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Article

Maternal experiences of intimate partner violence and C-reactive protein levels in young children in Tanzania

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ABSTRACT

Intimate partner violence (IPV) is a critical public health issue that impacts women and children across the globe. Prior studies have documented that maternal experiences of IPV are associated with adverse psychological and physical health outcomes in children; however, research on the underlying physiological pathways linking IPV to these conditions is limited. Drawing on data from the 2010 Tanzania Demographic and Health Survey, we examined the relationship between maternal report of IPV in the past 12 months and inflammation among children ages 6 months to 5 years. Our study included 503 children who were randomly selected to provide a blood sample and had a mother who had ever been married and who had completed the Domestic Violence Module, which collected information on physical, sexual, and emotional violence. Analyses were stratified based on a threshold for acute immune activation status, defined by the threshold of CRP > 1.1 mg/L for young children in Tanzania. In bivariate analyses, healthy children whose mothers reported IPV showed a marginally elevated median CRP level compared to children whose mothers did not report IPV (0.35 vs. 0.41 mg/L; $p = 0.13$). Similarly, among children with active or recent infections, those whose mothers reported IPV had an elevated median CRP compared to children whose mothers did not (4.06 vs 3.09 mg/L; $p = 0.03$). In adjusted multiple variable regression models to account for child, mother, and household characteristics, maternal IPV was positively associated with (log) CRP in both healthy children and children with active or recent infection. Although longitudinal research with additional biomarkers of inflammation is needed, our results provide support for the hypothesis that inflammation may function as a biological pathway linking maternal IPV to poor psychological and physical health outcomes among children of mothers who are victimized—and this may extend to very young children and children in non-Western contexts.

Introduction

Intimate partner violence (IPV) is a critical public health issue that impacts women and children across the globe. An estimated 30–38% of women worldwide have experienced either physical or sexual violence from their intimate partners during their lifetime (World Health Organization, 2013), and women in low and middle income countries are particularly vulnerable, including in Tanzania. According to the 2010 Tanzania Demographic and Health Survey (TDHS), among women ages 15 to 49 who had ever been married, 39% report experiences of emotional violence, 36% reported experiences of physical violence, and 17% reported experiences of sexual violence by their current or most recent sexual partner or husband (National Bureau of Statistics (NBS) [Tanzania] & ICF Macro, 2011). IPV is a significant cause of mortality and morbidity among women (World Health Organization/London School of Hygiene and Tropical Medicine, 2010), and IPV also affects the children who witness this violence (Wood & Sommers, 2011). Research has linked experiences of IPV with diseases related to the cardiovascular (Coker, Smith, Bethea, King, & McKeown, 2000), reproductive (Coker et al., 2000), immune and endocrine systems (Woods et al., 2005), and to mental health (Kramer, Lorenzon, & Mueller, 2004) among adults, and to a variety of adverse psychological and physical health consequences in children (Repetti, Taylor, & Seeman, 2002a; Repetti, Taylor, & Seeman, ; 2002b; Wood & Sommers, 2011; Yount, DiGirolamo, & Ramakrishnan, 2011). To date, research on the underlying physiological pathways linking IPV to adverse psychological and physical health outcomes in children has been limited. Elevated inflammation, a marker of increased activation of innate immune

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function and the stress-response system, is implicated in the etiology of multiple physical and mental health disorders (Black & Garbutt, 2002; Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008) and may be a common mechanism linking maternal IPV to the wide range of negative outcomes associated with maternal victimization. The aim of this study is to examine the relationship between maternal experiences of IPV in the past 12 months and inflammation among children ages 6 months to 5 years, using data from 2010 TDHS.

Maternal IPV and child wellbeing

Maternal experience of IPV is a well-established risk factor for psychological problems in children (Davies, Winter, & Cicchetti, 2006; Repetti et al., 2002a, 2002b). Although there is increasing interest in the physiological consequences of childhood adversity (Shonkoff, Boyce, & McEwen, 2009; Shonkoff & Garner, 2012), only a small number of studies of children, all in Western contexts, have documented associations between family conflict and dysregulation of the stress response system (Yount et al., 2011). In prior studies, children exposed to martial conflict or domestic violence displayed lower sympathetic nervous system activity (Davies, Sturge-Apple, Cicchetti, Manning, & Zale, 2009) and vagal tone (Gottman & Katz, 1989; Rigterink, Fainsilber Katz, & Hessler, 2010), and elevated heart rate (Saltzman, Holden, & Holahan, 2005), parasympathetic nervous system activity (Davies et al., 2009), urinary dopamine (Gottman & Katz, 1989), and salivary cortisol activity (Davies, Sturge-Apple, Cicchetti, & Cummings, 2008; Davies et al., 2009; Saltzman et al., 2005) compared to non-exposed children. Prior studies in lower and middle-income countries have documented associations between IPV and poorer child health outcomes, including mortality (Ackerson & Subramanian, 2009; Rico, Fenn, Abramsky, & Watts, 2011), malnutrition (Ziaei, Naved, & Ekström, 2014), and incomplete immunizations (Sabarwal, McCormick, Silverman, & Subramanian, 2012)); however, to our knowledge, no population-based studies—in the U.S. or elsewhere—have examined maternal IPV in relation to inflammation in young children.

