Associations of postpartum sleep, stress, and depressive symptoms with LPS-stimulated cytokine production among African American and White women

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A R T I C L E   I N F O

A B S T R A C T

Background: Postpartum is a period of unique psychosocial stress characterized by sleep disturbance, risk for depressed mood, and heightened parenting stress. However, data on effects of these exposures on inflammatory immune function are limited.

Methods: This study examined associations among sleep, psychosocial stress (i.e., parenting stress, general perceived stress), mood (i.e., depressive symptoms), serum cytokine levels, and LPS-stimulated proinflammatory cytokine production among 69 women (32 African American, 37 White) assessed at 7–10 weeks postpartum.

Results: No associations between behavioral measures and serum cytokine levels were observed among women of either race. In African American women, but not Whites, poorer sleep quality, greater parenting stress, and greater depressive symptoms were associated with greater LPS-stimulated IL-6 and IL-8 production (ps ≤ 0.05). Also in African Americans, greater general perceived stress was associated with greater IL-8 production, and greater depressive symptoms with greater stimulated TNF-α production (ps ≤ 0.05). Simple mediation models highlighted the bidirectional relationship between stress and sleep in relation to inflammation among African American women.

Conclusions: Significant effects of both stress/distress and poor sleep quality on proinflammatory cytokine production during postpartum were observed uniquely among African American women. These data are consistent with an allostatic load model which predicts that conditions of chronic stress impart vulnerability to dysregulated responses to novel stressor exposures. The bidirectional nature of the stress-sleep relationship has clinical relevance. Studies examining whether interventions focused on one or both of these psychological factors during postpartum is beneficial for inflammatory profiles would be informative. In addition, examination of these models in relation to maternal health at postpartum, including delivery related wounds and other infections, is warranted.

1. Introduction

Postpartum is a period of unique psychosocial stress characterized by sleep disturbance and heightened stress. Reflecting partial sleep deprivation and fragmentation in response to the newborn's sleep-wake cycle, women spend an estimated 3 times longer awake after nocturnal sleep onset during the first several weeks postpartum compared to pregnancy or non-postpartum women with children. (Yamazaki et al., 2005; Doering, 2013; Montgomery-Downs et al., 2010; Nishihara and Horiuchi, 1998; Swain et al., 1997; Gay et al., 2004). Severity of postpartum sleep disruption is predictive of declines in marital satisfaction as well as risk for depression (Medina et al., 2009; Sleep, 2015; Bhati and Richards, 2015; Hiscock et al., 2006; Bayer et al., 2007; Hiscock et al., 2008; Dennis and Ross, 2005; Okun et al., 2011a). Moreover, stress specific to parenting is of particular relevance at postpartum; caring for a newborn entails changes in daily tasks, reductions in personal and partner time, and may introduce new financial challenges (Abidin, 1992; East and Barber, 2014; Chang et al., 2004). Further, a mother's experience of parenting as rewarding versus stressful is affected by her perceptions of both bonding with the child and the child's temperament (Abidin, 1992; East and Barber, 2014; Chang et al., 2004).

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In the context of these significant psychosocial stressors, women also experience unique physiological vulnerabilities. Infections are the most common cause of serious maternal morbidity at postpartum (Hebert et al., 1999). In the US, an estimated 32.2% of women undergo Cesarean-section, while 11.6% of those delivering vaginally have an episiotomy, which both necessitate wound healing (Friedman et al., 2015; Hamilton et al., 2015). Infections of delivery-related wounds, as well as uterine, bladder, and kidney infections and mastitis are common in postpartum, affecting 6.0–7.4% of women in the first month alone (Yokoe et al., 2001). As the majority of US deliveries occur in a hospital setting, exposure to hospital acquired-infectious illnesses including methicillin-resistant *Staphylococcus aureus* (MRSA) and group A streptococcus (GAS) can be of concern (Saiman et al., 2003; Chuang et al., 2002). Further, based on epidemiological data, the CDC now recognizes women in the first two weeks postpartum as a high risk group for acquiring seasonal influenza virus infection (Louie et al., 2009; Grohskopf et al., 2013). For these reasons, implications of sleep- and stress-induced immune dysregulation at postpartum have particular relevance.

The psychoneuroimmunology literature has delineated bi-directional associations between behavioral factors and immune function, with extensive research linking both impaired sleep and exposure to stressors with inflammatory dysregulation (Christian, 2012; Segerstrom and Miller, 2004a). Various studies link poorer self-reported sleep as well as experimentally-induced sleep restriction with increases in circulating inflammatory mediators and exaggerated ex vivo stimulated cytokine production (Van Leeuwen et al., 2009; Frey et al., 2007; Meier-Ewert et al., 2004; Prather et al., 2009; Okun et al., 2007a; Irwin et al., 2010). Moreover, data from the past three decades has established that both stressor exposure and depressive symptoms are associated with elevations in circulating inflammatory markers, as well as enhanced stimulated cytokine production (Haapakoski et al., 2015; Steptoe et al., 2007; Dowlati et al., 2010; Rohleder, 2014). Although similar effects have been observed in perinatal women, data specific to this population is sparse and has focused primarily on pregnancy rather than postpartum. For example, sleep disturbance in pregnant women has been associated with elevations in serum proinflammatory cytokines, including IL-6 and IL-8 (Okun et al., 2007b, 2011b; Blair et al., 2015). Moreover, among pregnant women, greater stress or depressive symptoms have been associated with elevations in serum cytokines, exaggerated ex vivo stimulated cytokine production, and exaggerated inflammatory responses to the seasonal flu vaccine (Coussons-Read et al., 2007; Christian et al., 2013a, 2009a).

