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Maternal intimate partner violence exposure, child cortisol reactivity and child asthma☆

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ABSTRACT

Psychosocial stressors like intimate partner violence (IPV) exposure are associated with increased risk of childhood asthma. Longitudinal studies have not investigated the role of hypothalamic-pituitary-adrenal (HPA) axis reactivity (and associated alterations in cortisol release) in the child IPV exposure–asthma association. We sought to investigate this association, and to assess whether this relationship differs by child HPA reactivity. This secondary analysis used longitudinal cohort data from the Family Life Project. Participants included 1,292 low-income children and mothers; maternal interview and child biomarker data, including maternal report of IPV and child asthma, and child salivary cortisol obtained with validated stress reactivity paradigms, were collected when the child was 7, 15, 24, 35, and 48 months. Using structural equation modeling, maternal IPV when the child was 7 months of age predicted subsequent reports of childhood asthma ($B = 0.18$, $p = .002$). This association differed according to the child's HPA reactivity status, with IPV exposed children who were HPA reactors at 7 and 15 months of age – defined as a $\geq$10% increase in cortisol level twenty minutes post peak arousal during the challenge tasks and a raw increase of at least .02 μg/dl – being significantly at risk for asthma (7 months: $B = 0.17$, $p = .02$; 15 months: $B = 0.17$, $p = .02$). Our findings provide support that children who are physiologically reactive are the most vulnerable to adverse health outcomes when faced with environmental stressors.

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Asthma is a significant public health challenge, with ~9% of children diagnosed with this chronic condition (Myers & Tomasio, 2011). Asthma remains one of the top causes of pediatric ambulatory visits and hospitalizations, costing the health care system more than $15 billion annually (Clark, 2011). The development of asthma stems from a complex interaction of risk factors that has not been completely elucidated, though exposure to psychosocial stress is increasingly being implicated as a source of risk (Herman, 2011). The National Heart, Lung and Blood Institute (NHLBI) states that "stress can. . . possibly act as a risk factor for an increase in the prevalence of asthma" (p. 181), as well as a risk factor for asthma exacerbations ("National Asthma Education and Prevention Program Expert Panel Report 3: Guidelines for the Diagnosis and Management of Asthma," 2007). NHLBI also acknowledges the role of psychosocial stress in asthma exacerbations (Sandberg et al., 2000).

Few psychosocial stressors have the potential to impact health more than intimate partner violence (IPV) exposure. Over 15 million children in the US are exposed to IPV each year, and children less than five are disproportionately represented in homes with IPV (Fantuzzo, Boruch, Beriama, Atkins, & Marcus, 1997; McDonald, Jouriles, Ramisetty-Mikler, Caetano, & Green, 2006). Young children are dependent on their primary caregiver (Davies & Woitach, 2008); thus, their immediate safety may be threatened by IPV, and direct exposure to violence against a primary caregiver is distinctly traumatic (Davies & Cummings, 1994). Finally, in homes with IPV, care-giving relationships – an important buffer to the effects of stressors – are often disrupted (Davies & Cummings, 1994; Johnson, Riley, Granger, & Riis, 2013).

The impact of family psychosocial stressors, like IPV, on children's asthma is related to alterations in children's stress response, particularly the hypothalamic-pituitary-adrenal (HPA) axis (Dougherty, Klein, Rose, & Laptook, 2011; Gump et al., 2009; Moss, Vanyukov, Yao, & Kirillova, 1999). The development of the HPA axis is under strong social regulation throughout early childhood (Dougherty et al., 2011; Evans & Kim, 2007). When children experience stressful events, the HPA axis is activated resulting in release of cortisol from the adrenal glands. Acute HPA axis activation is imperative in the short-term, allowing children to mobilize physiological resources to meet environmental challenges. In the long-term, however, repeated activation may lead to dysregulation and increased risk for disease (Gunnar & Quevedo, 2007; Miller, Chen, & Cole, 2009; Wright, Cohen, & Cohen, 2005). HPA dysregulation due to chronic stress can lead to exaggerated or blunted cortisol reactivity, depending on the nature, timing, and severity of the stressor (Miller, Chen, & Zhou, 2007; Slopen, McLaughlin, & Shonkoff, 2014).

One way to measure the HPA axis is to assess salivary cortisol reactivity to a laboratory stressor (Gunnar, Talge, & Herrera, 2009). In young children, these stressors include exposure to a novel person or frustrating task (Gunnar et al., 2009). The limited number of studies exploring HPA reactivity to a laboratory stressor in children with and without IPV exposure have yielded mixed results, with some demonstrating exaggerated cortisol responses, while others demonstrate no difference between groups (Hibel, Granger, Blair, Cox, & Investigators, 2009, 2011).

With regard to asthma expression, chronic HPA activation and the associated cortisol release can impact neuroendocrine regulation of immune and inflammatory processes (Chen & Miller, 2007; Wright, 2011; Wright et al., 2005). Repeatedly activating the stress response system may trigger hormone release that favors T helper 2 cell (Th2) versus T-helper cell 1 (Th1) predominance (Wright et al., 2005). An imbalance between the cytokines associated with Th2 and Th1 lymphocytes has been associated with asthma development (Wright et al., 2005). In addition, Miller and Chen (2006) reported that asthmatic children experiencing chronic stress had a 5.5-fold reduction in glucocorticoid receptor mRNA expression. Diminished glucocorticoid receptor expression can lead to excessive inflammation and increased airway reactivity.

Although evidence links childhood IPV exposure and childhood asthma (Breiding & Ziembroski, 2011; Suglia, Enlow, Kullowatz, & Wright, 2009) and alterations in cortisol and asthma (Chen & Miller, 2007; Wright, 2011; Wright et al., 2005), we are not aware of literature investigating the role, over time, of cortisol reactivity in the association between IPV and child asthma in young children. We therefore conducted a secondary analysis using data collected as part of the Family Life Project (FLP) prospective cohort study which enrolled families at the time of a child's birth, with interview and/or biomarker data collected when the target child was 7, 15, 24, 35, and 48 months. Our objectives were to determine the association between reports of maternal IPV and childhood asthma, and to assess whether this association differed by child cortisol reactivity.

Methods

Human Subjects

The parent study and the current analyses were institutional review board approved. Participants enrolling in the initial FLP study provided written informed consent.

Study Design, Setting and Sample

This secondary analysis examined data collected for the FLP. Details of the FLP design have been described elsewhere (Blair et al., 2011; Hibel et al., 2009). FLP is a population-based longitudinal cohort study examining the multiple levels that influence the development of rural children. Complex sampling procedures were used to recruit a representative sample of 1,292 families from North Carolina (NC) and Pennsylvania (PA) at the time of birth. Low-income families in NC and PA, and African American families in NC were over-sampled. In both states, recruitment occurred daily in hospitals from September