subgroups of women. Some women experience postpartum depression that remits after several months, while others show a chronic course of depression (Campbell, Matestic, von Stauffenberg, Mohan, & Kirchner, 2007; Vliegen et al., 2014). Prevalence studies find a peak in depression occurrence around 3 months postpartum (Gavin et al., 2005). However, only a few studies have followed a community sample of women longitudinally to determine the course of depression symptoms over the full year postpartum and beyond (Mayberry, Horowitz, & Declercq, 2007).

The participants in the current study were at high-risk for depression due to oversampling for childhood trauma. Childhood trauma included physical, sexual, and emotional abuse, physical and emotional neglect, witnessing violence, and early traumatic loss. Women who have experienced childhood trauma are at increased risk for mental health problems including depression and post-traumatic stress disorder (PTSD; Chapman et al., 2004; Kilpatrick et al., 2003; Kiser, Heston, Millsap, & Pruitt, 1991). Childhood trauma could potentially lead to a more chronic course of depression for these women during the peripartum period as well, which may in turn affect their children.

Past research has shown that parental psychopathology, including depression, is partially responsible for the intergenerational transmission of abuse (Pears & Capaldi, 2001). The current study examined depression symptoms in a community sample of women at 4, 6, 12, 15, and 18 months postpartum to elucidate the course of depression symptoms over time in this nonclinical, yet high-risk group.

Postpartum depression and child behavior problems

Maternal postpartum depression has been associated with the development of behavior problems in infancy (Feldman et al., 2009), toddlerhood (Moore, Cohn, & Campbell, 2001), childhood (Campbell et al., 2007; Murray et al., 1999), and adolescence (Verbeek et al., 2012). The first year of life is a time of malleable child neurobiological, emotional, and behavioral development, leaving an infant especially vulnerable to negative influences. For instance, Verbeek et al. (2012) demonstrated that maternal depression in the postpartum period was associated with later adolescent internalizing symptoms, even when accounting for mothers' lifetime psychopathology. Additionally, chronicity of mothers' depressive symptoms is an important factor for child outcomes. Campbell et al. (2007) found that chronic high or increasing maternal depression trajectories during infancy to early childhood predicted higher child internalizing and externalizing problems in first grade, as compared to low or moderate depression symptom trajectories. Further, the effect of maternal depression on child outcomes persisted in the absence of shared genetic risk (Tully, Iacono, & McGue, 2008), indicating an environmental pathway of transmission. These internalizing and externalizing problems may have come to clinical attention during the preschool or school age years, but the mechanisms that lead to their development were likely present much earlier in life. Identifying those at high risk during infancy can significantly enhance prevention efforts.

Maternal postpartum depression on child cortisol stress response regulation

The limbic Hypothalamic-Pituitary-Adrenal (HPA) axis is involved in the mammalian response to stress. A stressor in the environment (whether psychological or physiological, e.g., pain) begins a cascading reaction in the system, leading to the release of cortisol. A typical cortisol response peaks approximately 20–25 min following a stressor, with a slow return to baseline in the subsequent 20–40 min (Gunnar & Quevedo, 2007). There are mixed findings, however, in regards to cortisol reactivity in infancy, especially to psychological stressors (Jansen, Beijers, Riksen-Walraven, & de Weerth, 2010). Some studies find a typical increase in cortisol following a stressor (Haley & Stansbury, 2003); however, others find no change or even decreases (Fortunato, Dribin, Granger, & Buss, 2008). Few studies examined individual differences that might explain these differential patterns (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996), thus further research is needed to fully understand these disparate results.

Both excessive and deficient levels of glucocorticoids can impair behavioral and physical functions, potentially leading to pathological conditions (Chrousos, 2009). Although the HPA system begins to develop in utero, literature highlights the role of early postpartum experiences (i.e., mother–infant interactions) in shaping its functioning (Doom & Gunnar, 2013; Gunnar, 1998; Gunnar & Donzella, 2002). Early life stress can lead to long-term alternations in HPA functioning (Koss, Mliner, Donzella,

& Gunnar, 2016). Maternal depression during postpartum can therefore be very influential in the early shaping of the infant HPA regulatory system (Feldman et al., 2009; Field, Diego, & Hernandez-Reif, 2009). Infants of depressed mothers have been shown to present altered cortisol baseline (prior to a stressor) levels (Conradt et al., 2016; Essex, Klein, Cho, & Kalin, 2002), diurnal patterns (Dougherty et al., 2013; Laurent et al., 2013) and reactivity in response to stressors (Brennan et al., 2008; Feldman et al., 2009) compared to infants of healthy mothers.

The majority of studies found *increased* HPA activity in offspring of mothers experiencing postpartum depression. For example, Brennan et al. (2008) found both prenatal and postpartum (but not lifetime) depression was significantly associated with increased infant baseline cortisol as well as reactivity to laboratory stressors (abrupt noise and arm restraint). Ashman, Dawson, Panagiotides, Yamada, and Wilkinson (2002) found maternal depression during the first two years of life to be significantly associated with increased child baseline cortisol prior to a stressor at age 7 years. Feldman et al. (2009) found 9-month-old infants of depressed mothers to have increased cortisol reactivity across a stressful mother–child interaction task compared to matched controls. Additionally, postpartum depression has been shown to have long-standing effects such that even adolescent and young adult children of mothers who had experienced postpartum depression have increased morning cortisol (Halligan, Herbert, Goodyer, & Murray, 2004) and showed greater cortisol reactivity in response to a social stress task, compared to controls (Barry et al., 2015).

Conversely, some research showed that early life stress leads to *decreased* cortisol and a flattened daily rhythm in toddlers (Gunnar & Vazquez, 2001). Additionally, not all studies have unearthed associations between postpartum depression and infant cortisol. For example, Grant and colleagues found that prenatal anxiety and maternal parenting sensitivity, but not postpartum depression, were linked to infant cortisol reactivity (Grant et al., 2009). Thus, more research is needed to fully elucidate the relationship between maternal postpartum depression and infant HPA functioning.

HPA regulation association with internalizing and externalizing problems in early childhood

Abnormal cortisol levels and reactivity have been linked to both internalizing and externalizing behaviors in children. Notably, however, the direction of the effect differed across studies. While some studies found that increased cortisol was related to heightened symptoms (e.g., higher baseline cortisol in preterm infants predicted internalizing and externalizing symptoms; Brummelte et al., 2011), many other studies found that hypocortisolism including decreased basal cortisol (McBurnett, Lahey, Rathouz, & Loeber, 2000), a flattened diurnal slope (Gunnar & Vazquez, 2001), blunted reactivity (van Goozen et al., 1998), or an attenuated cortisol awakening response (Freitag et al., 2009), was indicative of more problems. These mixed findings might be due to the distinct outcomes examined in different studies (i.e., internalizing versus externalizing). Some evidence pointed toward increased cortisol levels in children with internalizing disorders but decreased levels in children with externalizing problems (for stress reactivity; e.g., van Goozen et al., 1998; and baseline cortisol, e.g., Shirtcliff, Granger, Booth, & Johnson, 2005; Smider et al., 2002; see also Buss & Goldsmith, 2007). However, high comorbidity of internalizing and externalizing symptoms in children lead many to question this conclusion, and indeed, other studies showed

that cortisol levels predict general symptom severity rather than internalizing versus externalizing symptoms (low basal cortisol and flat diurnal rhythms, Shirtcliff & Essex, 2008).