As described by the allostatic load model, while neuroendocrine and immune responses are adaptive in the face of stressful situations, repeated or prolonged activation of the stress response can impair the body's ability to maintain allostasis (McEwen, 1998). This model predicts that conditions of chronic stress impart vulnerability to dysregulated responses to novel or additional stressor exposures (Juster et al., 2010). Thus, allostatic load is a pathway by which chronic stress associated with racial minority status may confer risk for poor health outcomes (Williams, 1999; Chen et al., 2014). Consistent with this notion, prior data, including that from our group, suggest that African American women may be particularly vulnerable to stress-induced immune dysregulation. For example, our data show that during pregnancy and non-pregnancy, African-American women exhibit more exaggerated increases in serum interleukin(IL)-6 upon exposure to an laboratory acute stressor (Trier Social Stress Test) as compared to White women (Christian et al., 2013b). Moreover, our data have shown that, compared to Whites, African American women show greater increases in serum IL-8 and related increases in risk for preterm birth in the context of poor sleep as measured at mid-gestation (Blair et al., 2015). However, racial differences in associations between psychosocial factors and inflammation at postpartum remain relatively unexamined.

The current study examined associations among sleep, psychosocial stress (i.e., parenting stress, general perceived stress), mood (i.e., depressive symptoms), serum cytokine levels, and LPS-stimulated proinflammatory cytokine production among 69 women (32 African American, 37 White) assessed at 7–10 weeks postpartum. It was hypothesized that women reporting poorer sleep, greater stress (e.g., general perceived stress, parenting stress), and/or depressive symptoms would exhibit elevated serum proinflammatory cytokine levels and exaggerated cytokine production. It was also hypothesized that these effects would be exacerbated among African Americans versus Whites. Potential mediating pathways linking sleep, stress/distress, and inflammation were examined.

2. Methods

2.1. Study design and participants

This study enrolled 84 women from The Ohio State University Wexner Medical Center (OSUWMC) and surrounding community of Columbus, Ohio. Exclusion criteria consisted of multi-fetal gestation, diagnosed fetal anomaly, health conditions or use of medications with a clear immunological or endocrinological component (e.g., cancer), illicit drug use other than marijuana, and consumption of > 2 alcoholic beverages per week per self-report or medical record at time of enrollment. Women reporting acute illness, such as cold- or flu-like symptoms, or antibiotic use within ten days of a study visit were rescheduled.

The full study included three prenatal assessments. However, the current analyses focused only on data from the 7–10 week postpartum visit. At the study visit, women provided a blood sample and completed psychosocial questionnaires. Women were excluded from the current analyses if they did not attend the postpartum visit (n = 11), were missing cytokine data (n = 2), or experienced fetal death or infant mortality (n = 2), resulting in a final analytic sample of 69. The study was approved by The Ohio State University Biomedical Institutional Review Board. Written informed consent was obtained from all participants and modest compensation provided.

2.2. Demographic characteristics

Race, marital status, age, education, annual household income, parity (primiparous/multiparous), and breastfeeding status (yes/no) were determined by self-report. Maternal body mass index (BMI; kg/m^2) was calculated using weight and height measured by nursing staff at the study visit.

2.3. Psychosocial and behavioral measures

The Pittsburgh Sleep Quality Index (PSQI) was used to assess overall sleep quality (Buysse et al., 1989). A score > 5 is indicative of clinically disturbed sleep. This measure includes seven subscales: subjective sleep quality, sleep latency (i.e., time to fall asleep), sleep duration, habitual sleep efficiency (i.e., time asleep/time in bed*100), sleep disturbance, use of sleeping medications, and daytime dysfunction. In the current study, all subscales were reported as sum scores using their original scale (e.g., minutes). The total PSQI score was calculated per guidelines (Buysse et al., 1989). The PSQI has high diagnostic sensitivity and specificity in distinguishing good and poor sleepers (Buysse et al., 1989).

The Parenting Stress Index – Short Form (PSI-SF) is a widely used 36-item measure of stress as a result of the parent-child relationship. (Abidin, 1990; Haskett et al., 2006) Three subscales comprise the PSI: parental distress, parent-child dysfunctional interaction, and difficult

child. The Center for Epidemiological Studies Depression Scale (CES-D) is a 20-item measure of depressive symptoms with 4 subscales: depressed mood, somatic symptoms, absence of positive affect, and interpersonal difficulties (Radloff, 1977). The 10-item Perceived Stress Scale (PSS), is a well-validated measure of stress and assesses a construct independent of depressive symptomatology (Cohen et al., 1983).

2.4. Serum cytokines

Whole blood was collected into vacutainer tubes while participants were in a seated position. Samples were immediately centrifuged, aliquoted, and placed in −80 °C freezer storage until analysis. Serum levels of IL-6, TNF-α, and IL-8 were assayed in duplicate on either single spot ultra-sensitive (for IL-6) or multiplex V-Plex (for TNF-α and IL-8) kits from Meso Scale Discovery (MSD, Meso Scale Discovery, 1601 Research Blvd, Rockville, MD). Plates were read by an MSD SECTOR Imager 2400 measuring electrochemiluminescence. Sample concentrations were extrapolated from a standard curve calculated using a four parameter logistic fit using MSD Workbench 3.0 software. The limits of detection (LOD) were 0.31 pg/mL for IL-6, 0.17 pg/mL for TNF-α, and 0.27 pg/mL for IL-8. All samples were above the limit of detection. The inter- and intra- assay coefficients of variation were 8.69% and 5.89% for IL-6, 5.12% and 5.34% for TNF-α, and 5.27% and 3.71% for IL-8, respectively.

