2018

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

Natalie Slopen
University of Maryland at College Park

Jing Zhang
University of Maryland at College Park

Samuel S. Urlacher
CUNY Hunter College

Gretchen De Silva
University of Maryland at College Park

Mona Mittal
University of Maryland at College Park

SSM - Population Health 6 (2018) 107–115

Contents lists available at ScienceDirect

SSM - Population Health

journal homepage: www.elsevier.com/locate/ssmph

Article

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

Natalie Slopen[a,*], Jing Zhang[a], Samuel S. Urlacher[b], Gretchen De Silva[a], Mona Mittal[c]

[a] Department of Epidemiology and Biostatistics, School of Public Health, University of Maryland, College Park, MD, United States
[b] Department of Anthropology, Hunter College, City University of New York, New York, NY, United States
[c] Department of Family Sciences, School of Public Health, University of Maryland, College Park, MD, United States

ARTICLE INFO

Keywords:
Children
Intimate partner violence
C-reactive protein
Inflammation
Tanzania Demographic and Healthy Survey

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

* Correspondence to: Department of Epidemiology and Biostatistics, University of Maryland College Park, School of Public Health, (Bldg #255) 4200 Valley Drive College Park, MD 20742, United States.
E-mail address: nslopen@umd.edu (N. Slopen).

https://doi.org/10.1016/j.ssmph.2018.09.002
Received 4 July 2018; Accepted 7 September 2018

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

used to measure optical densities of CRP levels in duplicate. If coefficients of variation (CVs) between the optical densities of the duplicates exceeded 10% the samples were reanalyzed. Research on 3–5 year old children in Tanzania identified that CRP values of > 1.1 mg/L indicate acute infection status (i.e., association with overt symptoms of acute infection) for young children in this region (Wander, Brindle, & O'Connor, 2012), and prior research using the 2010 Tanzanian DHS survey (Hadley & Decaro, 2014) has used this threshold as a rough proxy to identify children with immune activation (i.e., active or recent infection). Accordingly, for children with CRP values below the 1.1 mg/L threshold, CRP is providing a measure of chronic, baseline inflammation; for children with CRP values above 1.1 mg/L, CRP is providing a measure of acute inflammatory immune response. Given the skewness of CRP levels, we used log-transformed CRP in our regression models, similar to prior studies of CRP in children (Danese et al., 2011; Slopen, Kubzansky, McLaughlin, & Koenen, 2012).

Past year maternal IPV

Our primary exposure, maternal IPV in the past year, was a binary variable based on a woman's report of having experienced any form of emotional, physical, or sexual violence perpetrated by her husband or partner in the last 12 months. The items to assess physical or sexual violence asked: *(Does/did) your (last) husband/partner ever do any of the following things to you? a) Push you, shake you, or throw something at you? b) Slap you? c) Twist your arm of pull your hair? d) Punch you with his fist or with something that could hurt you? e) Kick you, drag you or beat you up? f) Try to choke you or burn you on purpose? g) Threaten or attack you with a knife, gun, or any other weapon? h) Physically force you to have sexual intercourse with him even when you did not want to?* and *i) Force you to perform any sexual acts you did not want to?* The items to assess emotion violence asked: *(Does/did) your (last) husband/partner ever: a) Say or do something to humiliate you in front of others? b) Threaten to hurt or harm you or someone close to you?* and *c) Insult you or make you feel bad about yourself?* If the woman replied "yes" to any of these items, she was asked to report the frequency in the past 12 months: *often, only sometimes, or not at all*. We constructed binary outcomes for each type of violence by combining reports of "often" and "sometimes", and a binary outcome for any violence by combining across types of violence.

Covariates

Informed by prior literature (Hadley & Decaro, 2014; O'Connor et al., 2009; Wilunda, Massawe, & Jackson, 2013), we included a number of child, maternal, and household characteristics as covariates and potential confounders. Child characteristics included age, sex, and maternal reports of whether the child is currently breastfeeding and recent illnesses in the past two weeks. Child body mass index was computed based on height and weight, as measured by trained interviewers. Height was assessed using a measuring board by Shorr Productions. Children younger than 24 months were measured while lying down, and children older than 2 years were measured while standing. Weight was measured using SECA scales. Children's weight for height was converted to a z-score based on World Health Organization (WHO) growth standards (de Onis, Onyango, Borghi, Garza, & Yang, 2006). Maternal and household characteristics were collected in the Mother and Household Questionnaires, including highest maternal education (4-category variable: no education, primary incomplete, primary complete, and secondary or more), household size (number of residents, operationalized as a 4-category variable), location (urban vs. rural), family wealth (5-category variable; a standard variable calculated for most DHS surveys using principal component analysis, based on information about household assets and consumer goods collected in the survey) (Rutstein, 2008), maternal marital status (current vs. divorced/separated/widowed), and maternal illiteracy (i.e., unable to read various sentences in Swahili).