Developmental effects also likely play a role in the conflicting findings (Alink et al., 2008; McGinnis, Lopez-Duran, Martinez-Torteya, Abelson, & Muzik, 2016; Ruttle et al., 2011), as the link between child cortisol reactivity and problem behavior might be age dependent. For example, the meta-analysis by Alink et al. (2008) found that behavior problems were associated with higher basal cortisol in preschool children but lower basal cortisol in elementary aged children. Similarly, others have proposed heightened reactivity in child HPA axis to adversity early on in life leading to subsequent down-regulation of the system with ongoing contextual adversity (Gunnar & Vazquez, 2001; Gustafsson, Anckarsäter, Lichtenstein, Nelson, & Gustafsson, 2010). Therefore, early chronic heightened stress reactivity or elevated basal cortisol levels might predict later hypocortisolism. Thus, either elevated or blunted cortisol might be associated with psychopathology depending on when it is measured, when stress occurred, and individual differences in HPA axis functioning (Heim, Ehlert, & Hellhammer, 2000). Mechanistically, there might be a nonlinear relationship between cortisol and child outcomes, where either too much or too little cortisol is indicative of problems (for review see Doom & Gunnar, 2013).

Overall, research on infant and toddler cortisol reactivity and subsequent behavior problems is quite limited. However, multiple conceptual models propose that experiences in the first years of life are critical for the developing HPA system, which in turn might affect risk for psychopathology (Gunnar & Vazquez, 2001). Cross-sectional findings also support these associations. Cortisol reactivity was related to concurrent negative affect in 4- to 6-month-old infants (Lewis & Ramsay, 2005), including children of depressed mothers (Huot, Brennan, Stowe, Plotsky, & Walker, 2004). In a sample of toddlers born prematurely, a heightened cortisol response between 18 and 60 months was associated with higher concurrent internalizing and externalizing symptoms (Bagner, Sheinkopf, Vohr, & Lester, 2010). In contrast, lower diurnal cortisol at 54 months in children of depressed mothers predicted heightened internalizing symptoms (Laurent et al., 2013). To date, no studies that we are aware of have examined the longitudinal associations between abnormal cortisol reactivity *in infancy* and subsequent internalizing and externalizing behavior problems.

HPA regulation as a mediator

The evidence reviewed above suggests that maternal depression leads to atypical functioning of the offspring HPA axis system, and in turn, abnormal cortisol levels and reactivity in childhood are associated with increased behavior problems. Depression likely leads to increased child behavior problems through both biological (e.g., genetics, heightened negative emotionality passed on to infant) and environmental (parenting, symptoms generating more stressors that directly affect the child) pathways. HPA axis reactivity appears to be a good candidate mechanism because (1) it is shaped by both biological predispositions and early experiences and (2) has demonstrated longitudinal associations with psychopathology in early childhood and beyond.

Only a few studies have directly tested the mediation effect of HPA functioning, and all in older children or adolescents. In one study examining depression in children of depressed and nondepressed mothers, Halligan and colleagues (2007) found that

maternal depression measured during the first postpartum year predicted higher, more variable morning cortisol levels in offspring at age 13, and youth self-reported depression at age 16. The adolescents' cortisol levels mediated the effect of maternal depression on youth depressive symptoms. A study by Brooker, Davidson, and Goldsmith (2016) found that maternal trait related negative affect in the first year postpartum also led to increased internalizing symptoms in offspring at age 7 and that this effect was mediated by the child's concurrent diurnal cortisol pattern (i.e., flattened curve). Finally, a recent study by Apter-Levi et al. (2016) examined a sample of chronically depressed versus nondepressed women and their 6-year-old children, excluding women for comorbid conditions or high psychosocial stress. In this sample, maternal postpartum depression longitudinally predicted more restricted cortisol variability to a stressor, which, in turn, was associated with higher child psychopathology and social withdrawal concurrently.

The studies reviewed leave several questions unanswered. First, it has yet to be determined whether child HPA regulation is a mediator of the effect of maternal depression on child outcomes over the full spectrum of depressive symptoms, rather than for only those offspring with mothers with clinical level depressions. Additionally, research with very young infants and children is needed to determine whether effects on the HPA axis are detectable at young ages and confer meaningful risk for later behavioral problems. Further, it will be important to evaluate whether the mediational pathway differs for internalizing versus externalizing symptoms. Finally, it is crucial to test the mediation model in a longitudinal design, with child behavior measured after child HPA activity, as well as to control for later maternal depression to determine if the effect is specific to postpartum depression.

The current study

The current study sought to address these gaps and expand knowledge in this area by longitudinally examining the effects of postpartum depression (PPD) in a sample of women at high risk for chronic depression (by oversampling women with childhood trauma histories). We first examined the trajectories of maternal PPD as well as the patterns of infant cortisol reactivity. Next, in a latent growth curve structural equation model, we examined the effect of maternal PPD on infant cortisol reactivity and later toddler behavior problems controlling for demographic risk, maternal history of PTSD, and child gender (covariates which might separately influence cortisol and/or child behavior problems). Due to the likely high degree of comorbidity between depression and PTSD among participating mothers, PTSD was included as a covariate.

We hypothesized that group means in depression symptoms would be the highest at 4 months postpartum given prior reports that postpartum depression peaks 3 months after delivery (Gavin et al., 2005) and decreases thereafter, with relative stability across time. We also predicted that PPD would be associated with atypical infant cortisol reactivity. Given the mixed findings regarding the effects of stress on infant HPA functioning, we did not predict the direction of the effect. Additionally, based on prior literature (Moore et al., 2001) we hypothesized that PPD would be associated with increased toddler internalizing and externalizing problem behaviors. Although we acknowledged the comorbidity of symptoms in internalizing and externalizing domains at this age, we examined pathways to each set of symptoms separately because of the

aforementioned studies that suggest differential effects of infant stress reactivity on internalizing versus externalizing behaviors. Finally, we tested a mediational model, where we predicted that infant cortisol reactivity would mediate the relationship between maternal depression and child behavior problems.

METHOD

The current study utilized self-report and observed data collected from mothers and their infants at 4, 6, 12, 15, and 18 months postpartum. Data were drawn from the Maternal Anxiety During the Childbearing Years study (MACY; Muzik et al., 2013), a larger parent study assessing the longitudinal impact of maternal childhood abuse on the transition to motherhood, postpartum psychopathology, parenting efficacy and behaviors, the development of the mother–child relationship and intergenerational transmission of risk.

