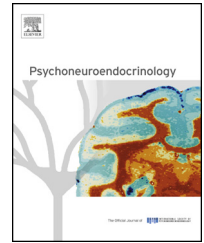




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# Prenatal stress, partner support, and infant cortisol reactivity in low-income Mexican American families



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**Summary** Maternal exposure to significant prenatal stress can negatively affect infant neurobiological development and increase the risk for developmental and health disturbances. These effects may be pronounced in low SES and ethnic minority families. We explored prenatal partner support as a buffer of the impact of prenatal stress on cortisol reactivity of infants born to low-income Mexican American women. Women ( $N = 220$ ; age 18–42; 84% Spanish-speaking; 89% foreign born; modal family income \$10,000–\$15,000) reported on economic stress and satisfaction with spousal/partner support during the prenatal period (26–38 weeks gestation), and infant salivary cortisol reactivity to mildly challenging mother–infant interaction tasks was assessed at women's homes at six weeks postpartum. Multilevel models estimated the interactive effect of prenatal stress and partner support on cortisol reactivity, controlling for covariates and potential confounds. Infants born to mothers who reported high prenatal stress and low partner support exhibited higher cortisol reactivity relative to those whose mothers reported high support or low stress. The effects did not appear to operate through birth outcomes. For low-income Mexican American women, partner support may buffer the impact of prenatal stress on infant cortisol reactivity, potentially promoting more adaptive infant health and development.

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The foundations for lifespan health begin in the prenatal environment. For low-income and ethnic minority women, the prenatal period can be associated with a number of physical, emotional, social, and financial stressors (Bloom et al., 2013; Bermúdez-Millán et al., 2011; Dominguez et al., 2008). Stressful experiences have negative implications for mothers' well-being, but can also have deleterious consequences for fetal and infant development. Prenatal stress can be conceptualized as a teratogen, a

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factor that can directly affect the developing fetus and is capable of interfering with normal development (DiPietro, 2012). Effects of prenatal stress have been documented on a wide range of infant outcomes including low birth weight, pre-term birth, dysregulated neurobiological activity, and behavioral, emotional, and neurodevelopmental disturbances (Lazinski et al., 2008). The impact of prenatal stress can persist into childhood, adolescence, and adulthood (Talge et al., 2007).

The development of infant physiological stress response systems represents a potential mechanism linking prenatal exposures to infant health and developmental outcomes. The hypothalamic–pituitary–adrenocortical (HPA) axis, a primary mediator of the stress response, regulates the production and release of the glucocorticoid hormone cortisol. Scholars investigating associations between maternal stress and infant birth, health, and developmental outcomes have described a “fetal programming” hypothesis, wherein high stress during pregnancy results in an adverse intrauterine environment that can negatively impact infant growth and contribute to a variety of negative health effects across the lifespan. In part, prenatal stress may affect fetal development via stress-related activation of the maternal HPA axis, resulting in transmission of cortisol across the placenta (Reynolds, 2013). High maternal stress and resulting excess fetal cortisol exposure is believed to disrupt the normal adaptive function of coregulatory maternal stress systems (nervous, endocrine, and immune) that support healthy fetal development (Coussons-Read, 2012). Interference in the normal development of the HPA axis may disrupt subsequent capacities for physiological and behavioral regulation (Weinstock, 2005). O’Connor et al. (2013) reported that higher cortisol exposure in utero was associated with dysregulated cortisol reactivity in infancy.

Deleterious effects on birth outcomes represent a potential mechanism through which prenatal stress may be linked to HPA regulation and later infant health and developmental outcomes, although evidence for such a pathway is limited. Reynolds (2013) outlined a theoretical model in which low birth weight mediates the effects of maternal distress on child HPA dysregulation, which in turn increases the risk of obesity and metabolic disorders later in life. A fairly consistent literature identifies prenatal stress, depression, and anxiety as risk factors for preterm birth and low birth weight (Dunkel Schetter, 2011). However, the role of birth outcomes in the relation of prenatal stress to infant HPA axis activity is less clear. Field et al. (2004) reported that low birth weight and prematurity did not explain the poorer neonatal outcomes (i.e., less habituation, less orientation, poorer motor skills, and decreased autonomic stability) observed in infants exposed to maternal prenatal depression.

The impact of prenatal stress may be accentuated in low-income Mexican or Mexican American families. Hispanic women commonly face stressors such as discrimination, occupational barriers, language barriers, and deportation fears (Cervantes et al., 1991). Compared to Caucasian families, Hispanic families are more than twice as likely to live below the poverty level (\$21,954 for a family of 4; 22.7% of Hispanic families compared to 9.3% of Caucasian respondents; US Census Bureau, 2012), and considerable research documents deleterious effects of poverty on birth outcomes,

child stress physiology, and child health and developmental outcomes (Blair et al., 2011; Evans, 2003). However, despite high traditional risk factors, Hispanic immigrant women tend to have more positive birth outcomes compared to African American and other ethnic minority groups in the US (i.e., lower incidence of low birth weight infants, fewer preterm births), and comparable or lower risk for low birth weight compared to non-Hispanic white groups (Flores et al., 2012; McGlade et al., 2004). Better than expected birth outcomes may reflect a Healthy Migrant effect, which posits that health benefits are due to better mental and physical health of those who choose to emigrate compared to those who stay. Alternatively, the “Latina Paradox” hypothesizes that Latinas in the US experience protective cultural factors that promote resilience in the presence of stressful conditions (McGlade et al., 2004), potentially translating into better birth outcomes. Powerful culture-specific values such as strong family and kinship ties and a high value placed on the maternal role may provide protective emotional and practical support for pregnant Latina women.

Support from a romantic partner or spouse is a particularly salient protective factor for pregnant women (Rini et al., 2006). For low-income Mexican American women, strong cultural values relating to the family may enhance the protective effects of support during pregnancy (Bender and Castro, 2000; Diaz et al., 2007; Campos et al., 2008). Further, partner support during pregnancy may promote subsequent infant well-being. More perceived support from a partner has been associated with reduced emotional distress among mothers and distress to novelty among their six to eight week infants (Stapleton et al., 2012). With regards to infant cortisol, existing studies have predominantly focused on postpartum partner support, with relatively consistent findings that partner relationship functioning can significantly affect child cortisol (e.g., Pendry and Adam, 2007).

Many of these studies have been limited by cross-sectional designs that preclude the ability to determine the directionality of influence. DiPietro (2012) notes a number of related methodological issues in the inference of causality in research on the implications of maternal stress for infant outcomes. These issues include a reliance on subjective maternal reports for both predictor (e.g., stress) and outcome (e.g., child health), shared genetic contributions, and potential confounding by other prenatal and postnatal exposures and behaviors. For example, despite being unique psychological constructs, maternal depression and stress are generally significantly correlated, creating a challenge to empirically separate their effects. Further, effects of prenatal stress are difficult to disentangle from effects due to stress in the postpartum environment. In short, while intriguing evidence supports hypothesized causal effects of prenatal stress and support on infant development, longitudinal research beginning in the prenatal period that addresses potential confounding factors is needed to better understand the transmission of risk and protective influences from mother to infant.

Existing literature points to negative influences of prenatal stress and positive influences of support on infant health and developmental outcomes, but little is known about the extent to which partner support may buffer the impact of stress on infant outcomes, particularly in ethnic

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