Analyses

First, we calculated means and proportions for all variables for the full sample of children. Second, we calculated these statistics stratified by: 1) whether the child's mother reported exposure to IPV in the past year; and 2) the child's immune activation status, i.e., whether or not the child's CRP value was above 1.1 mg/L, the threshold identified in prior research on 3–5 year old children in Tanzania to indicate active/recent infection (Wander et al., 2012). Third, we reported the median and inter-quartile ranges of CRP for children exposed and unexposed to any type of maternal IPV, and then repeated this analysis stratified by acute infection status. Finally, we computed a series of linear regression models stratified based on acute infection status, using model building to observe changes as potential confounders were added to the model. In Model 1, we adjusted for child age and sex. In Model 2, we additionally adjusted for child BMI z-score and breastfeeding status. In Model 3, we additionally adjusted for maternal and household characteristics (as described in the Measures section and presented in Table 3). In sensitivity analyses, we replicated our descriptive analyses and linear regression models for each type of maternal IPV separately (i.e., physical, sexual, and emotional). A two-sided test for statistical significance was used, and analyses were conducted in R statistical software using commands to account for the complex survey design, including clustering of observations within households and primary sampling units.

Results

Descriptive statistics

In the total sample of 503 children, 39.6% had mothers who reported any IPV in the past 12 months; physical (32.4%) and emotional (31.2%) violence were more common than sexual violence (11.9%). The average child age was 32 months, and just over half (51.7%) of the children were female (see Table 1). The highest educational attainment for the majority of mothers is primary school (63.6%), one-third of mothers were unable to read in Swahili, and 22% of children resided in an urban location. Compared to children whose mothers did not experience IPV in the past 12 months, children of mothers who reported IPV had lower BMI z-scores (1.67 vs. 3.66) and were more likely to have had a common childhood illnesses in the past 2 weeks (i.e., diarrhea, cough, fever). Just over half of children in the sample (52%, n = 262) had a CRP value below 1.1 mg/L, meaning that 48 percent of children's CRP indicated active or recent infection. Considering child and household characteristics by child immune activation status, relative to the healthy group, children with active/recent infection were younger, more likely to have maternal reports of common illnesses in the past 2 weeks (46.9% vs. 38.9%, respectively) and a mother with no formal education (32.4% vs. 18.3%, respectively), and were less likely to live in urban area (19.5% vs. 24.4%).

Any maternal IPV in the past 12 months and child CRP

In the full sample, we do not observe differences in children's median CRP values by any maternal IPV in the past 12 months. The median CRP value for children of mothers exposed to any IPV in the past 12 months was 0.84 mg/L (interquartile range (IQR) = 2.74), compared to 1.17 mg/L (IQR = 3.31) for children of mothers not exposed (p = 0.87). In the healthy subset of the sample, there is trend towards a difference in median CRP values by any maternal IPV in the past 12 months (see Fig. 1A), with a higher value for children of mothers reporting IPV in the past 12 months (median = 0.41 mg/L) relative to children whose mothers did not (median = 0.35 mg/L); p-value = 0.13. Considering median CRP values in the subset of children

Table 1
Demographics characteristics of sample and by maternal exposure to IPV in past 12 months and elevated CRP status, Tanzania Demographic and Health Survey, 2010.

	Full sample	Any maternal IPV, past 12 m		CRP value > 1.1 mg/l	
	(n = 503)	Yes (n = 199)	No (n = 304)	No ("healthy") (n = 262)	Yes ("active/recent infection") (n = 241)
Child Characteristics					
Age, in months, mean (SE)	32.74 (0.68)	33.32 (1.03)	32.37 (0.88)	34.60 (0.92)	30.73 (1.04)
BMI z-score, mean (SE)	2.87 (0.72)	1.67 (0.87)	3.66 (1.07)	3.08 (1.07)	2.65 (0.99)
Sex, % female	51.69	54.27	50.00	56.11	46.89
Any illness in past 2 weeks, %	42.74	46.73	40.13	38.93	46.89
Diarrhea past 2 weeks, %	20.48	24.12	18.09	16.41	24.90
Cough, past 2 weeks, %	26.44	30.15	24.01	23.66	29.46
Fever, past 2 weeks, %	27.63	30.65	25.66	24.81	30.71
Currently breastfeeding, %	53.88	52.76	54.61	49.62	58.51
C-reactive protein, mg/l, Median (IQR)	1.01 (2.94)	0.84 (3.31)	1.17 (2.74)	0.35 (0.48)	3.55 (3.76)
Maternal and Household Characteristics					
Maternal Education, %					
No education	25.05	22.11	26.97	18.32	32.37
Primary	63.62	72.86	57.57	67.56	59.34
Secondary or higher	11.33	5.03	15.46	14.12	8.30
Maternal illiteracy[a], %	33.00	31.16	34.21	25.95	40.66
Wealth Quintile, %					
Poorest	25.44	24.62	25.99	26.72	24.07
Poorer	17.69	18.09	17.43	16.41	19.09
Middle	20.08	25.13	16.78	17.94	22.41
Richer	20.68	20.60	20.72	21.37	19.92
Richest	16.10	11.56	19.08	17.56	14.52
Currently married/cohabiting, %	89.07	85.93	91.11	88.17	90.04
Urban residential location, %	22.07	20.60	23.03	24.43	19.50
# of usual household members, %					
[Q1]	28.23	27.14	28.95	26.72	29.88
[Q2]	35.39	25.13	34.54	36.26	21.58
[Q3]	11.53	22.61	11.84	13.36	34.85
[Q3]	24.85	25.13	24.67	23.66	13.69

[a] Unable to read various sentences in Swahili. IPV = intimate partner violence; CRP = C-reactive protein; BMI = body mass index.

with immune activation, children of mothers who reported any IPV in the past 12 months had a higher median CRP relative to children whose mothers who did not experience IPV in the past 12 months (4.1 vs. 3.1 mg/L; see Fig. 1B; $p = 0.03$).