2.5. LPS-stimulated proinflammatory cytokine production

PBMCs at a concentration of 1×10^6 cells/ml were stimulated with 1μg/ml LPS in RPMI-1640 supplemented with 10% human male serum for 24 h. A non-LPS media control was incubated simultaneously. Next, samples were centrifuged and aliquots removed and frozen at -80 °C until assayed. Media samples were assayed neat; LPS samples were diluted 1:6. Samples were assayed in duplicate for IL-6, TNF-α, IL-1β, and IL-8 (pg/ml) using human ProInflammatory II multiplex tissue culture kits from Meso Scale Discovery (MSD; 1601 Research Blvd., Rockville, MD). Plates were read by an MSD Sector Imager 2400 measuring electrochemiluminescence. The inter-assay coefficients of variation were 8.28%, 6.02%, 8.59%, and 9.23%, for IL-6, TNF-α, IL-1β, and IL-8, respectively. The intra-assay coefficients of variation were 3.2%, 2.36%, 1.91%, and 2.93%, for IL-6, TNF-α, IL-1β, and IL-8, respectively.

2.6. Statistical analyses

Only one woman endorsed use of sleep medication per the PSQI and 81.2% of women scored a zero on the interpersonal difficulties subscale of the CES-D; thus, these subscales were not included in examinations beyond those with total scores as outcomes. To examine group differences on demographic characteristics and psychosocial factors, chi-square tests, t-tests, or Mann-Whitney U tests were used, when appropriate. Pearson's or Spearman's correlation coefficients were used to assess relationships among psychosocial factors, serum cytokine levels, and LPS-stimulated proinflammatory cytokine production. Mediation was tested using bias-corrected 90% confidence intervals for the indirect effects, based on 10,000 bootstrap samples. A significant indirect effect is present when the confidence interval does not include 0 (Hayes, 2013). All other tests were evaluated at the p < 0.05 level of significance. No adjustments were made for multiple comparisons. Partial correlations adjusting for BMI were depicted graphically by obtaining residuals from 2 regression models: the LPS-stimulated cytokine regressed on BMI and the psychosocial measure regressed on BMI. The mean value of each outcome (cytokine and psychosocial

Table 1
Demographic characteristics by race and for the full sample, reported as mean (SD) or count (%).

Variable (units)	African American (n = 32)	White (n = 37)	t-Test or χ^2 p value
Age (years)	26.5 (4.6)	25.9 (3.7)	0.60
BMI (kg/m^2)	29.1 (5.5)	29.4 (6.0)	0.83
Parity			
Primiparous	6 (18.8%)	17 (45.9%)	0.02
Multiparous	26 (81.2%)	20 (54.1%)	
Weeks postpartum	8.4 (0.8)	8.5 (0.6)	
Currently breastfeeding	Yes = 16 (50.0%) No = 16 (50.0%)	Yes = 20 (45.9%) No = 17 (54.1%)	0.74
Marital status			
Single	6 (18.8%)	2 (5.4%)	0.11
In a relationship, not married	16 (50.0%)	16 (43.2%)	
Married	10 (31.3%)	19 (51.4%)	
Annual household income			
Less than $15,000	12 (37.5%)	7 (18.9%)	0.11
$15,000–$29,999	6 (18.8%)	16 (43.2%)	
$30,000–$49,999	8 (25.0%)	6 (16.2%)	
$50,000 and above	6 (18.8%)	8 (21.6%)	
Education			
High school graduate or less	5 (15.6%)	10 (27.0%)	0.13
Some college	18 (56.3%)	12 (32.4%)	
College graduate	9 (34.8%)	15 (40.5%)	
Currently employed	Yes = 22 (68.8%) No = 10 (31.3%)	Yes = 25 (67.6%) No = 12 (32.4%)	0.92

BMI: body mass index.

Table 2
Descriptive statistics for psychosocial factors by race and for the full sample, reported as mean (SD).

Variable (units)	African American (n = 32)	White (n = 37)	p-Values
Pittsburgh Sleep Quality Inventory	7.5 (3.7)	7.2 (2.7)	0.69
Subjective quality[a]	1.2 (0.9)	1.3 (0.7)	0.61
Sleep latency (min) (Yamazaki et al., 2005)	18.4 (16.0)	15.4 (19.2)	0.28
Sleep duration (hrs) (Yamazaki et al., 2005)	5.8 (1.4)	6.2 (1.3)	0.28
Sleep efficiency (%) (Yamazaki et al., 2005)	67.6 (18.9)	65.6 (21.6)	0.80
Sleep disturbance (Yamazaki et al., 2005)	4.5 (2.9)	6.1 (3.5)	0.07
Daytime dysfunction[a]	1.5 (1.5)	1.5 (1.4)	0.97
Parenting Stress Index – Short Form	62.1 (18.0)	62.2 (17.0)	0.99
Parental distress	26.3 (9.0)	25.3 (9.2)	0.65
Difficult child	19.5 (6.6)	20.1 (6.8)	0.72
Parent-child dysfunctional interaction[a]	16.3 (6.0)	16.8 (5.0)	0.26
Perceived Stress Scale	14.6 (6.0)	14.4 (6.9)	0.89
CES-D Scale	11.8 (7.8)	9.6 (7.6)	0.23
Depressed mood[a]	3.1 (3.1)	2.2 (2.6)	0.21
Somatic symptoms	5.8 (3.7)	5.1 (3.1)	0.38
Absence of positive affect (Yamazaki et al., 2005)	2.7 (3.1)	2.0 (2.5)	0.43

Note: Independent samples t-tests were used to test for group differences, unless otherwise noted.