IPV and immune dysregulation

Basic research within psychoneuroimmunology has clarified the biological cascade that follows exposure to traumatic stressors (Danese & Lewis, 2017; Miller, Chen, & Parker, 2011). Children who experience or witness violence may experience upregulation of the HPA-axis, which can induce a cascade of elevated levels of cortisol, followed by decreased responsiveness of immune cells to glucocorticoid signaling which typically functions to down-regulate inflammatory processes, ultimately resulting in an increase in circulating markers of inflammation (Miller, Chen, & Cole, 2009; Miller et al., 2011). Prior research has documented higher mean interferon (IFN)-γ levels (Woods et al., 2005) and impaired immune control over herpes simplex virus type 1 (Garcia-Linares, Sanchez-Lorente, Coe, & Martinez, 2004) among women who reported abuse compared to non-abused women. Other research shows that women with histories of IPV have persistently elevated biomarkers of inflammation, even after leaving abusive relationships (Newton et al., 2011). Insights into biological dysregulation in children following exposure to maternal IPV may elucidate opportunities for intervention, children in the greatest need of intervention, and/or evaluation of whether treatment programs for exposed children are working (Garner et al., 2012; Shonkoff, 2012).

The present study

Drawing on data from the 2010 TDHS, we examined the association between maternal reports of IPV in the past 12 months and a common measure of inflammation—C-reactive protein (CRP), a widely used biomarker of inflammation that is prospectively associated with future cardiovascular and metabolic diseases in industrialized populations (Danesh et al., 2004; Ridker, 2003, 2007). Studies in industrialized populations show that CRP concentration tracks from childhood to adulthood (Juonala et al., 2006), and accelerates atherosclerosis progression, even in children (Jarvisalo et al., 2002). We hypothesize that the children of mothers who experienced IPV in the past 12 months will have higher levels of CRP relative to children of mothers who do not report recent IPV. By describing the associations between maternal IPV and inflammation in young children in Tanzania, we may advance knowledge about the pathways by which witnessing violence may influence risk for mental and physical disorders in children, and the contexts in which this may occur.

Methods

Sample and procedures

We used data from the 2010 TDHS, a nationally representative cross-sectional probability sample of 10,300 households focused on maternal and child health issues. The sample was selected in two-stages. First, 475 clusters were selected from the 2002 Population and Housing Census. Second, households from the selected clusters were then systematically selected to participate (N = 10,300 households in total; 22 per cluster in all regions except for Dar es Salaam where 16 households were selected). Women aged 15 to 49 who were permanent residents in the selected household or visitors in the household on the night before the survey were eligible to participate (recruited N = 10,522; 96.4% response rate). Health information was collected on children less than 5 years of age in the household (N = 7175). Full details of the study design and procedure can be found elsewhere (ICF-Macro & National Bureau of Statistics, 2011; National Bureau of Statistics (NBS) [Tanzania] & ICF-Macro, 2011). If there was privacy to ensure confidential responses, one eligible woman per household was randomly selected to complete the Domestic Violence Module (N = 7048 women, 5289 of whom were ever-married).

In the 2010 TDHS, investigators collected blood samples in order to characterize micronutrients among women and children. Level of CRP was measured for a random subsample of women and children in order to control for the potential influence of infection on levels of vitamin A. The current study is based on the 503 children of ever-married women who completed the Domestic Violence Module and also had CRP data. Blood spots were collected via finger or heel prick; skin was prepared with a 70% isopropyl alcohol swab, which air-dried and was then pricked using a disposable self-retracting lancet (Hadley & Decaro, 2014; ICF-Macro & National Bureau of Statistics, 2011). The first three drops were discarded or used for Hb testing, and the subsequent five drops were placed onto Watman 903 filter paper and then air-dried overnight. The filter paper was then stored in low-permeability zip-close bags with desiccant and humidity indicator and sent to the National Public Health Laboratory to be assayed. As described elsewhere (Hadley & Decaro, 2014), characteristics of children in the CRP subsample are similar to those of the whole sample.

Measures

C-reactive protein

CRP—an acute phase response protein—was used as a biomarker of inflammation. CRP was measured from dried blood spots (DBS) following a standardized procedure. Specifically, one 3.2 mm disc was punched from each of the DBS; the disc was placed into a micro-centrifuge tube, and then 500 μL of CRP diluted Assay Buffer Concentrate was added (Bender MedSystems GmbH, Vienna, Austria). The tubes were vortexed and centrifuged before incubating overnight at 4 °C (see details provided elsewhere (ICF-Macro & National Bureau of Statistics, 2011)). The next day, a high sensitivity commercial ELISA test kit was