Using linear regression models, we examined the association between maternal IPV in the past 12 months and (log) child CRP (see Table 2). In the healthy group, maternal report of any IPV was associated with higher offspring (log) CRP in a model adjusted for child age and sex (Model 1a: β = 0.26, 95% confidence interval (CI) = 0.02, 0.49). The association between maternal IPV and child (log) CRP was similar after child BMI z-score and breast feeding were added to the model (Model 2a: β = 0.27, 95% CI = 0.03, 0.50), and weakened only slightly when other maternal and household characteristics were added to the model (Model 3a: β = 0.24, 95% CI = -0.02, 0.50). Among children with active/recent infection, a similar pattern was detected, whereby maternal IPV in the past 12 months was associated with offspring (log) CRP across all three models (Model 1b: β = 0.18, 95% CI = 0.02, 0.35; Model 2b: β = 0.17, 95% CI = 0.01, 0.34; Model 3b: β = 0.21, 95% CI = 0.04, 0.37; all p-values < 0.05). There were some inconsistent associations between other covariates and CRP; for example, females had higher (log) CRP in the healthy group, but not in the group with active/recent infection, marital/cohabitation status was associated with lower (log) CRP in the group with active/recent infection, but not in the healthy children.

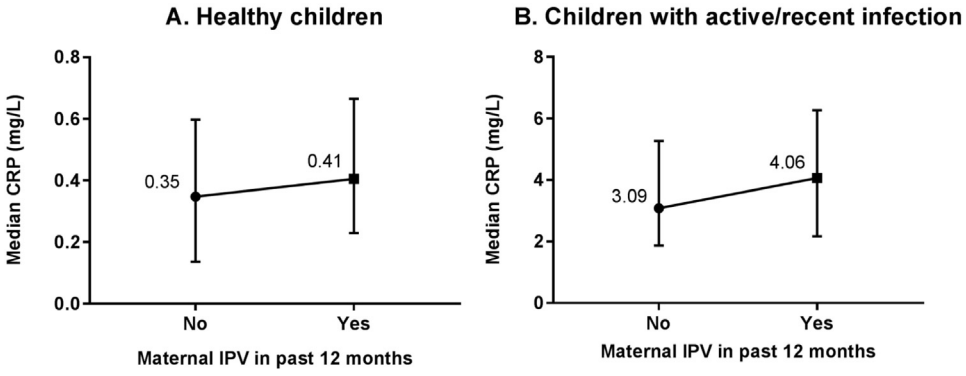

Fig. 1. Median C-reactive protein values (and interquartile ranges) by exposure to maternal IPV in the past 12 months for (A) healthy children (defined as CRP < 1.1 mg/L; n = 262) and (B) children with active/recent infection (defined as CRP > 1.1 mg/L; n = 241). *P*-values, obtained from Mann-Whitney *U* Test, are 0.125 and 0.03 for healthy children, and children with an active/recent infection respectively. Any maternal IPV in past 12 months reflects positive endorsement of physical, sexual, or emotional abuse. CRP = C-reactive protein; IPV = intimate partner violence.

Table 2
Adjusted regression models of associations between maternal exposure to any IPV in past 12 months and child's (log) CRP: Tanzania Demographic and Health Survey, 2010.

	Healthy children (n = 262)			Children with active/recent infection (CRP > 1.1 mg/l) (n = 241)		
	Model 1a β (95% CI)	Model 2a β (95% CI)	Model 3a β (95% CI)	Model 1b β (95% CI)	Model 2b β (95% CI)	Model 3b β (95% CI)
Any maternal IPV	0.26 (0.021, 0.49)*	0.27 (0.03, 0.50)*	0.24 (-0.02, 0.50)~	0.18 (0.02, 0.35)*	0.17 (0.01, 0.34)*	0.21 (0.04, 0.37)*
Child Characteristics						
Age (months)	-0.01 (-0.01, 0.00)	-0.01(-0.01, 0.00)	-0.01(-0.02, 0.00)	-0.001 (-0.01, 0.00)	-0.001 (-0.01, 0.00)	-0.001 (-0.01, 0.00)
Sex (female)	0.21 (-0.02, 0.44)~	0.21 (-0.03, 0.45)~	0.25 (0.02, 0.49)*	0.05 (-0.20, 0.11)	-0.04 (-0.20, 0.11)	-0.04 (-0.20, 0.12)
BMI z-score		0.002 (-0.01, 0.01)	0.003 (-0.01, 0.01)		0.003 (-0.002, 0.01)	0.004 (-0.00, 0.01)
Currently breastfeeding (vs. not)		-0.03 (-0.28, 0.21)	-0.05 (-0.31, 0.21)		-0.05 (0.22, 0.11)	-0.07 (-.24, 0.10)
Maternal and Household Characteristics						
Maternal Education						
No education			–			–
Primary			-0.45 (-1.04, 0.14)			-0.04 (-0.33, 0.25)
Secondary or higher			-0.76 (-1.45, -0.07)*			0.03 (-0.39, 0.44)
Maternal illiteracy (vs. literate)			-0.11 (-0.67, 0.45)			-0.14 (-0.38, 0.11)
Wealth Quintile			–			–
Poorest			0.27 (-0.15, 0.69)			-0.19 (-0.46, 0.08)
Poorer			0.17 (-0.26, 0.61)			-0.22 (-0.46, 0.03)~
Middle			0.23 (-0.25, 0.71)			-0.02 (-0.27, 0.22)
Richer			0.56 (-0.10, 1.22)			-0.11 (-0.47, 0.26)
Richest						
Currently married/cohabiting			-0.06 (-0.43, 0.32)			-0.27 (-0.51, -0.04)*
Urban residence (vs. rural)			0.32 (-0.18, 0.81)			-0.02 (-0.25, 0.22)
# of usual household members						
[Q1]			–			–
[Q2]			0.20 (-0.14, 0.53)			0.24 (0.04, 0.43)*
[Q3]			-0.05 (-0.50, 0.41)			0.08 (-0.20, 0.37)
[Q3]			0.20 (-0.14, 0.54)			0.13 (-0.10, 0.35)

