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Perinatal depression influences on infant negative affectivity: Timing, severity, and co-morbid anxiety

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ABSTRACT

Accumulating evidence suggests that antenatal depression predicts infants' negative affectivity, albeit with variable effect sizes. With a prospective longitudinal design, we sought to explain that variability by addressing questions about timing of the depression across pregnancy and the early postpartum, the role of high symptom levels relative to diagnosed depression, comorbidity with anxiety, and the potential mediating role of neuroendocrine functioning. Primiparous women ($n = 77$) with histories of depression prior to pregnancy were assessed for cortisol levels monthly beginning by mid-pregnancy. Depression symptom levels and diagnostic status were similarly assessed monthly in pregnancy and also until infants reached three months of age, when mothers completed the Infant Behavior Questionnaire-Revised to measure infant negative affectivity. Antenatal depression symptoms and infant negative affectivity were positively associated ($r = .39$). Controlling for depression symptom levels in other trimesters, only second trimester depression symptoms predicted higher infant negative affectivity ($\beta = .44$). With postpartum depression symptom levels in the model, only antenatal depression symptoms predicted infant negative affectivity ($\beta = .45$). In the context of depression, neither antenatal anxiety symptoms nor anxiety disorder diagnosis were associated with infant NA scores. The hypothesized role of elevated maternal cortisol as a mechanism for the association between antenatal depression and infant NA was not supported. Our findings contribute to efforts to more precisely identify infants of perinatally depressed mothers who are at greater risk for elevated negative affectivity, suggesting a window of vulnerability in mid pregnancy and the need for further study of potential mechanisms.

Negative affectivity (NA) is the superordinate construct that reflects the pervasive individual differences in one's propensity to experience negative emotions (Watson & Clark, 1984). NA in infants is of particular concern for several reasons. First, NA can be reliably identified as early as three months of age (Gartstein & Rothbart, 2003) and is relatively stable over time, even from early infancy (Lee & Bates, 1985; Putnam, Rothbart, & Gartstein, 2008; Roberts & DelVecchio, 2000). Second, elevated levels of NA in infants are prospectively associated with symptoms of depression and anxiety, including internalizing symptoms at age 2 (Putnam & Stifter, 2005), internalizing and externalizing symptoms at age 4 (Gartstein, Putnam, & Rothbart, 2012), and fear, shyness, sadness, and anger/frustration at 7 years of age (Rothbart, Derryberry, & Hershey, 2000). Third, high NA in infancy is of concern because of its potential role in transactional processes (Pesonen et al., 2008; Sameroff,

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1975). That is, infants high in NA may shape their environments by evoking negative responses from their caregivers, with these transactional processes in turn potentially contributing to the development of psychopathology.

Given that this measurable and stable construct has important links to the later development of psychopathology and emerges so early in development, researchers have attempted to identify its predictors. Among potential predictors, much interest has focused on pregnancy, given theory and evidence from animal and human studies that prenatal exposures may alter fetal development of neuroregulatory systems related to aspects of behavioral and emotion regulation central to the concept of temperament (DiPietro, 2012; Monk, Webb, & Nelson, 2001). Consistent with these ideas, in multiple studies, antenatal depression has been identified as a predictor of NA in infants (Austin, Hadzi-Pavlovic, Leader, Saint, & Parker, 2005; Cutrona & Troutman, 1986; Davis et al., 2007; Davis et al., 2004; Huot, Brennan, Stowe, Plotsky, & Walker, 2004). However, effect sizes have ranged broadly among the published studies, from $r = .11$ to $r = .52$. A likely factor in that variability of effect sizes is that antenatal depression is typically treated as a unitary factor, defined either as meeting diagnostic criteria or exceeding established cut scores for clinically significant levels of symptoms. Yet the construct of 'depression' comprises broad variability in terms of clinical characteristics (Boland & Keller, 2009). Thus, by taking that variability into account, we may be able to help explain for whom or under what situations antenatal depression will be associated with infant NA. We sought to understand what it is about maternal depression that matters for infant NA by testing hypotheses on theory-based aspects of antenatal depression that would be expected to be related to NA in infants, including the timing of the antenatal depression, whether depression is defined diagnostically or by high symptom levels, comorbidity with anxiety, and the role of the often correlated postpartum depression. In addition, we sought to expand on knowledge of potential mechanisms for the association between antenatal depression and infant NA by testing the potential mediating role of neuroendocrine functioning.