Procedure

Women were recruited for the MACY study ($N = 268$) either as a postpartum follow-up to a separate prenatal study on the effects of PTSD on childbearing (STACY; Seng, Low, Sperlich, Ronis, & Liberzon, 2009), or from the community when they were within 4 months postpartum. Participants receiving prenatal care were approached directly during their antenatal visit at three pregnancy clinics in a large Metropolitan Area. Community recruitment was conducted using informational flyers posted in childcare centers and pediatric healthcare offices throughout the same area. Participants were nonpsychiatrically referred English speaking women, ages 18 and older. Exclusion criteria included the use of illegal or nonprescription drugs during pregnancy, maternal history of bipolar disorder or psychotic illness, child prematurity (<36 weeks gestation at birth), or severe child developmental disability or illness reported at 4 months. Women who met eligibility criteria were invited to participate in the study and provided oral assent for participation. During the first in-person assessment (at 6 months) mothers provided written consent for participation.

During the screening interview, women were administered the Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003). Mothers self-rated their childhood maltreatment experiences on a 28-item, 5 point Likert scale (1-never true to 5-very often true). The CTQ yields subscale scores for childhood emotional, physical, or sexual abuse, as well as physical and emotional neglect. For each of the 5 subscale domains, there is a cut-off score that determines whether a person qualifies as having experienced the particular type of abuse (Y/N on sexual, physical, emotional abuse, and physical or emotional neglect; Bernstein & Fink, 1998). In this study, we counted women who scored above cutoff on any of the five maltreatment types as meeting criteria for "any abuse"). In the parent sample, 66% of women endorsed "any abuse" in childhood.

Participants completed telephone interviews at 4, 6, 12, and 18 months postpartum, two home visits at 6 months postpartum, and a playroom visit at 15 months postpartum. Data collected during study interviews and visits, included: childhood abuse history, demographics and mothers' postpartum psychopathology, mother–infant interaction tasks including a stress task, the Still-Face Procedure (SFP; Tronick, Als,

Adamson, Wise, & Brazelton, 1978), and infant/child biological and socio-emotional outcomes. For this investigation, we narrowed the analysis to maternal abuse history and postpartum psychopathology (depression and PTSD), infant stress biomarkers (cortisol) following the SFP, and toddler problem behavior ratings.

Participants

The current study included data on a subset of the overall sample of women who completed longitudinal assessments beyond the initial baseline assessment at 4 months postpartum, and whose infants provided cortisol samples at 6 months postpartum. Participants were 137 mothers and their infants (53% boys). The inclusion criterion for the current study was the presence of data: (1) on the maternal depression rating scale (Postpartum Depression Screening Scale, PDSS, Beck & Gable, 2000) for at least one of the multiple time-points, and (2) on at least one measurement of infant cortisol following the SFP. Of the 137 mothers, 72.3% reported a history of childhood trauma ("any abuse" on the CTQ, see above); 28.7 and 22.8% met diagnostic criteria for PTSD and depression at 6 months postpartum, respectively (see Table 1). Overall, 12.4% of women were above the clinical threshold on measures of both depression and PTSD at 6 months postpartum (also see correlations between depression and PTSD symptom counts in Table 2). More than half (65.6%) were Caucasian, 22.2% were African American, and the remaining 12.2% were Asian/Pacific Islander, Latina or biracial. At 6 months postpartum, nearly a fourth (21.2%) were not married or living with an intimate partner. Regarding level of maternal education, 8.1% had a high school diploma or GED, 28.7% had completed some college (Associate's or vocational degree), 33.1% had a Bachelor's degree, and 25% had a Master's or Doctoral degree. Participants' family income was bimodal such that 30.4% reported less than $25,000 per year, and 20% reported an income of more than $100,000 per year. None of these subset demographics differed significantly from the demographics from the overall parent sample. The present study was conducted according to guidelines laid down in the Declaration of Helsinki, with written informed consent obtained from a parent or guardian for each child before any assessment or data collection. All procedures involving human subjects in this study were approved by the Institutional Review Board at the University of Michigan.

Measures

Maternal measures

Depression. Maternal depression symptomology was assessed using the Postpartum Depression Screening Scale when infants were 4, 6, 12, 15, and 18 months old (PPDS; Beck & Gable, 2000). While we were primarily interested in postpartum depression (0–12 months), we continued to measure depression into the child's second year in order to chart the course of depressive symptoms and to control for concurrent depression at the time child outcomes were assessed (18 months). The PPDS is a 35-item self-report questionnaire using a Likert scale ranging from 1 to 5. It yields a total symptom count and a diagnosis of Major Depressive Disorder (MDD) as indicated by a total symptom count of >80 (Beck & Gable, 2000). The PPDS has a sensitivity of .78,

TABLE 1
Descriptive Statistics

	M (SD)	*Min–max*	*% Above clinical cutoff*
Maternal depression symptoms			
4 months	64.85 (22.93)	35–155	19%
6 months	65.79 (22.51)	35–132	26.3%
12 months	66.77 (21.29)	35–127	22.6%
15 months	66.48 (21.29)	35–153	22.1%
18 months	64.93 (20.55)	35–128	19.9%
Consistently Across all time points			9.6%
	M (SD)	*Min–max*	
Infant cortisol level			
Baseline	.22 (.14)	.01–.87	
20 min poststressor	.19 (.14)	.01–.72	
40 min poststressor	.20 (.14)	.01–.92	
60 min poststressor	.21 (.13)	.01–.63	
	M (SD)	*Min–max*	*% Above clinical cutoff (% at risk)*
Toddler behavior problems			
Internalizing symptoms *T*-score	46.30 (7.49)	29–71	0.7% (2.9%)
Externalizing symptoms *T*-score	49.58 (7.77)	32–82	4.4% (7.3%)
	M (SD)	*Min–max*	*% Above clinical cutoff*
Covariates			
Lifetime PTSD symptoms	6.73 (5.50)	0–17	28.7%
Cumulative risk score	1.02 (1.27)	0–5	–

specificity of .99, and positive predictive value of .93 compared with a Structured Clinical Interview for DSM (SCID) diagnosis of depression (the gold standard measurement of mental disorders). In the current study, total symptom count was used as a continuous measure of depression. Percent of women above the clinical threshold was reported for illustrative purposes in Table 1. The standardized Cronbach's alpha for this variable was .96. PDSS scores ranged from 35 to 132 in this sample (M = 65.79, SD = 22.51).

Cumulative risk index. Maternal demographic information was collected at the screening phone interview and updated at each subsequent data collection time point. The demographic questionnaire assessed five domains: yearly household income, maternal race, age, level of education, and relationship status. Each domain was dummy coded into dichotomous variables based on the presence (1) or absence (0) of a risk threshold/status: income (<$20,000 per year), minority status (non-Caucasian), age (<22), education (≤high school), and partner status (not partnered). A cumulative risk index was computed (0–5) by summing across the five variable scores. This coding scheme is similar to those used in other research on maternal risk and parenting (Sameroff, Seifer, Baldwin, & Baldwin, 1993). Cumulative risk was used as a covariate.