Notes: CRP = C-reactive protein; IPV = intimate partner violence; BMI = body mass index; CI = confidence interval.
***p < 0.0001.
**p < 0.01.
* p < 0.05.
~ p < 0.10.

Sensitivity analysis: Subtypes of maternal IPV and child CRP

In the sample overall, maternal experiences of physical (32.4%) and emotional (31.2%) violence were more common than sexual violence (11.9%), and each type of violence was slightly more common among the healthy sample relative to children with immune activation (See Appendix A). In the full sample, similar to the comparisons for any type of IPV, we do not observe differences in children's median CRP values based on the separate types of IPV. When each type of IPV is considered individually within the healthy group, we do not see evidence for differences in children's median CRP values for any type of IPV. Among children with active or recent infection, the median CRP is higher among children whose mothers reported physical ($p = 0.05$), sexual ($p = 0.02$), and emotional violence ($p = 0.06$) in the past 12 months, relative to children whose mothers did not report these experiences. In linear regression models of healthy children (See Appendix B), only maternal physical IPV in the past 12 months was associated with children's (log) CRP (Model 3: $\beta = 0.24$, 95% CI = 0.01, 0.49). Among children with active or recent infection, children whose mothers reported each type of IPV had elevated (log) CRP relative to children whose mothers who were unexposed (i.e., betas for fully adjusted models: physical IPV, $\beta = 0.22$, 95% CI = 0.05, 0.40; sexual IPV, $\beta = 0.37$, 95% CI = 0.13, 0.60; and emotional IPV, $\beta = 0.19$, 95% CI = 0.02, 0.36).

Discussion

In this sample of children ages 6 months to 5 years in Tanzania, our findings support the hypothesis that a mother's IPV experience in the past 12 months is positively associated with her child's CRP level, controlling for child and household characteristics. Importantly, this association was evident among children with CRP values in the "healthy" range (i.e., < 1.1 mg/L), and also among children showing immune activation due to active or recent infection (i.e., CRP > 1.1 mg/L). Among the healthy subset of children, the association was slightly attenuated after adjustment for relevant covariates, whereas the association among children with active/recent infection became larger once we added child health and maternal and household characteristics to the model. These findings make a unique contribution to our underdeveloped understanding of the biological embedding of childhood adversity in non-Western contexts (McDade, 2002; Worthman & Costello, 2009; Worthman & Panter-Brick, 2008), and suggest that a threat-based exposure like IPV may influence inflammatory processes in young children within an ecologic setting that is very different from the material, nutritional, social, and pathogenic context where most prior research on childhood stress and development has occurred. This study has identified yet another intergenerational consequence of violence against women, and may be relevant to practitioners interested in designing health promotion and prevention strategies based on high-prevalence risk factors. Our study also highlights the value of biomarkers to assess the intergenerational burden of IPV, as biomarkers can avoid recall or reporting biases that are common for parental report of child symptoms, yet can detect subclinical immune system activity that could be relevant to child development and health (McDade et al., 2007; Worthman & Costello, 2009).

While it is common to exclude individuals with CRP above a specified certain threshold from research on CRP as a chronic disease risk biomarker (typically, > 10 mg/L in adults), evidence from young adults in the U.S. found that common indicators of disease risk were associated with very high CRP (i.e. > 10 mg/L) (Shanahan, Freeman, & Bauldry, 2014). Rather than exclude all children showing immune activation from our study, we examined the association of interest in children with and without active or recent infection. From a physiological perspective, these results suggest that the decreased ability of children exposed to maternal IPV to down-regulate inflammation may impact both chronic CRP production (those with CRP < 1.1 mg/L) and the acute inflammatory response to infection (those with CRP > 1.1 mg/L). Although chronic CRP levels are most commonly studied in population-based health research, the magnitude of CRP elevation in response to infection may also be informative for understanding dysregulation of inflammation. There is a need for additional research to determine whether this patterning among children with acute or recent infection can be replicated and generalized to other samples.