[a] Mann-Whitney U Test; CES-D: Center for Epidemiologic Studies Depression Scale.

Table 3

Descriptive statistics for LPS-stimulated cytokines by race and for the full sample, reported as mean (SD).

	African American (n = 32)	White (n = 37)	*p*-Values
IL-1β (pg/ml)[a]	4248.7 (2284.8)	5703.9 (2969.0)	0.007
IL-6 (pg/ml)	19,589.0 (5111.0)	19,604.7 (6069.8)	0.99
IL-8 (pg/ml)	180,938.7 (25,989.2)	181,337.8 (25,719.4)	0.95
TNF-α (pg/ml)	3056.8 (1011.0)	2885.2 (695.5)	0.41

Note: Independent samples *t*-tests were used to test for group differences, unless otherwise noted.

[a] Mann-Whitney *U* Test; LPS: lipopolysaccharide; IL: interleukin; TNF: tumor necrosis factor.

measure) was added to each residual to return to the range of the original scale. The values of the residuals-plus-means were displayed as scatterplots with least-squares regression lines. These figures display graphically the associations adjusting for BMI. All analyses were performed using SPSS 24.0 and PROCESS macro v2.16.
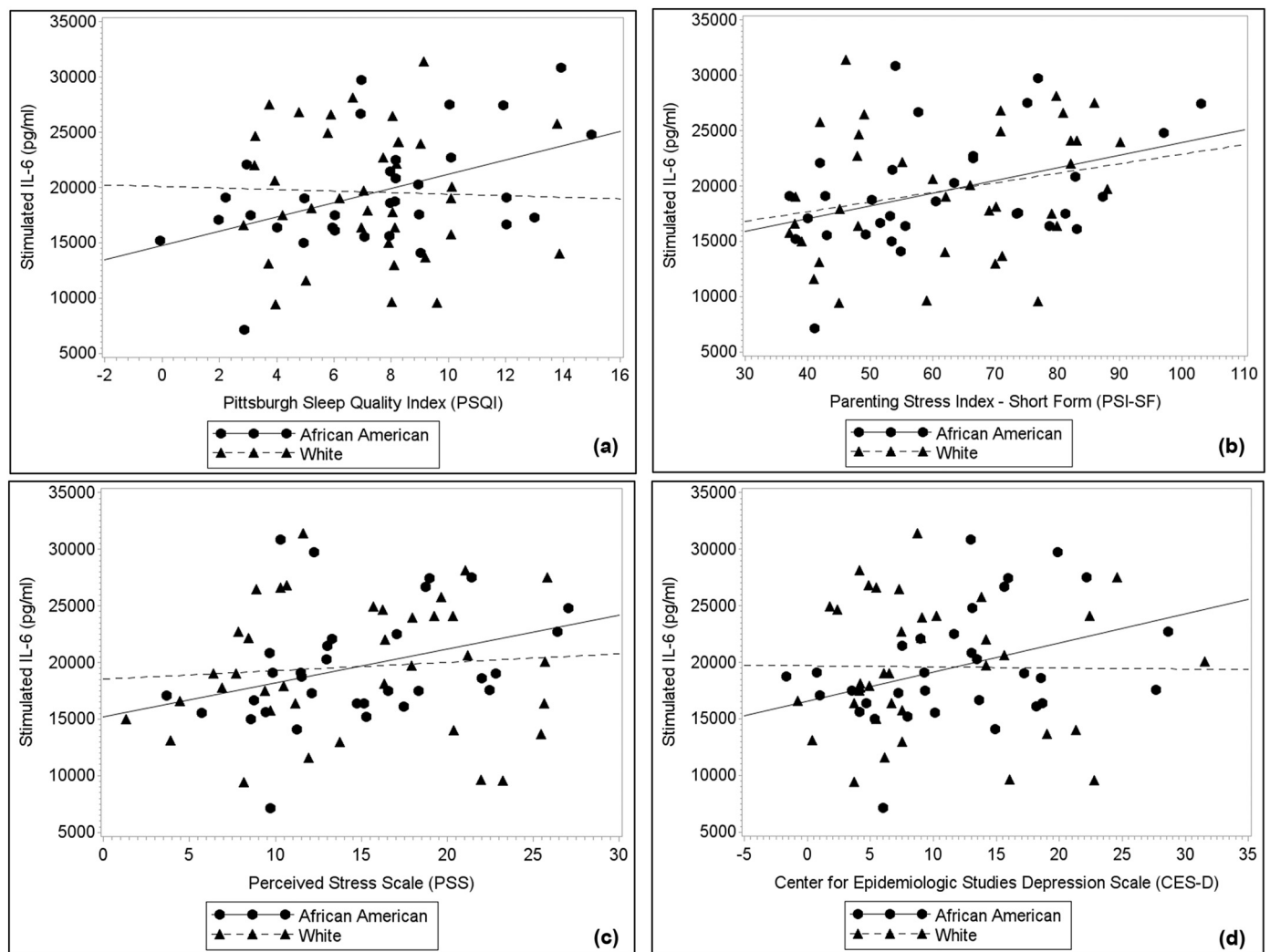
3. Results

3.1. Sample characteristics

The sample was 46% African American (n = 32) and 54% White (n = 37). Groups did not differ in age, body mass index, weeks postpartum, breastfeeding status, marital status, income, education or employment (Table 1). In addition, no racial differences were observed in psychosocial factors assessed (Table 2). Overall, women exhibited poor sleep quality, with 71% (n = 49) meeting criteria for clinically disturbed sleep (PSQI > 5). In comparison to African Americans, White women exhibited greater stimulated IL-1β production ($U = 368.0$, $z = -2.70$, $p = 0.007$; Table 3). No differences by race were observed for production of IL-6, IL-8, or TNF-α.