Post-traumatic stress disorder. Post-Traumatic Stress Disorder (PTSD) symptoms were assessed to be used as covariate using the National Women's Study PTSD

TABLE 2
Bivariate Correlations Among Study Variables

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*
Maternal depression												
1. 4 months	1											
2. 6 months	.76***	1										
3. 12 months	.80***	.84***	1									
4. 15 months	.72***	.72***	.81***	1								
5. 18 months	.69***	.75***	.85***	.84***	1							
Infant cortisol												
6. Baseline	.09	.04	−.01	−.02	−.01	1						
7. 20 min	−.14	−.17*	−.23**	−.19*	−.19*	.51***	1					
8. 40 min	−.04	−.07	−.05	−.03	−.02	.48***	.60***	1				
9. 60 min	−.09	−.11	−.15	−.11	−.11	.68***	.82***	.59***	1			
Toddler behavior problems												
10. Internalizing *T*-score	.12	.16	.15	.19*	.31***	.06	−.09	−.04	−.01	1		
11. Externalizing *T*-score	.15	.18*	.20*	.22**	.34***	.02	−.02	−.04	.04	.63***	1	
Coviarates												
12. Lifetime PTSD symptoms	.48***	.42***	.53***	.49***	.48***	.01	−.21*	−.08	−.14	.12	.09	1
13. Cumulative risk score	.17*	.09	.11	.10	.09	.06	.10	.16	.04	.05	.01	.14

*$p < .05$, **$p < .01$, ***$p < .001$.

Module (Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993) at 6 months postpartum. This scale measures all 17 symptoms of PTSD and yields both a dichotomous diagnosis and a continuous symptom count score. PTSD diagnoses were created using DSM-IV criteria of having at least 1 symptom of re-experiencing, 3 symptoms of avoidance, and 2 symptoms of hyperarousal, in concordance with previous literature (Resnick et al., 1993). The standardized Cronbach's alpha for this variable was .88. PTSD scores ranged from 0 to 17 in this sample ($M = 4.22$, $SD = 4.41$).

Infant measures

Infant interactive stress task, still-face procedure. Infants' saliva was collected before and after an interactive, relational challenge task, the Still-Face Paradigm (SFP; Tronick et al., 1978). The SFP is a widely used, well-validated observational paradigm designed to evaluate individual differences on maternal and infant behavioral and physiological responses before and after a social interactive challenge (mother holding a "still-face"). The SFP was videotaped in the home setting at 6 months postpartum, with prior research demonstrating feasibility of the setting and infant age (Meijssen et al., 2010). During the SFP, the infant was seated in car seat on the floor facing the mother. A mirror was placed behind the dyad to allow for optimal camera views of each partner. The SPF comprised three successive 2-min episodes: (1) a "normal" dyadic play episode (Play 1), during which the mother was asked to play with her infant in an *en face* position in a normal manner, followed by (2) a maternal still-face episode (Still-Face), during which the mother was instructed to hold a neutral/unresponsive face while looking at her infant and to refrain from touching or talking to the infant, followed by (3) a re-engagement episode (Reunion) during which the mother resumes her normal social play with the infant. No toys or pacifiers were allowed during the SFP. Following the SFP, mothers completed a number of questionnaires, and cared for their infant as normal during this period. Saliva was collected at 20-min increments following the SFP.

Infant salivary cortisol. Repeated saliva samples were collected from the infant before (baseline) and after the SFP. The baseline was collected 15 min into the home visit, after the consent form was explained and signed, and before any interactive tasks had begun. Consistent with established and standardized protocols (Granger et al., 2007), cortisol was collected again 20, 40 and 60 min following the SFP. To collect infant saliva, mothers were asked to place a cotton roll, securely attached to a strand of floss, in their infant's mouth until it was saturated with saliva. When the cotton roll was fully saturated, it was put into a Salivette Tube (Sarstedt, Sevelen, Switzerland). Saliva samples were centrifuged at 5378 g for 10 min, and stored at −20°C until assayed. Samples were assayed using one of three techniques based on laboratory and equipment availability: ELISA (40%), RIA (21%) or Immulite (39%). All samples from the same infant were assayed in the same batch to minimize inter-assay variation. Inter- and intra-assay coefficients of variation were consistently below 10%. Following the assays, cortisol samples were cleaned for outliers. Because the cortisol assay range of detection is .003 to 3 μg/dl, values above 3 were deleted, as they represented problems with the assaying process. Then, outliers were examined and values above 3 *SD*s were winsorized to maintain rank ordering and address normality issues, following

conventional practices (Dettling, Parker, Lane, Sebanc, & Gunnar, 2000). In the final database, cortisol values ranged from .00 to .92 mg/dl for infants.

Toddler behavior problems. At the 18-month phone survey, mothers reported on their toddlers' problem behaviors using the Child Behavior Checklist (CBCL/1.5–5, Achenbach & Rescorla, 2000). Scoring yields two broadband scores: Internalizing and Externalizing, which have high internal reliability in the current sample (α = .77, and .88, respectively). Internalizing *T* scores ranged from 29 to 71 in this sample (M = 46.32, SD = 9.13). Externalizing *T* scores ranged from 32 to 82 in this sample (M = 49.66, SD = 9.37).

Missing data

Results of preliminary bivariate analyses revealed no evidence for differential attrition: The subsample included in the present analysis (N = 137) did not differ significantly from the participants in the larger study (N = 268) on key demographic characteristics, including maternal age, race/ethnicity, education, or partner status, the family's total annual income, or on infant race/ethnicity or gender.

Data were missing on measures for reasons unrelated to the study variables, confirmed via nonresponse analyses, and therefore were determined to be missing at random (MAR). Missingness for each variable ranged from 0 to 36.5% of reported data from participants. For infant salivary cortisol, the most common reason for missing scores was the infant being asleep at the time or unable to provide enough saliva for the assay. Those infants with complete salivary cortisol data did not differ from those with missing samples on any demographic or independent variable evaluated. Bivariate associations showed no relationships between any cortisol measures and time of sample collection, infants' birth weight, infants' health at the time of the 6-month visit, or time from last feeding or sleeping. Therefore, these factors were not included in final statistical models.

Reasons for missing behavioral data included technical problems with videotaping, participant scheduling conflicts, or examiner error. Mother–infant dyads with complete observational data did not differ from those with missing scores on any demographic or risk variable included in this study. Thus, there was no indication of sample bias due to missing data. Data were imputed using the Expectation Maximization algorithm (Dempster, Laird, & Rubin, 1977) in SPSS 20, a maximum likelihood approach, using measures not included in the current study to inform imputation; each variable was imputed separately. This approach is considered ideal for structural equation modeling (SEM) and when data are determined to be MAR (Musil, Warner, Yobas, & Jones, 2002; Schafer & Graham, 2002).