Our findings align with studies using European samples that have documented that childhood adversity is associated with elevated CRP levels in late childhood (Danese et al., 2011; Howe et al., 2010; Slopen et al., 2012) and adolescence (Fuligni et al., 2009; Murasko, 2008), and advance knowledge about the biological embedding of childhood adversity in three important ways. First, most previous research on the association between childhood adversity and inflammation in children or adolescents has taken place in Western contexts, with some notable exceptions (Decaro, Manyama, & Wilson, 2016; McDade et al., 2005), including a study of 88 mother-infant dyads in Mwanza, Tanzania which found that high maternal depressive symptoms and severe food insecurity were each associated with "high" CRP (i.e., above the sample median) (Decaro et al., 2016). Our study finds that maternal IPV—a form of childhood psychosocial adversity—is evident among children in Tanzania, thus providing additional evidence that this process of biological embedding may generalize to non-Western contexts. Second, the majority of existing studies on the association between childhood adversity and chronic low-grade inflammation have included older childhood or adolescents (Slopen, Kubzansky, & Koenen, 2011), and studies that have included children under the age of 5 years are rare (Decaro et al., 2016; Dowd, Zajacova, & Aiello, 2010; Hadley & Decaro, 2014; McDade et al., 2005). Our study sample, comprised of children 6 months to 5 years, provides insights on the outstanding question of when the effect of childhood adversity on inflammation may emerge (Danese et al., 2011). This question of timing of emergence is critically important, as it has implications for understanding, and intervening on, the early life origins of disease risk across the life course (Shonkoff et al., 2009). Third, to our knowledge, this is the first study to report on maternal IPV (as a form of childhood adversity) in relation to inflammation or other biomarkers of immune activation, in either a Western or non-Western context. In future research, it will be important to examine whether maternal IPV is an independent risk factor for elevated inflammation in children due to the psychological trauma of witnessing a mother being abused, or if child maltreatment or maternal depression may be implicated in this association. The 2010 TDHS did not include maternal report of child maltreatment or depression; therefore, we cannot explore any potential indirect or synergistic effects in this study.

There are several other limitations to the present study that are important to acknowledge. First, this is an observational cross-sectional study; therefore, information on maternal IPV in the past 12 months and child CRP were collected at the same time and we cannot infer direct causal relationships. Of note, consistent with this study, experimental research in adults has documented elevations in circulating inflammatory biomarkers in response to acute psychological stress (Steptoe, Hamer, & Chida, 2007). Prospective studies are needed to document associations over time and across development. Second, nearly half of the children had CRP levels that indicated active or recent infection, which was expected (Hadley & Decaro, 2014); because it was important to conduct our analyses stratified by immune activation status, this resulted in relatively small sample sizes of healthy children and children with active/recent infection. Related, the threshold to identify healthy versus unhealthy children is only a rough proxy, and there are likely false positives and false negatives. Third, mothers were not administered the Domestic Violence Module if it was not possible to

ensure privacy during the interview, which reduces the generalizability of our sample. Fourth, individual, household, or community factors could influence the accuracy of reported IPV; although we adjusted for a range of covariates, it is possible that we omitted other relevant factors that could influence reporting. Fifth, we do not have information severity of infection, which could be explain some of the variation in CRP levels among those categorized as having an active or recent infection. Finally, only one biomarker of inflammation, CRP, was available; in the future, it would be ideal to address this question using multiple biomarkers of inflammation, including upstream pro-inflammatory cytokines.

In summary, this study identified an association between maternal IPV in the past 12 months and CRP levels in a population-based sample of young children in Tanzania, in both healthy children, and in children with an active or recent infection. Building on prior studies of maternal IPV and other forms of physiological dysregulation in children and adolescents, this finding is important given that violence against women is a pervasive public health crisis around the world (World Health Organization, 2013), and that inflammation is implicated in the development and worsening of multiple health conditions (Danese & Baldwin, 2017; Howren, Lamkin, & Suls, 2009; Jarvisalo et al., 2002; Nusslock & Miller, 2015). Although prospective research is needed, our results provide support for the hypothesis that inflammation may function as a biological pathway linking maternal IPV (or childhood adversity more broadly) to the poor psychological and physical health outcomes observed for the children of women who are victimized—and this may extend to very young children and children in non-Western contexts.

Acknowledgments

We thank all the respondents for participating in collection of this data.

Appendix A

See Table A1.

Table A1
Percentage of children with mothers who reported physical, sexual, and emotional violence in the past 12 months, and median CRP (and interquartile ranges) stratified by probable infection status; Tanzania Demographic and Health Survey, 2010.

	Healthy Sample (CRP < 1.1 mg/l) (n = 262)			Active/recent infection (CRP > 1.1 mg/l) (n = 241)		
	%	Median (IQR)	*p*-value	%	Median (IQR)	*p*-value
Physical abuse			0.15			0.05
Yes	34.35	0.36 (0.41)		30.29	4.05 (4.06)	
No	65.65	0.35 (0.45)		69.71	3.12 (3.55)	
Sexual abuse			0.29			0.02
Yes	13.36	0.30 (0.41)		10.37	4.89 (3.95)	
No	86.64	0.38 (0.48)		89.63	3.26 (3.56)	
Emotional abuse			0.48			0.06
Yes	33.59	0.34 (0.42)		28.63	4.05 (4.16)	
No	66.41	0.35 (0.48)		71.37	3.17 (3.48)	

Notes: All abuses happened in the past 12 months; p-values are obtained from Mann-Whitney *U* Test. CRP = C-reactive protein; IPV = intimate partner violence; IQR = interquartile range

Appendix B

See Table B1.

Financial disclosure

None.