3.2. Psychosocial factors and LPS-stimulated proinflammatory cytokine production

Partial correlations between stimulated levels of each cytokine and

Fig. 1. Partial correlations between (a) sleep quality, (b) parenting stress, (c) perceived stress, and (d) depressive symptoms with LPS-stimulated IL-6 after adjusting for body mass index. In African American women, but not Whites, poorer sleep quality, greater parenting stress, and greater depressive symptoms were associated with greater LPS-stimulated IL-6 production (ps ≤ 0.05). In addition, a trend was observed between greater perceived stress and greater LPS-stimulated IL-6 among African American women ($p \leq 0.10$). Plots represent the values of the residuals-plus-means with least-squares regression lines.
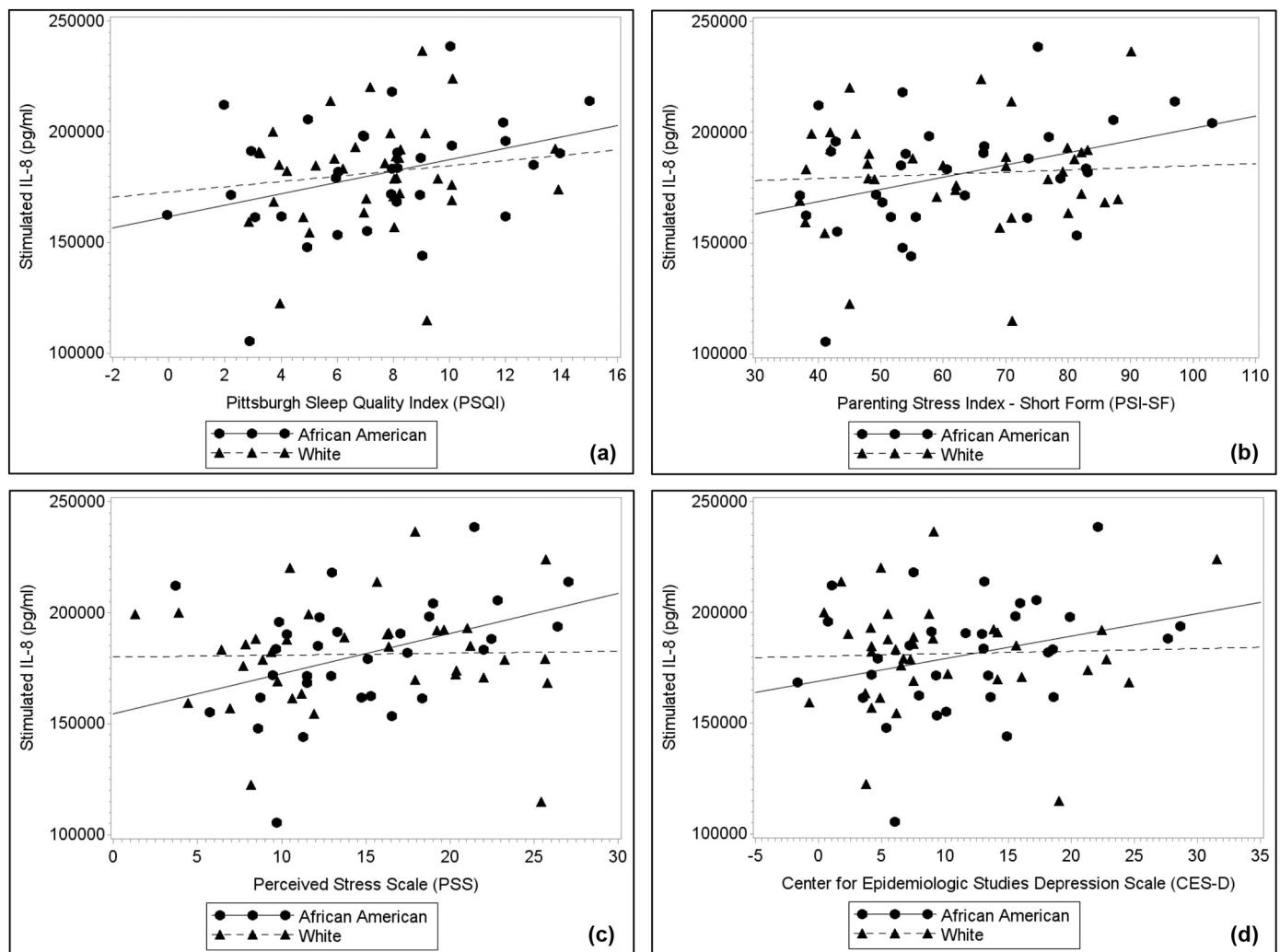
Fig. 2. Partial correlations between (a) sleep quality, (b) parenting stress, (c) perceived stress, and (d) depressive symptoms with LPS-stimulated IL-8 after adjusting for body mass index. In African American women, but not Whites, poorer sleep quality, greater parenting stress, greater perceived stress, and greater depressive symptoms were associated with greater LPS-stimulated IL-8 production (ps ≤ 0.05). Plots represent the values of the residuals-plus-means with least-squares regression lines.