Greater understanding of the role of clinical characteristics of the mother's depression on infant NA has the potential to: (1) help to explain the variability in effect sizes that has been found in tests of association between antenatal depression and infant NA and (2) highlight subgroups of infants who are at greater risk for elevated NA relative to others, based on the qualities of the mothers' depression.

First, looking at the timing of antenatal depression, the literature has been somewhat mixed. Huot et al. (2004) found that depression in the first or second trimester (combined due to their small sample of women with first trimester depression) was significantly associated with NA while depression in the third trimester was not, whereas Davis et al. (2007) found significant although small associations between antenatal depression and 2-month-old infants' "negative reactivity" (measured by the Fear subscale of the Infant Behavior Questionnaire-Revised) regardless of whether depression was measured during the second or third trimester. The discrepancy in findings may be explained by Huot et al. (2004) having measured NA in offspring ranging from 6 months to 5 years of age. Further, there is some support for an increase in heritability of NA across development. In particular, a study of twin neonates found the heritability estimates of NA—like neurobehavioral indices to be no different than zero (Riese, 1990), while a longitudinal study found heritability to increase across twelve to thirty months of age (Matheny, 1989). This suggests that there might be different predictors of NA at different points in development. Therefore, we sampled infants at 3 months of age exclusively, the earliest point in development at which NA can be reliably measured (Gartstein & Rothbart, 2003), so as to delineate predictors of NA at this precise point in development. Our study further aimed to help resolve these disparate findings regarding the role of timing of antenatal depression exposure in predicting infant NA by assessing depression at multiple timepoints throughout pregnancy. In the context of the mixed findings, we also took into consideration knowledge that the neurons involved in the relevant neural circuits (e.g. limbic system and associated regions of the cortex) proliferate, differentiate, and migrate between the eighth and 24th week of gestation (Nowakowski & Hayes, 2002), and thus hypothesized that infants would be most adversely impacted by second trimester depression.

The disparate findings on timing may also be attributable to the approach to measurement of NA. Although both Huot et al. (2004) and Davis et al. (2007) measured NA with the Infant Behavior Questionnaire (IBQ) (Rothbart, 1981), Davis et al. (2007) relied on only one of the four subscales (Fear) within the IBQ-derived NA dimension. In an effort to bridge the gap between these two studies, we tested primary hypotheses with the NA dimension of the IBQ-R, but also explored associations with the subscales that comprise that dimension. We expected to replicate the Davis et al. finding on the fear subscale and examined the other three subscales in an exploratory manner.

Second, we sought to explore the extent to which clinically significant depressive symptomatology that falls short of meeting Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (DSM-IV) (American Psychiatric Association, 1994) Major Depressive Episode (MDE) criteria contributes to infant NA. Women with sub-clinical depression during pregnancy have been found to not differ significantly from women with diagnosed depression on stress, anxiety, and severity of depression symptoms (Goodman & Tully, 2009). In a study comparing neonatal behavioral functioning of infants, although infants of women with antenatal depression showed poorer neonatal neurobehavioral functioning than infants of women with no antenatal depression, infants of women with MDE did not differ from infants of women with sub-clinical depression (Goodman, Rouse, Long, Ji, & Brand, 2011). Consistent with this evidence, we hypothesized that infants of women with depression during pregnancy, whether defined by high depression symptoms alone or MDE diagnosis, would have significantly higher levels of NA than infants of women who did not experience clinically-significant depression during pregnancy.

A third unanswered question about the association between antenatal depression and infant NA is the role of comorbid antenatal anxiety. In previous findings from the sample from the present study, we found that nearly 40% of women who