Funding source

This secondary data analysis was supported by a grant to NS, JZ, and MM from the University of Maryland ADVANCE program.

Conflict of interest

The authors have no conflicts of interest to disclose.

Ethics review for secondary data analysis

The first author, Natalie Slopen, submitted an IRB application to the University of Maryland College Park (UMCP) IRB. The UMCP IRB conducted an administrative review of our project ("[936462-1] Maternal intimate partner violence victimization and immune activation in women and children in Tanzania"). The IRB declared that the project was "not human subjects research" given that we exclusively relied on de-identified data.

Ethics review for the DHS survey

Procedures and questionnaires for standard DHS surveys have been reviewed and approved by ICF Institutional Review Board (IRB). Additionally, country-specific DHS survey protocols are reviewed by the ICF IRB and typically by an IRB in the host country. ICF IRB ensures that the survey complies with the U.S. Department of Health and Human Services regulations for the protection of human subjects (45 CFR 46), while the host country IRB ensures that the survey complies with laws and norms of the nation.

Table B1
Adjusted regression models of associations between maternal exposure to physical, sexual, or emotional IPV in past 12 months and child's (log) CRP: Tanzania Demographic and Health Survey, 2010.

	Healthy children (n = 262)			Children with active/recent infection (CRP > 1.1 mg/l) (n = 241)		
	Model 1a β (95% CI)	Model 2a β (95% CI)	Model 3a β (95% CI)	Model 1b β (95% CI)	Model 2b β (95% CI)	Model 3b β (95% CI)
Physical violence						
Yes	0.25 (0.02, 0.49)*	0.26 (0.03, 0.50)*	0.24 (-0.01, 0.49)~	0.17 (-0.003, 0.35)~	0.17 (-0.01, 0.35)~	0.22 (0.05, 0.40)*
No	–	–	–	–	–	–
Sexual violence						
Yes	-0.15 (-0.49, 0.18)	-0.15 (-0.49, 0.18)	-0.21 (-0.53, 0.09)	0.32 (0.05, 0.58)*	0.30 (0.04, 0.57)*	0.37 (0.13, 0.60)**
No	–	–	–	–	–	–
Emotional violence						
Yes	0.15 (-0.08, 0.39)	0.16 (-0.07, 0.40)	0.13 (-0.13, 0.40)	0.17 (-0.01, 0.34)~	0.16 (-0.01, 0.33)~	0.19 (0.02, 0.36)*
No	–	–	–	–	–	–

***p < 0.0001.

Notes: Model 1 is adjusted for child's age and sex; Model 2 is additionally adjusted for child's BMI z-score and breastfeeding status; Model 3 is additionally adjusted for highest maternal education (no education, primary incomplete, primary complete, and secondary or more), household size (number of residents, quartiles), location (urban vs. rural), family wealth (quintiles), maternal marital status (current vs. divorced/separated/widowed), and maternal illiteracy (i.e., unable to read various sentences in Swahili).

CRP = C-reactive protein; IPV = intimate partner violence; BMI = body mass index

** p < 0.01.

* p < 0.05.

~ p < 0.10.

References

Ackerson, L. K., & Subramanian, S. V. (2009). Intimate partner violence and death among infants and children in India. *Pediatrics, 124*, e878–e889.

Black, P. H., & Garbutt, L. D. (2002). Stress, inflammation and cardiovascular disease. *Journal of Psychosomatic Research, 52*, 1–23.

Coker, A. L., Smith, P. H., Bethea, L., King, M. R., & McKeown, R. E. (2000). Physical health consequences of physical and psychological intimate partner violence. *Archives of Family Medicine, 9*, 451–457.

Danese, A., & Baldwin, J. R. (2017). Hidden wounds? Inflammatory links between childhood trauma and psychopathology. *Annual Review of Psychology, 68*, 517–544.

Danese, A., Caspi, A., Williams, B., Ambler, A., Sugden, K., Mika, J., et al. (2011). Biological embedding of stress through inflammation processes in childhood. *Molecular Psychiatry, 16*, 244–246.

Danese, A., & Lewis, S. J. (2017). Psychoneuroimmunology of early-life stress: The hidden wounds of childhood trauma? *Neuropsychopharmacology, 42*, 99–114.

Danesh, J., Wheeler, J. G., Hirschfield, G. M., Eda, S., Eiriksdottir, G., Rumley, A., et al. (2004). C-reactive protein and other circulating markers of inflammation in the prediction of coronary heart disease. *New England Journal of Medicine, 350*, 1387–1397.

Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: When the immune system subjugates the brain. *Nature Reviews Neuroscience, 9*, 46–56.

Davies, P. T., Sturge-Apple, M. L., Cicchetti, D., & Cummings, E. M. (2008). Adrenocortical underpinnings of children's psychological reactivity to interparental conflict. *Child Development, 79*, 1693–1706.

Davies, P. T., Sturge-Apple, M. L., Cicchetti, D., Manning, L. G., & Zale, E. (2009). Children's patterns of emotional reactivity to conflict as explanatory mechanisms in links between interpartner aggression and child physiological functioning. *Journal of Child Psychology and Psychiatry, 50*, 1384–1391.