RESULTS

Descriptive statistics are presented in Table 1 and bivariate correlations between study variables are found in Table 2. We utilized structural equation modeling (SEM) to fit latent growth curves (LGC) and test their associations in LISREL 9.2. We chose cross-domain latent growth curve modeling to test our main hypotheses because it supports the assumptions of nested data and controls for shared error variance (Burchinal,

Nelson, & Poe, 2006; Duncan, Duncan, & Hops, 1998). We first tested an unconstrained LGC for postpartum depression and one for infant cortisol reactivity. The purpose of testing these models *unconstrained*, without additional modifying variables, was to determine the shape and fit of the data. Next, we tested a constrained model, hypothesizing a mediating effect of infant cortisol at 6 months on the relationship between early postpartum depression (at 4 months postpartum) and later toddler problem behaviors, controlling for lifetime occurrence of maternal post-traumatic stress disorder, cumulative risk, and child gender. SEM fit indices are reported based on published criteria for good fit as applied in the psychological sciences (RMSEA < .08, GFI > .90, SRMR < .08; Browne & Cudeck, 1993; Hu & Bentler, 1999). Models with poorer fit are presented for illustrative purposes and not to test hypotheses.

Depression across postpartum

An unconstrained single domain LGC was estimated for maternal PPD to determine the shape and fit of the data over time. Women's depressive symptoms abated over time with peak mean scores earlier in the postpartum period ($\chi^2 = 9.71$, $df = 6$, $p = .13$; RMSEA = .05; GFI = .99). There was significant variance in level of depressive symptoms at 4 months postpartum but nonsignificant variability in rate of change of symptomology during the postpartum period, suggesting an overall trend of stability in rank ordering of participant women.

Infant cortisol reactivity

An unconstrained single domain LGC was estimated for infant cortisol reactivity to determine the shape and fit of the data over the 60 min period (baseline, 20, 40, 60 min). A quadratic pattern improved upon the fit of a linear or no-growth pattern in the data, with acceptable fit ($\chi^2 = 6.17$, $df = 3$, $p = .10$; RMSEA = .08; GFI = .99). Infants demonstrated significant between-person variability in intercept and slope covariance, suggesting heterogeneity in the sample in both level and change over time.

Postpartum depression, infant cortisol, and toddler behavior

The constrained model, including the LGCs for maternal depression over the first 18 months and infant cortisol reactivity to a stressor at 6 months, covariates (maternal demographic risk, maternal PTSD symptoms, and child gender), and outcomes (toddler CBCL internalizing and externalizing symptoms) demonstrated a good fit with the data ($\chi^2 = 88.83$, $df = 62$, $p = .03$; RMSEA = .05; GFI = .93; SRMR = .08; see Figure 1) and explained 43% (externalizing) and 45% (internalizing) of the variance in toddler problem behaviors. The effect of the slope factor on outcomes reflects the effect of within-person variance on children's outcomes. Modeling the data in this way allowed us to control for women's own depression scores across the postpartum period, including the women's depression scores concurrent with child outcomes in toddlerhood.

Published guidelines for testing mediation in SEM (Holmbeck, 1997) were followed. Those steps are: (1) good model fit for the complete model; (2) good model fit when constraining to zero the path between X and M and the path between M and Y, only estimating the direct effect; and (3) no improvement in model fit for the complete model when only the direct effect is constrained to zero. Via this strategy, we

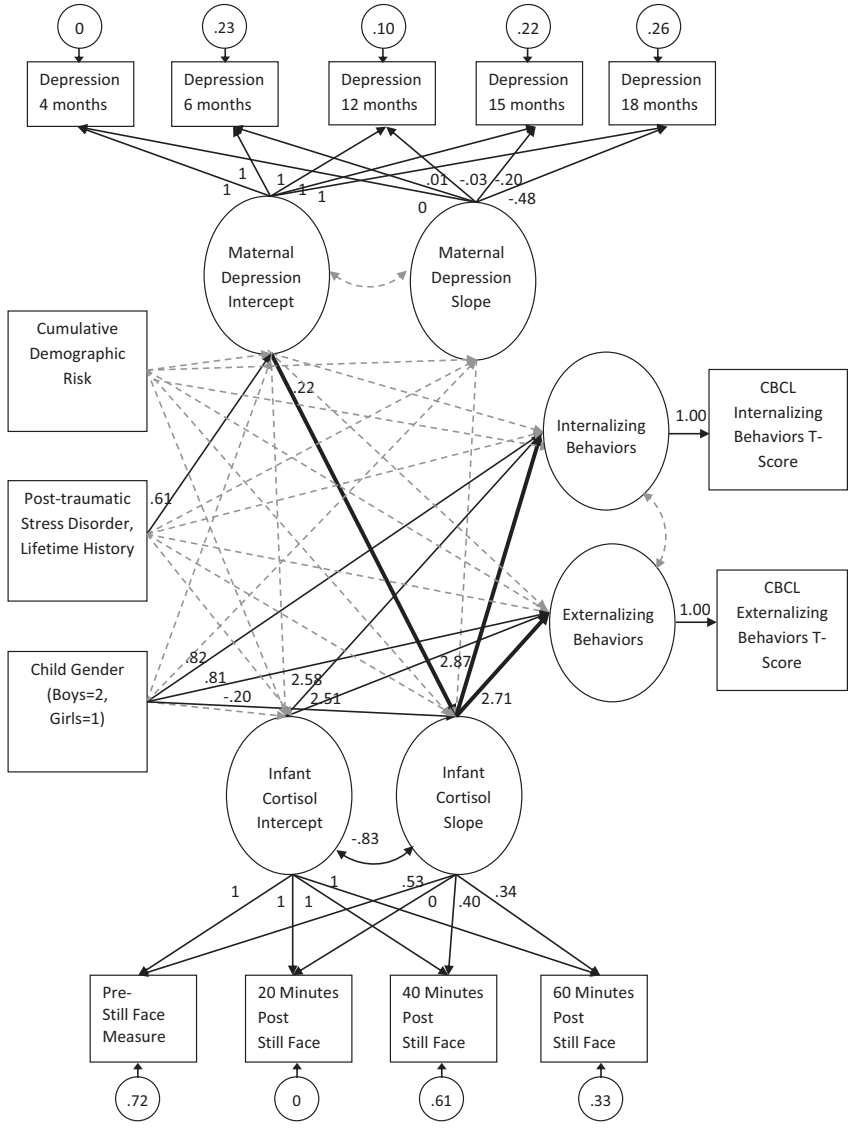

Figure 1 Model depicting full mediation of infant cortisol on the relationship between maternal depression and later toddler internalizing and externalizing behaviors (both direct and indirect effects estimated).