psychosocial factors were examined separately by race, after controlling for BMI. Key results are depicted in Figs. 1 & 2 and Tables 4 & 5. In relation to serum markers, no significant associations between psychosocial factors and serum cytokine levels were observed among women of either race (ps > 0.05). Moreover, among White women, no significant relationships between psychosocial factors and stimulated production of IL-6, IL-8, or IL-1β (ps ≥ 0.06) were observed. However, greater general stress (PSS) was associated with lower TNF-α production ($r = -0.33$, $p = 0.05$).

Among African American women, controlling for BMI, poorer sleep quality, greater parenting stress, and greater depressive symptoms (per CES-D total score and/or subscales) were associated with both greater stimulated IL-6 and IL-8 production (ps ≤ 0.05; Tables 4 & 5). Greater stimulated IL-8 production was also observed in relation with greater general perceived stress ($p \leq 0.05$) Table 5. In addition, greater depressive symptoms (per CES-D depressed mood subscale), were associated with greater LPS-stimulated TNF-α production ($r = 0.42$, $p = 0.02$). No associations between psychosocial factors and IL-1β production were observed among African Americans (ps ≥ 0.29).

3.2.1. Mediating models

Expected associations between stimulated cytokine production and psychosocial factors were observed exclusively among African American women in the cohort and most consistently in relation to IL-6 and IL-8 production. Thus, subsequent mediation analyses focused on these markers among African American women. Correlations among key psychosocial variables are shown in Table 6. In a series of simple mediation models, we first tested overall sleep quality (PSQI total score), as a potential mediator linking psychosocial stress/distress (PSI-SF, PSS, and CES-D) with stimulated IL-6 and IL-8 production (Table 7). As shown, controlling for BMI, sleep quality played a mediating role in the relationship of both parenting stress and depressive symptoms with stimulated production of both cytokines.

Next, models were tested whereby sleep quality (PSQI total score) served as the primary causal factor (Table 8). As shown, models linking PSQI scores to stimulated IL-6 and IL-8 production via parenting stress (PSI-SF) were significant. In addition, the model linking sleep quality (PSQI) with stimulated IL-6 production via depressive symptoms (CES-D) was significant, while this model for IL-8 approached significance.

Table 4
Correlations between stimulated interleukin-6 and psychosocial factors by race and for the full sample.

	African American (n = 32)	White (n = 37)	Total (N = 69)
Pittsburgh Sleep Quality Inventory - Total	0.46**	− 0.02	0.22[†]
Subjective quality[a]	0.33[†]	− 0.05	0.09
Sleep latency[a]	0.25	− 0.09	0.05
Sleep duration[a]	− 0.51**	− 0.19	− 0.29*
Sleep efficiency[a]	− 0.38*	0.03	− 0.12
Sleep disturbance[a]	0.47**	0.10	0.25*
Daytime dysfunction[a]	0.29	0.15	0.27*
Parenting Stress Index -Total	0.41*	0.26	0.30*
Parental distress	0.48**	0.22	0.30*
Difficult child	0.35[†]	0.29[†]	0.28*
Parent-child dysfunctional interaction[a]	0.18	0.14	0.16
Perceived Stress Scale	0.33[†]	0.08	0.14
CES-D Scale - Total	0.37*	− 0.02	0.13
Depressed mood[a]	0.46**	− 0.01	0.17
Somatic symptoms	0.29	0.04	0.14
Absence of positive affect[a]	0.36*	0.09	0.17

All analyses control for BMI.
Note: Higher Total PSQI scores, subjective quality rating, and sleep latency reflect greater severity of sleep problems.
CES-D: Center for Epidemiologic Studies - Depression Scale.
[a] Spearman correlation. Pearson correlations were used unless otherwise noted.
[†] *p* ≤ 0.10.
[*] *p* ≤ 0.05.
[**] *p* ≤ 0.01.

Table 5
Correlations between stimulated interleukin-8 and psychosocial factors by race and for the full sample.

	African American (n = 32)	White (n = 37)	Total (N = 69)
Pittsburgh Sleep Quality Inventory – Total	0.36*	0.14	0.27*
Subjective quality[a]	0.30	0.01	0.12
Sleep latency[a]	0.07	0.13	0.10
Sleep duration[a]	− 0.47**	− 0.10	− 0.28*
Sleep efficiency[a]	− 0.18	− 0.04	− 0.12
Sleep disturbance[a]	0.37*	0.30[†]	0.35**
Daytime dysfunction[a]	0.34[†]	− 0.15	0.17
Parenting Stress Index – Total	0.39*	0.07	0.22[†]
Parental distress	0.39*	0.10	0.22[†]
Difficult child	0.39*	0.21	0.27*
Parent-child dysfunctional interaction[a]	0.16	− 0.12	0.02
Perceived Stress Scale	0.42*	0.02	0.17
CES-D Scale - Total	0.32[†]	0.02	0.16
Depressed mood[a]	0.47**	0.07	0.23[†]
Somatic symptoms	0.14	0.11	0.12
Absence of positive affect[a]	0.31[†]	0.11	0.15

All analyses control for BMI.
Note: Higher Total PSQI scores, subjective quality rating, and sleep latency reflect greater severity of sleep problems.
CES-D: Center for Epidemiologic Studies - Depression Scale.
[a] Spearman correlation. Pearson correlations were used unless otherwise noted.
[†] *p* ≤ 0.10.
[*] *p* ≤ 0.05.
[**] *p* ≤ 0.01.