Davies, P. T., Winter, M. A., & Cicchetti, D. (2006). The implications of emotional security theory for understanding and treating childhood psychopathology. *Development and Psychopathology, 18*, 707–735.

de Onis, M., Onyango, A. W., Borghi, E., Garza, C., & Yang, H. (2006). WHO Multicentre Growth Reference Study Group: Comparison of the World Health Organization (WHO) Child Growth Standards and the National Center for Health Statistics/WHO international growth reference: Implications for child health programmes. *Public Health Nutrition, 9*, 942–947.

Decaro, J. A., Manyama, M., & Wilson, W. (2016). Household-level predictors of maternal mental health and systemic inflammation among infants in Mwanza, Tanzania. *American Journal of Human Biology, 28*, 461–470.

Dowd, J. B., Zajacova, A., & Aiello, A. E. (2010). Predictors of inflammation in U.S. children aged 3-16 years. *American Journal of Preventive Medicine, 39*, 314–320.

Fuligni, A. J., Telzer, E. H., Bower, J., Cole, S. W., Kiang, L., & Irwin, M. R. (2009). A Preliminary study of daily interpersonal stress and c-reactive protein levels among adolescents from latin american and european backgrounds. *Psychosomatic Medicine, 71*, 329–333.

Garcia-Linares, M. I., Sanchez-Lorente, S., Coe, C. L., & Martinez, M. (2004). Intimate male partner violence impairs immune control over herpes simplex virus type 1 in physically and psychologically abused women. *Psychosomatic Medicine, 66*, 965–972.

Garner, A. S., Shonkoff, J. P., Siegel, B. S., Dobbins, M. I., Earls, M. F., McGuinn, L., et al. (2012). Early childhood adversity, toxic stress, and the role of the pediatrician: Translating developmental science into lifelong health. *Pediatrics, 129*, E224–E231.

Gottman, J. M., & Katz, L. F. (1989). Effects of marital discord on young children's peer interaction and health. *Developmental Psychology, 25*, 373–381.

Hadley, C., & Decaro, J. A. (2014). Testing hypothesized predictors of immune activation in tanzanian infants and children: Community, household, caretaker, and child effects. *American Journal of Human Biology, 26*, 523–529.

Howe, L. D., Galobardes, B., Sattar, N., Hingorani, A. D., Deanfield, J., Ness, A. R., et al. (2010). Are there socioeconomic inequalities in cardiovascular risk factors in childhood, and are they mediated by adiposity? Findings from a prospective cohort study. *International Journal of Obesity, 34*, 1149–1159.

Howren, M. B., Lamkin, D. M., & Suls, J. (2009). Associations of depression with C-reactive protein, IL-1, and IL-6: A meta-analysis. *Psychosomatic Medicine, 71*, 171–186.

ICF-Macro, & National Bureau of Statistics (2011). *Micronutrients: Results of the 2010 Tanzania demographic and health survey.* Dar es Salaam: NBS and ICF Macro.

Jarvisalo, M. J., Harmoinen, A., Hakanen, M., Paakunainen, U., Viikari, J., Hartiala, J., et al. (2002). Elevated serum C-reactive protein levels and early arterial changes in healthy children. *Arteriosclerosis Thrombosis and Vascular Biology, 22*, 1323–1328.

Juonala, M., Viikari, J. S. A., Ronnemaa, T., Taittonen, L., Marniemi, J., & Raitakari, O. T. (2006). Childhood C-reactive protein in predicting CRP and carotid intima-media thickness in adulthood - The Cardiovascular Risk in Young Finns Study. *Arteriosclerosis Thrombosis and Vascular Biology, 26*, 1883–1888.

Kramer, A., Lorenzon, D., & Mueller, G. (2004). Prevalence of intimate partner violence and health implications for women using emergency departments and primary care clinics. *Women'States Health, 14*, 19–29.

McDade, T. W. (2002). Status incongruity in Samoan youth: A biocultural analysis of culture change, stress, and immune function. *Medical Anthropology Quarterly, 16*, 123–150.

McDade, T. W., Leonard, W. R., Burhop, J., Reyes-Garcia, V., Vadez, V., Huanca, T., et al. (2005). Predictors of C-reactive protein in Tsimane' 2 to 15 year-olds in lowland Bolivia. *American Journal of Physical Anthropology, 128*, 906–913.

McDade, T.W., Reyes-García, V., Blackinton, P., Tanner, S., Huanca, T., & Leonard, W.R. (2007). Ethnobotanical knowledge is associated with indices of child health in the Bolivian Amazon. Proceedings of the National Academy of Sciences, 10, 4, 6134, –6139.

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual Review of Psychology, 60*, 501–524.

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin, 137*, 959–997.

Murasko, J. E. (2008). Male-female differences in the association between socioeconomic status and atherosclerotic risk in adolescents. *Social Science & Medicine, 67*, 1889–1897.

National Bureau of Statistics (NBS) [Tanzania], & ICF Macro (2011). *Tanzania Demographic and Health Survey 2010.* Dar es Salaam, Tanzania: National Bureau of Statistics, and ORC Macro.