Note. Solid lines indicate significant effects. Standardized estimates are reported next to significant pathways. CBCL = Child Behavior Checklist.

confirmed a mediating effect of infant cortisol reactivity on the relationship between maternal depression at 4 months and later toddler outcomes. There was no significant relationship between maternal depression and baseline cortisol, nor between change in maternal depression over time and child HPA functioning. The direct effect of maternal depression at 4 months on toddler outcomes was significant without the indirect effects estimated (see Figure 2; $\chi^2 = 144.12$, $df = 70$, $p = .00$; RMSEA = .09;

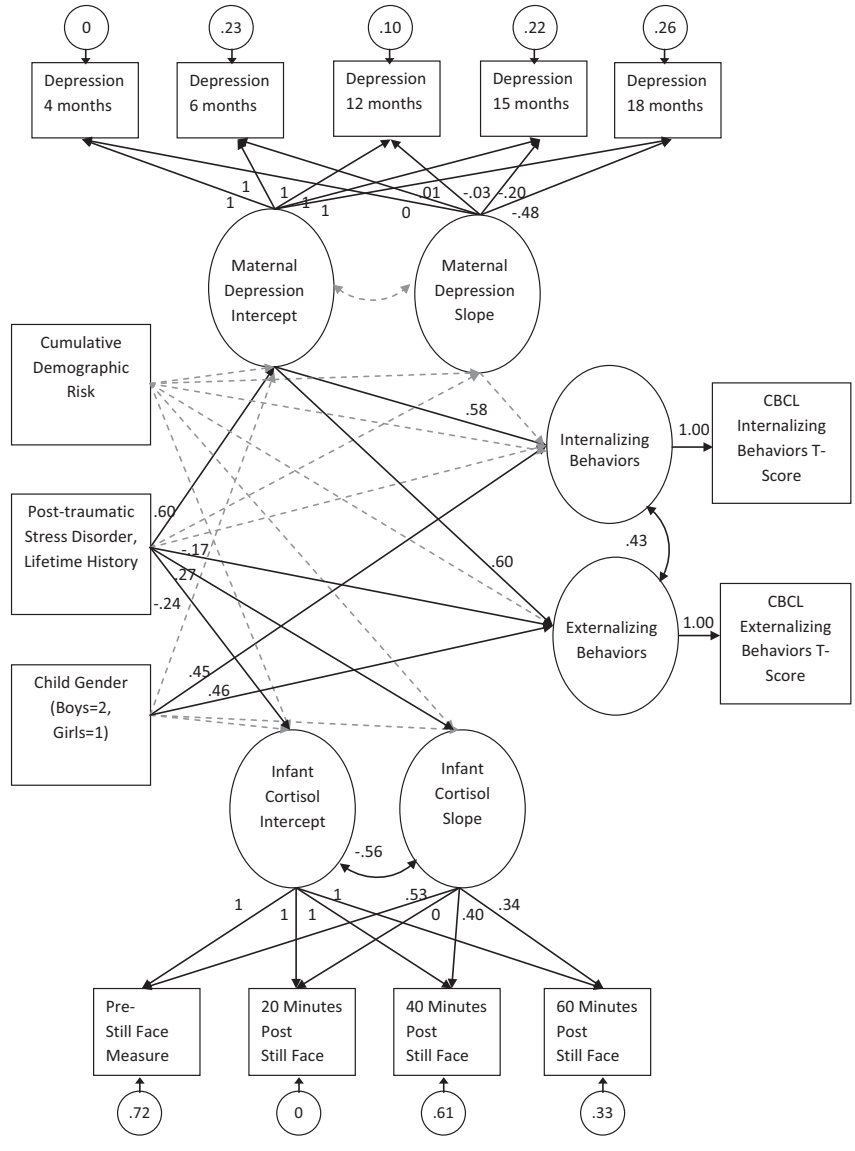

Figure 2 Model depicting relationship between maternal depression and later toddler internalizing and externalizing behaviors (only direct effect estimated).
Note. Solid lines indicate significant effects. Standardized estimates are reported next to significant pathways. CBCL = Child Behavior Checklist.

GFI = .87; SRMR = .17). The relationships between maternal depression and problem behaviors in toddlerhood were not significant when the relationships between infant cortisol change factors (intercept and slope) and toddler outcomes were estimated (see Figure 1; $\chi^2 = 88.83$, $df = 62$, $p = .03$; RMSEA = .05; GFI = .93; SRMR = .08.). There was no significant improvement in model fit when the direct effect was constrained to zero ($\chi^2 = 109.63$, $df = 65$, $p = .00$; RMSEA = .07; GFI = .91). Thus, all three steps indicated significant mediation.

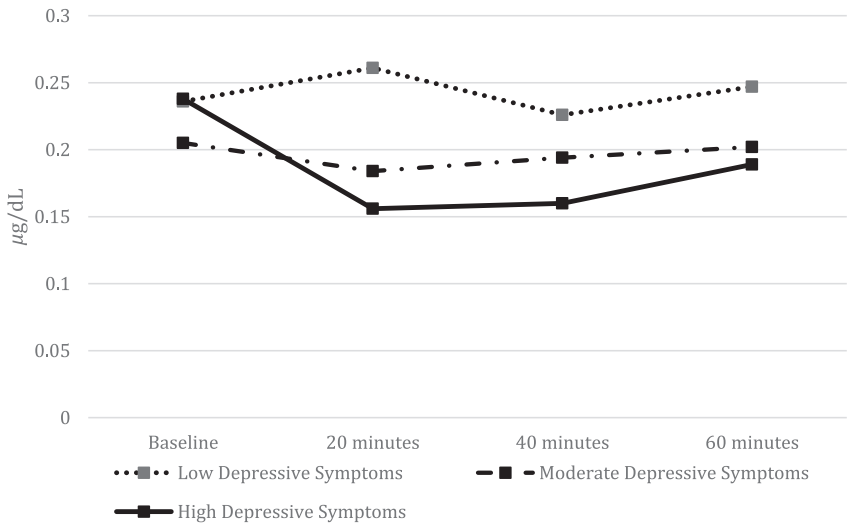

Figure 3 Child cortisol reactivity patterns by maternal depression grouping.
Note. Maternal Depression grouping based on *SD* from mothers' mean depression score: low (below −1 *SD*), moderate (between −1 *SD* and 1 *SD*), and high (above 1 *SD*). *X*-axis depicts time since stressor (still-face paradigm); *y*-axis depicts salivary cortisol level in mcg/dl.

In addition, the indirect effect coefficient of the mediation pathway was calculated in LISREL as the product of the A and B pathways (MacKinnon, Lockwood, & Williams, 2004) and confidence intervals were estimated for significance testing. The indirect effect predicting internalizing behaviors was .02, with a 95% bootstrapped confidence interval of .020, −.036. The coefficient of the mediation pathway predicting externalizing behaviors was .03, with a 95% confidence interval of −.006, −.028. Taken together, the two separate approaches to assess mediation converged to suggest partial mediation was present for internalizing behaviors. Partial mediation was supported for externalizing behaviors with the first approach, but the confidence interval in the second approach included a zero and suggested a nonsignificant finding.

To illustrate the effect of maternal depression on child cortisol, we grouped mothers in three groups based on their depression scores at 6 months (low, moderate and high; see Figure 3). The LGC model showed that mothers with higher depressive symptoms had children who showed a more atypical response to the stressor (higher slope; see mean cortisol values in Figure 3) and had more internalizing and externalizing symptoms a year later.

