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Prenatal depression effects and interventions: A review

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ABSTRACT

This review covers research on the negative effects of prenatal depression and cortisol on fetal growth, prematurity and low birthweight. Although prenatal depression and cortisol were typically measured at around 20 weeks gestation, other research suggests the stability of depression and cortisol levels across pregnancy. Women with Dysthymia as compared to Major Depression Disorder had higher cortisol levels, and their newborns had lower gestational age and birthweight. The cortisol effects in these studies were unfortunately confounded by low serotonin and low dopamine levels which in themselves could contribute to non-optimal pregnancy outcomes. The negative effects of depression and cortisol were also potentially confounded by comorbid anxiety, by demographic factors including younger age, less education and lower SES of the mothers and by the absence of a partner or a partner who was unhappy about the pregnancy or a partner who was depressed. Substance use (especially caffeine use) was still another risk factor. All of these problems including prenatal depression, elevated cortisol, prematurity and low birthweight and even postpartum depression have been reduced by prenatal massage therapy provided by the women's partners. Massage therapy combined with group interpersonal psychotherapy was also effective for reducing depression and cortisol levels. Several limitations of these studies were noted and suggestions for future research included exploring other predictor variables like progesterone/estriol ratios, immune factors and genetic determinants. Further research is needed both on the potential use of cortisol as a screening measure and the use of other therapies that might reduce prenatal depression and cortisol in the women and prematurity and low birthweight in their infants.

Prenatal depression has contributed to prematurity and low birthweight (Li, Liu, & Odouli, 2009; Marcus, 2009). Some have suggested that high prenatal cortisol levels associated with depression may result in elevated fetal cortisol, delayed fetal growth and prematurity (Van den Berg, Mulder, Mennes, & Glover, 2005) as well as impaired fetal brain development (Weinstock, 2005). Elevated norepinephrine and uterine artery resistance have also been implicated in fetal growth delays (Teixeira, Fisk, & Glover, 1999). Uterine artery resistance would limit uterine blood flow and thereby limit the supply of oxygen and nutrients to the fetus resulting in growth deprivation and shorter gestation (Maina et al., 2008; Sjostrom, Valentin, Thelin, & Marsal, 1997).

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1. Prenatal depression increases prematurity and low birthweight

Higher cortisol levels in prenatally depressed women have been noted in several samples (see Field & Diego, 2008 for a review). In addition, depressed women have lower dopamine and serotonin levels and are more likely to deliver prematurely and have low birthweight infants (Field et al., 2004a). The newborns of the depressed mothers in the Field et al. (2004a) study also had higher cortisol levels and lower dopamine and serotonin levels, thus mimicking their mothers' prenatal levels. And, in another study from our lab, the mothers' cortisol levels at 26 weeks accounted for 40% of the variance in the newborns' cortisol levels (Diego et al., 2004). Also, like their mothers, the newborns of depressed mothers had other signs that have been associated with chronic depression including greater relative right frontal EEG activation and lower vagal activity (Field et al., 2004a). Finally, they received lower scores on the Brazelton Neonatal Behavioral Assessment Scale. A path analysis on the effects of the mothers' prenatal depression and biochemical profile on their newborns' gestational age and birthweight suggested a path from prenatal depression to prenatal cortisol and norepinephrine levels, and, in turn, a path from prenatal cortisol to prematurity and low birthweight. These studies led to the following research including additional mechanism studies.

2. Prenatal cortisol contributes to fetal growth delays and prematurity

To further investigate the prenatal cortisol effects, relationships were assessed between the mothers' prenatal depression and biochemistry and fetal activity, fetal growth and neonatal outcomes (Diego et al., 2006; Field, Diego, Hernandez-Reif, Gil, & Vera, 2005a; Field et al., 2006c). In these studies, growth measures were taken on fetuses of depressed and non-depressed women during ultrasound sessions at approximately 16–20 weeks gestation. On fetal growth measures (estimated weight, femur length, abdominal circumference, biparietal diameter, and head circumference) the fetuses of depressed versus those of non-depressed women showed growth delays (see Fig. 1). Growth measures, in turn, were inversely related to fetal activity and low birthweight. In addition, 24% of the fetuses of depressed mothers (versus 7% of the fetuses of non-depressed mothers) were born prematurely. Prenatal cortisol levels have been notably predictive of preterm birth at an even earlier gestational age (Sandman et al., 2006). In that study, cortisol levels were higher at 15 weeks gestation and at 19 weeks gestation in women who later delivered preterm infants.

Others have reported similar relationships between prenatal depression and decreased fetal growth (Hoffman & Hatch, 2000) and between cortisol and shorter gestation by others (Dayan et al., 2006). However, even though approximately 10% of maternal cortisol is thought to cross the placenta (Gitau, Cameron, Fisk, & Glover, 1998), other data are not definitive (Shea et al., 2007).

3. Stability of mood states and cortisol across pregnancy

Cortisol and mood states across pregnancy appear to be stable (from 20 to 32 weeks gestation) and cortisol was related to both depression and anxiety (Field, Hernandez-Reif, & Diego, 2006a). Significant stability was noted between the 20-week and the 32-week measures including depression, anxiety and cortisol. These were, in turn, correlated with each other and

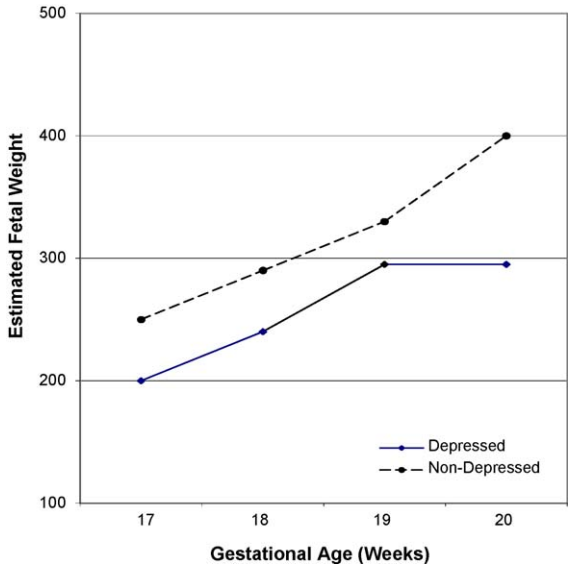

Fig. 1. Estimated weight of fetuses of depressed and non-depressed mothers across early gestation.