Newton, T. L., Fernandez-Botran, R., Miller, J. J., Lorenz, D. J., Burns, V. E., & Fleming, K. N. (2011). Markers of inflammation in midlife women with intimate partner violence

histories. *Journal of Women's Health, 20*, 1871–1880.
Nusslock, R., & Miller, G. E. (2015). Early-life adversity and physical and emotional health across the lifespan: A neuro-immune network hypothesis. *Biological Psychiatry*.
O'Connor, M. F., Bower, J. E., Cho, H. J., Creswell, J. D., Dimitrov, S., Hamby, M. E., et al. (2009). To assess, to control, to exclude: Effects of biobehavioral factors on circulating inflammatory markers. *Brain Behavior and Immunity, 23*, 887–897.
Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002a). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin, 128*, 330–366.
Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002b). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin, 128*, 330–366.
Rico, E., Fenn, B., Abramsky, T., & Watts, C. (2011). Associations between maternal experiences of intimate partner violence and child nutrition and mortality: Findings from Demographic and Health Surveys in Egypt, Honduras, Kenya, Malawi and Rwanda. *Journal of Epidemiology and Community Health, 65*, 360–367.
Ridker, P. M. (2003). C-reactive protein: A simple test to help predict risk of heart attack and stroke. *Circulation, 108*, e81–e85.
Ridker, P. M. (2007). C-reactive protein and the prediction of cardiovascular events among those at intermediate risk: Moving an inflammatory hypothesis toward consensus. *Journal of the American College of Cardiology, 49*, 2129–2138.
Rigterink, T., Fainsilber Katz, L., & Hessler, D. M. (2010). Domestic violence and longitudinal associations with children's physiological regulation abilities. *Journal of Interpersonal Violence, 25*, 1669–1683.
Rutstein, S. O. (2008). *The DHS Wealth Index: Approaches for rural and urban areas*.
Sabarwal, S., McCormick, M. C., Silverman, J. G., & Subramanian, S. V. (2012). Association between maternal intimate partner violence victimization and childhood immunization in India. *Journal of Tropical Pediatrics, 58*, 107–113.
Saltzman, K. M., Holden, G. W., & Holahan, C. J. (2005). The psychobiology of children exposed to marital violence. *Journal of Clinical Child and Adolescent Psychology, 34*, 129–139.
Shanahan, L., Freeman, J., & Bauldry, S. (2014). Is very high C-reactive protein in young adults associated with indicators of chronic disease risk? *Psychoneuroendocrinology, 40*, 76–85.
Shonkoff, J. P. (2012). Leveraging the biology of adversity to address the roots of disparities in health and development. *Proceedings of the National Academy of Sciences of the United States of America, 109*, 17302–17307.
Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, molecular biology, and the childhood roots of health disparities: Building a new framework for health promotion and disease prevention. *Journal of the American Medical Association, 301*, 2252–2259.
Shonkoff, J. P., & Garner, A. S. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics, 129*, E232–E246.
Slopen, N., Kubzansky, L. D., & Koenen, K. C. (2011). Childhood adversity and inflammatory and immune biomarkers associated with cardiovascular risk in youth: A systematic review. *Brain, Behavior, and Immunity, 26*, 239–250.
Slopen, N., Kubzansky, L. D., McLaughlin, K. A., & Koenen, K. C. (2012). Childhood adversity and inflammatory processes in youth: A prospective study. *Psychoneuroendocrinology*.
Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: A review and meta-analysis. *Brain Behavior and Immunity, 21*, 901–912.
Wander, K., Brindle, E., & O'Connor K, A. (2012). Sensitivity and specificity of C-reactive protein and alpha(1) -acid glycoprotein for episodes of acute infection among children in Kilimanjaro, Tanzania. *American Journal of Human Biology, 24*, 565–568.
Wilunda, C., Massawe, S., & Jackson, C. (2013). Determinants of moderate-to-severe anaemia among women of reproductive age in Tanzania: Analysis of data from the 2010 Tanzania demographic and health survey. *Tropical Medicine International Health, 18*, 1488–1497.
Wood, S. L., & Sommers, M. S. (2011). Consequences of intimate partner violence on child witnesses: A systematic review of the literature. *Journal of Child and Adolescent Psychiatric Nursing, 24*, 223–236.
Woods, A., Page, G., O'Campo, P., Pugh, L., Ford, D., & Campbell, J. (2005). The mediation effect of posttraumatic stress disorder symptoms on the relationship of intimate partner violence and IFN-γ levels. *American Journal of Community Psychology, 36*, 159–175.
World Health Organization (2013). *Global and regional estimates of violence against women: Prevalence and health effects of intimate partner violence and non-partner sexual violence.* Geneva: World Health Organization.
World Health Organization/London School of Hygiene and Tropical Medicine (2010). *Preventing intimate partner and sexual violence against women: Taking action and generating evidence.* Geneva: World Health Organization.
Worthman, C. M., & Costello, E. J. (2009). Tracking biocultural pathways in population health: The value of biomarkers. *Annals of Human Biology, 36*, 281–297.
Worthman, C. M., & Panter-Brick, C. (2008). Homeless street children in Nepal: Use of allostatic load to assess the burden of childhood adversity. *Development and Psychopathology, 20*, 233–255.
Yount, K. M., DiGirolamo, A. M., & Ramakrishnan, U. (2011). Impacts of domestic violence on child growth and nutrition: A conceptual review of the pathways of influence. *Social Science & Medicine, 72*, 1534–1554.
Ziaei, S., Naved, R. T., & Ekström, E.-C. (2014). Women's exposure to intimate partner violence and child malnutrition: Findings from demographic and health surveys in Bangladesh. *Maternal Child Nutrition, 10*, 347–359.