DISCUSSION

The aim of the current study was to examine the longitudinal course of maternal postpartum depression and its impact on infant cortisol reactivity and toddler behavior problems, and to test whether infant HPA functioning mediated the association between postpartum depression and child outcomes. First, we found that overall depression symptoms declined somewhat over the second postpartum year with the highest mean scores occurring when infants were 12 months old and the largest percent

of women above the clinical cutoff occurring at 6 months postpartum. This is in contrast to prior studies that showed peak point prevalence levels earlier in the postpartum period (e.g., 3 months postpartum, Gavin et al., 2005). In our sample, approximately 10 percent of women were above the clinical cutoff at all 5 time points, indicating a chronic course of postpartum depression for a subset of women. Our latent growth model provided further evidence for this by demonstrating stability in rank ordering of women in our sample, indicating that those with the highest depression symptoms remained high compared with other participants. This finding is critical for intervention efforts targeted at women experiencing postpartum depression. Those with high symptoms relatively early in the postpartum period are likely to continue along this trajectory over the first 18 months and may benefit significantly from early intervention. As expected, maternal depressive symptoms had a direct effect on toddler behavioral and emotional problems, with mothers experiencing more severe postpartum depression also reporting more symptoms for their toddlers at 18 months old.

Next, we found an unexpected pattern of infant cortisol reactivity in our entire sample. Mean cortisol values initially declined from baseline following the stressor and then rose at subsequent time points. This pattern was unexpected but not unprecedented, as previous research has also reported group mean cortisol levels decreasing following the still-face procedure in infancy (Tollenaar, Beijers, Jansen, Riksen-Walraven, & De Weerth, 2011). However, there was significant variability in cortisol patterns between infants. Therefore, we examined whether maternal depressive symptoms explain why some infants reacted differently to stressors. We found different cortisol reactivity patterns in different subsets of infant participants. As seen in Figure 3, the atypical U-shaped pattern was most marked in the children of mothers with high depressive symptoms and was associated with toddler behavior problems. On the other hand, children of mothers with low depressive symptoms demonstrated the more expected cortisol reactivity pattern, with a slight increase in cortisol following the stressor, followed by a recovery to baseline.

Given the state of the literature, it is hard to interpret whether the U-shaped pattern with a steeper initial decline and then a more rapid increase is indicative of dysregulated cortisol reactivity and recovery. However, our overall model demonstrated a continuous association between higher maternal postpartum depression symptoms and a higher slope infant cortisol response (i.e., steeper negative slope), which might suggest that this pattern was developed in response to the stressful experience of being reared by a depressed caregiver. This would be consistent with prior literature demonstrating a deleterious effect of chronic adversity, as other studies have also found alterations in stress reactivity following early life stress (see Bruce, Gunnar, Pears, & Fisher, 2013 for review). However, in contrast with our results, previous studies have reported that infants of highly depressed mothers show a heightened cortisol reaction (Brennan et al., 2008; Feldman et al., 2009). Variability in findings regarding cortisol response among at-risk children has been noted previously (Strüber, Strüber, & Roth, 2014; Tarullo & Gunnar, 2006) and may be explained by methodological differences. These prior studies of cortisol reactivity in infants of depressed mothers (Brennan et al., 2008; Feldman et al., 2009) used between group designs to compare children of clinically depressed mothers to children of healthy mothers, while we used a community sample in the current study. Additionally, different stressor paradigms were used in past studies (noise burst and arm restraint in Brennan et al., 2008 and a Laboratory Temperament Assessment Battery fear episode in Feldman et al., 2009). These

differences might explain the contrasting results (also see below for a discussion of potential confounds of our stressor task). As others have pointed out (Bruce et al., 2013; Strüber et al., 2014; Tarullo & Gunnar, 2006) additional contributing factors to variability in results might include timing of adversity, age, comorbidities, genetic factors, or other individual differences.

Our results are somewhat consistent with a hypocortisolism hypothesis (Gunnar & Vazquez, 2001; Heim et al., 2000). Although infants in this risk group did not show a complete lack of response, they showed an immediate decrease followed by a later increase. It is possible that infants who are faced with the ongoing stress of being cared for by a depressed caregiver experience sensitization of the HPA axis, resulting in rapid cortisol decrease due to negative feedback. Most studies examining infant cortisol reactivity to a stressor only looked at group effects, and did not examine environmental moderators of reactivity patterns. Thus, it is possible that subgroup patterns were masked by group level analysis in the majority of studies (most of which use generally lower risk samples). In fact, most studies acknowledged the presence of differential patterns (e.g., responders versus nonresponders; Jansen et al., 2010) but were not always able to shed light on these individual differences. One study that examined cortisol reactivity in preterm infants found a similar reactivity pattern to our findings (a steep initial decline following the stressor, followed by a return to baseline) that differentiated extremely preterm infants (born at 24–28 weeks) from other preterm or full-term infants, and also predicted to psychopathology (Brummelte et al., 2011). The authors speculated that NICU related early stress accounted for such differential HPA axis reactivity pattern. The stress experienced by infants of depressed mothers may affect the HPA axis in a similar way. Another well validated environmental moderator of cortisol reactivity is infant attachment security. However, in contrast to our findings, insecure or disorganized attachment patterns (high-risk groups) predict heightened cortisol reactivity patterns (Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996).

Finally, another possible contribution to differential HPA reactivity might be differential sensitivity to novelty among children of depressed mothers. In other words, children of mothers with varying depression levels experienced the encounter with the experimenter and the still-face procedure as differentially novel and/or stressful. Specifically, infants of mothers with high depression symptoms including withdrawal and anhedonia may not encounter as many new adults as children of healthy mothers, and thus could experience the introduction of the experimenter as more stressful. The timing of the baseline collection in our study (approximately 15 min after arrival of the experimenter to the home) may have captured a cortisol response to the introduction of an unfamiliar adult, which could account for the relatively (within group) higher cortisol values. However, contrary to this argument, mean cortisol levels in this group were not significantly higher at baseline than levels among infants of mothers without postpartum depression. It remains possible that the similar values in this group reflect a cortisol reaction from a lower (unmeasured) "true" baseline, consistent with a hypocortisolism pattern. Further research with careful examination of infant's cortisol measured over a longer period of time or collected by parents in the absence of a novel adult would help elucidate this effect.

Additionally, depressed mothers would likely show more flat affect than healthy mothers in their day-to-day interactions with their infants, so these infants might respond less strongly to the still-face procedure as they may be more "used to" the lack of affect. Field et al. (2007) found that infants of depressed mothers showed less

distress behaviors during the still-face paradigm than infants of healthy mothers. The lack of cortisol response might in fact be an adaptive response to a mother with frequent flat affect. In contrast, infants with mothers who are not depressed might not be used to frequent flat affect and thus be more reactive to the novelty of their mother's neutral facial expression in the still-face paradigm. It might be inherent in psychosocial stress paradigms that the subjective experience of the stressor differs across individuals. There is wide variability in cortisol reactivity in infants across studies (Jansen et al., 2010). In a systematic review, Jansen and colleagues found that results differed depending on infant age, the stress paradigm (particularly physical versus psychosocial), and the opportunity for caregiver buffering of the stress response. Additional variation between studies using the same paradigm could not be accounted for, but might represent individual differences among participants and the populations studied (Jansen et al., 2010). Future research exposing the same participants to a diverse range of stressors could tease apart these effects.