4. Discussion

In the current study, we examined associations between sleep quality and psychosocial stress/distress with serum proinflammatory cytokines and LPS-stimulated proinflammatory cytokine production among women at 7–10 weeks postpartum. Our results showed effects of parenting stress, general perceived stress, depressive symptoms, and poor sleep quality on enhanced proinflammatory cytokine production, uniquely among African American women. This is consistent with our prior data which show greater inflammatory dysregulation in response to acute stressor exposures as well as poor sleep among African American versus White women during pregnancy (Blair et al., 2015; Christian et al., 2013b). These findings are also in keeping with the concept of allostatic load, which posits that exposure to repeated or chronic stress impairs the ability to most adaptively respond to new or additional stressors (McEwen, 1998).

In the current investigation, we did not observe any associations between psychosocial parameters and serum levels of proinflammatory cytokines. This may, in part, reflect greater sensitivity provided by LPS-stimulation which induces a robust response, greatly enhancing variability within the sample. Thus, it is plausible that associations at the serum level could be observed in a larger cohort. However, it is of note that our prior data on acute stress reactivity demonstrate that African American and White women (in pregnancy and non-pregnancy) exhibit similar levels of serum IL-6 at baseline; racial differences in inflammatory profiles emerged only *after* the acute stressor exposure. (Christian et al., 2013a). In parallel, in the current study, while serum levels represent a "resting" state, ex vivo LPS-stimulation provides a proxy measure for how the cells respond under conditions of bacterial challenge. Thus, both our current and prior findings are in keeping with an allostatic load conceptualization. Together, these studies provide evidence that, compared to White women, African Americans exhibit impaired ability to maintain physiological allostasis upon exposure to both psychological and biological challenges.

Moreover, in the current study, associations were seen primarily in relation to LPS-stimulated production of IL-6 and IL-8. A large literature suggests that IL-6 is particularly responsive to psychosocial stress and sleep disturbance. (Segerstrom and Miller, 2004b; Irwin et al., 2016). Further, our data and others has previously linked disrupted sleep in perinatal women specifically with IL-8 (Okun et al., 2011b; Blair et al., 2015). IL-8 is a potent chemoattractant implicated in endothelial dysfunction which plays an important role in the development of atherosclerosis and is implicated in risk of cardiovascular events (Boekholdt et al., 2004; Gerszten et al., 1999). Thus, dysregulation of these inflammatory markers at postpartum may be of particular relevance.

In mediation analyses, which were conducted only among African American women, we tested two directional pathways by which sleep quality may be linked with psychological stress/distress in affecting inflammatory responses. Results supported both pathways: links from stress to inflammation via sleep quality as well as from sleep quality to inflammation via stress. This is consistent with the literature in that stress and sleep quality are bi-directionally related (Kahn et al., 2013; Minkel et al., 2012; Sin et al., 2017). These models assume that psychosocial factors are impacting inflammation, rather than the reverse; in the unique context of postpartum in which new stressors (e.g., parenting responsibilities, financial obligations) and sleep disturbance are introduced, this directional pathway is arguably most robust. However, effects of inflammation on sleep and stress/depressive symptoms cannot be ruled out. In addition, these data did not provide longitudinal assessment at postpartum, which would permit for clearer delineation of mediating pathways linking psychosocial parameters of interest.

Table 6
Correlations between sleep quality and psychological measures for African American women (n = 32).

	PSQI total	Sleep duration[a]	Sleep efficiency[a]	Sleep disturbance[a]	PSI total	PSI parental distress	PSI difficult child	PSS total	CES-D total[a]	CES-D depressed[a]	CES-D positive affect[a]
PSQI total		−0.80***	−0.62***	0.29	0.35*	0.50**	0.26	0.21	0.24	0.11	0.29
Sleep duration[a]			0.74***	−0.08	−0.29	−0.28	−0.42*	−0.04	0.01	−0.03	−0.15
Sleep efficiency[a]				0.05	−0.21	−0.22	−0.22	−0.00	0.03	0.06	−0.11
Sleep disturbance[a]					0.20	0.25	0.15	0.16	0.30†	0.18	0.24
PSI total						0.84***	0.89***	0.65***	0.47***	0.61***	0.49**
PSI parental distress							0.58***	0.67***	0.63***	0.63***	0.52**
PSI difficult child								0.51**	0.28	0.42*	0.36*
PSS total									0.66***	0.62***	0.60***
CES-D total[a]										0.82***	0.64***
CES-D depressed[a]											0.39*
CES-D positive affect[a]											

PSQI: Pittsburgh Sleep Quality Inventory; PSI: Parenting Stress Index; PSS: Perceived Stress Scale; CES-D: Center for Epidemiologic Studies - Depression Scale.

[a] Spearman correlations; Pearson correlations used unless otherwise noted.

† $p \leq 0.10$.

* $p \leq 0.05$.

** $p < 0.01$.

*** $p \leq 0.001$.

The bi-directional nature of the stress and sleep relationship is likely exacerbated at postpartum. Parenting a newborn is itself a substantial stressor exposure, involving changes in daily routines, work schedules, social and partner relationships, and acquisition of new skills (particularly for first time parents). Moreover, caring for a newborn commonly requires multiple sleep/wake cycles. Thus, for women who have difficulty falling or staying asleep, these issues are encountered not once, but multiple times in a given night. These characteristics of postpartum sleep may contribute to increased parenting stress and also exacerbate or enhance vulnerability to depressed mood. Studies examining whether interventions focused on one *or* both of these psychological factors during postpartum is beneficial for inflammatory profiles would be informative.

These findings may have implications for maternal health at postpartum. As described, this is a period of unique vulnerability in that the experience of delivery-related wounds which require healing is common. In addition, uterine, bladder, kidney infections and mastitis may occur. Via effects on immune function, stress and sleep disruption may contribute to poor healing and risk for infection. Studies examining stress, inflammation, and specific health outcomes including occurrence of delivery-related wounds and other infections would inform our understanding of the health implications of the current findings.

Of note, in the current study, we utilized the CES-D as our measure of depressive symptoms, rather than the Edinburgh Postnatal Depression Scale (EPDS). The CES-D is commonly administered and shows predictive validity in perinatal studies (Li et al., 2009; Phillips et al., 2010; Orr et al., 2002; Christian et al., 2009b; Christian et al., 2010). However the EPDS was specifically designed for use in postpartum (Cox et al., 1987). A key differentiating feature of these two scales is that the EPDS purposefully excludes assessment of somatic symptoms (e.g., sleep difficulties, fatigue, changes in appetite) with the rationale of avoiding overlap with typical perinatal experiences. This is problematic for studies of immune underpinnings of depression, because sickness behaviors elicited by inflammatory exposure are largely somatic in nature (Dantzer and Kelley, 2007; Dantzer et al., 2008; Raison et al., 2006). In keeping with the larger literature in non-perinatal adults, our prior data suggest that somatic symptoms are of importance in predicting immune and neuroendocrine dysregulation in perinatal women (Christian et al., 2016). While somatic symptoms were not the primary driver of observed associations between depressive symptoms and inflammatory profiles in the current investigation, assessment using the CES-D permitted for this determination. Thus, we propose that, despite some possible overlap with "normal" perinatal experiences, assessing somatic symptoms is critical for validity and clarity in studies linking depressive symptoms with biological factors in perinatal women.

The current study focused on predominately lower income African American and White women. Thus, generalizability to other groups is unknown. It is plausible that the observed effects would not be present among women with greater socioeconomic resources. In addition, although African American and White women were generally demographically similar, we did not assess characteristics such as family wealth; at the same education and income levels, Whites have markedly greater wealth than African Americans which substantially affects economic security (Killewald et al., 2017). Groups may differ in wealth, or in other meaningful ways that were not captured herein. Also, the sample size of this study introduced limitations with regard to statistical modeling; while we observed differential effects in African Americans versus Whites when examined separately, the study was not sufficiently powered to test formal moderation, or moderated mediation which would provide stronger evidence. The current analyses do not "rule out" the possibility that these effects are also applicable among White women, albeit effects may be smaller and thus require a larger sample to detect. These results should be considered within these limitations.

In sum, the current study demonstrates that among postpartum women, poorer sleep quality, as well as greater parenting stress,

Table 7
Mediation models testing overall sleep quality per PSQI total score as a mediator.

	Effect	SE	90%CI estimates		Effect	SE	90%CI estimates
PSI → PSQI → IL-6	35.33	25.22	6.71, 97.93[a]	PSI → PSQI → IL-8	128.57	107.83	7.77, 399.53[a]
PSS → PSQI → IL-6	70.53	72.26	− 13.31, 221.44	PSS → PSQI → IL-8	251.57	291.27	− 53.67, 899.90
CES-D → PSQI → IL-6	60.06	47.95	3.95, 166.79[a]	CES-D → PSQI → IL-8	237.97	194.62	10.63, 697.10[a]

[a] Significant effect; PSI: Parenting Stress Index; PSQI: Pittsburgh Sleep Quality Inventory; IL: interleukin; PSS Perceived Stress Scale; CES-D: Center for Epidemiologic Studies – Depression Scale.

Table 8
Mediation models testing psychological factors as mediators.

	Effect	SE	90%CI estimates		Effect	SE	90%CI estimates
PSQI → PSI → IL-6	135.52	126.09	4.08, 435.30[a]	PSQI → PSI → IL-8	752.78	618.01	39.39, 2142.06[a]
PSQI → PSS → IL-6	66.47	86.69	− 14.52, 288.21	PSQI → PSS → IL-8	530.26	596.59	− 128.11, 1889.77
PSQI → CES-D → IL-6	87.88	81.26	3.78, 287.60[a]	PSQI → CES-D → IL-8	408.18	453.65	− 19.17, 1524.33

[a] Significant effect; PSI: Parenting Stress Index; PSQI: Pittsburgh Sleep Quality Inventory; IL: interleukin; PSS Perceived Stress Scale; CES-D: Center for Epidemiologic Studies – Depression Scale.

perceived stress, and depressive symptoms are predictive of enhanced stimulated proinflammatory cytokine production. These effects were uniquely observed among African American women in this cohort. Mediation models within African American women supported sleep as both a mediator and initiator linking stress/distress with enhanced inflammatory responding, highlighting the bidirectionality of these constructs at postpartum. Future studies incorporating longitudinal design and specific health outcomes (e.g., postpartum infections) would further our understanding of the health implications of stress and sleep-induced immune dysregulation at postpartum.

Conflicts of interest

The authors declare no conflicts of interest.

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