Finally, while the period following the still-face procedure was intended to be low stress for the dyad to allow for recovery, it might have been experienced as stressful for a subset of infants. For example, mothers with high depressive symptoms might have been distressed by answering some of the questionnaires related to their mental health and this might have affected the responsiveness to their infants during this time. Further research examining both diurnal cortisol and reactivity in infants in relation to maternal postpartum depression is needed. Longitudinal assessment could also shed light on patterns of hyper- versus hypocortisolism over time. Future research should also continue to address nonnormative cortisol trajectories in at-risk populations in order to better investigate adaptive versus maladaptive outcomes.

In testing our main aim, we found that infant cortisol regulation mediated the effect of maternal depression on increased toddler symptomatology, consistent with our hypothesis. Based on the confidence interval approach, this effect was significant for internalizing problems but not significant for externalizing problems; however, the difference was likely negligible and primarily due to power (see limitations below). Overall, our findings supported and extended previous research demonstrating that changes in infant HPA regulation precede the development of child psychopathology in contexts of risk (Goodman & Gotlib, 1999; Keenan, 2000; Silk, Shaw, Skuban, Oland, & Kovacs, 2006). Past research has shown a mediational role of HPA activity in childhood and adolescence (Brooker et al., 2016; Halligan et al., 2007). Thus, HPA dysregulation has been proposed as a marker of neurodevelopmental vulnerability due to potential chronic brain exposure to neurotoxic effects of cortisol, which might impair systems associated with emotional and behavioral regulation (e.g., cortico-limbic circuits; Gunnar & Quevedo, 2007). Our results contribute evidence that this mechanism of effect begins early in infancy and places children on a trajectory toward emotional and behavioral problems beginning in toddlerhood. The longitudinal measurement of problems a year following measurement of HPA activity, and controlling for later maternal depression, highlights the importance of depression during the postpartum period and supports the hypothesized causal relationship between dysregulated HPA activity and later problems. These findings suggest that early imprinting of the HPA system via stress experienced when mothers are depressed have deleterious, lasting effects in toddlerhood.

Although infant socioemotional problems remain understudied, it is important to address internalizing and externalizing problems among this age group, as infants

growing up in high-risk environments can experience clinically significant impairment (Van Zeijl et al., 2006). One study found that 35% of 12- to 18-month-olds referred to protective services for child maltreatment allegations have clinically significant behavioral and emotional problems (Horwitz et al., 2012). Moreover, high levels of internalizing and externalizing problems during infancy persisted in the short term (1 year later; Briggs-Gowan, Carter, Bosson-Heenan, Guyer, & Horwitz, 2006) and predicted later emotional and behavioral disorders (school age; Briggs-Gowan & Carter, 2008), particularly among children exposed to high levels of family stress (Campbell, Shaw, & Gilliom, 2000). Our findings fit well with other studies that have also confirmed early infancy precursors to later psychopathology in contexts of maternal depression, even when controlling for concurrent maternal depression, highlighting that early care experiences are indeed a sensitive period for regulatory development (Moore et al., 2001).

Results also indicate that maternal depression in the postpartum period affects infants' cortisol and later behavior problems across the full spectrum of depressive symptoms, not just in circumstances of clinical levels of chronic depression. This has implications for intervention efforts. Early screening and prompt treatment for depressive symptoms in postpartum, even if symptoms are not severe, is essential for preventing deleterious effects on infant biological and psychological functioning. Reductions in maternal depression have been shown to mediate the effect of successful interventions on child behavior problems (Shaw, Connell, Dishion, Wilson, & Gardner, 2009). Future research should also examine whether changes in maternal depression lead to changes in child HPA activity and whether these changes explain the improvement in behavior.

These results also contribute to the body of literature examining the nature of externalizing versus internalizing symptoms in early childhood. Our models suggest a similar path from dysregulated HPA activity to both internalizing and externalizing symptoms, contrary to some findings that show different directionality in the relationships between HPA activity and internalizing versus externalizing symptomatology (Goodyer, Tamplin, Herbert, & Altham, 2000; Shirtcliff et al., 2005; Smider et al., 2002). The marginally higher estimate for internalizing symptoms may indicate a slightly stronger relationship; however, the direction and scale of the effect are quite similar across symptomatology. This joint pathway may diverge at later points in development based on child factors (e.g., temperament) or environmental factors (e.g., parenting).

The potential mechanisms of this mediational effect remain to be fully examined. It might be that mothers who are depressed are disengaged and less sensitive in their parenting resulting in more instances of infant stress and chronic activation of the HPA system (Goodman & Gotlib, 1999; Harnish, Dodge, & Valente, 1995). In this context, mothers and their infants would be less likely to engage in positive co-regulatory strategies that support early coping and buffer the effects of stress on the developing child (Hostinar, Sullivan, & Gunnar, 2014). More conscious and effortful forms of self-regulation build upon these unconscious and automatic processes of physiological regulation (Calkins & Hill, 2007). Behavioral and emotional self-regulation in turn have been implicated in both internalizing and externalizing disorders (Buckner, Mezzacappa, & Beardslee, 2009; Eisenberg, Spinrad, & Eggum, 2010). Alternatively, it might be that women who are depressed during postpartum have a genetic predisposition to biological sensitivity to negative stimuli, which is then passed on to their

offspring. It is likely a combination of both behavioral and biological pathways contributing to the intergenerational passing of risk (Goodman, 2003).

Limitations

Though the findings in the current study were robust, there were limitations to note. First, mothers reported on both their depression symptoms and toddler behavior problems, introducing potential mono-reporter bias. Past research has shown that mothers experiencing depression tend to report higher levels of children's problem behavior (Fergusson, Lynskey, & Horwood, 1993). To attempt to combat this effect, our analyses included 18-month maternal depression in order to control for this possibility. Additionally, the relation of symptoms to infant HPA activity strengthened our assumption that we were capturing meaningful variance in child behavior despite the potential error introduced by maternal interpretations of behavior. Future research could incorporate daycare provider or observer reports to shed light on whether problem behaviors were limited to maternal perceptions and/or the home environment.

Second, research has demonstrated that biobehavioral factors during pregnancy impact the development of the HPA system (Brennan et al., 2008), and the current study did not address prenatal depression or maternal HPA axis functioning during pregnancy. Future studies should measure depression and maternal HPA functioning starting in pregnancy and across the postnatal period to fully elucidate the contribution of depression at different stages. Third, our sample size was relatively modest leading to difficulties with power, particularly for the mediation analyses. While the mediation effect for externalizing symptoms was not significant according to the confidence interval, many scholars caution against strict adherence to significance cutoffs (Wasserstein, 2016). Finally, our study did not measure possible environmental mediators of the effect of maternal depression on infant outcomes, such as parenting behavior. Future research should examine multiple biological and environmental mediators as well as potential moderating factors to fully understand this relationship.

CONCLUSIONS

Despite these limitations, the current study contributed valuable evidence of a mediational pathway of infant HPA regulation on the effect of maternal postpartum depression on toddler emotional and behavioral problems. Interventions should target early detection and treatment of maternal depression to prevent the cascade to infant psychophysiological regulation and later outcomes.

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CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

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