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# Chronic Stress and C-Reactive Protein in Mothers during the First Postpartum Year

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## Abstract

**Objectives:** Elevated levels of C-reactive protein (CRP) are associated with increased risk of cardiovascular and metabolic disease. The current study tested associations between psychosocial stress and CRP in a large sample of women during the first postpartum year.

**Methods:** We analyzed data collected by the five site Community Child Health Network study, which studied a predominately poor population. Participants ($n$ = 1206 women; 54% African American, 23% White, 23% Hispanic/Latina) were recruited shortly after the birth of a child. Multiple linear regression analyses tested associations of psychosocial stress in several life domains (financial, neighborhood, family, co-parenting, partner relationship, discrimination, and interpersonal violence) with log-transformed CRP concentrations at 6 months and 1 year postpartum.

**Results:** Forty-eight percent of participants showed evidence of elevated CRP (≥3 mg/L) at 6 months postpartum, and 46% had elevated CRP at 12 months postpartum. Chronic financial stress at 1 month postpartum predicted higher levels of CRP at 6 ($b$ = .15, $SE$ = .05, $p$ = .006) and 12 months postpartum ($b$ = .15, $SE$ = .06, $p$ = .007) adjusting for race/ethnicity, income, education, parity, health behaviors, and chronic health conditions, though associations became non-significant when adjusted for body mass index (BMI).

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**Conclusion:** In this low income and ethnic/racially diverse sample of women, higher financial stress at one month post birth predicted higher CRP. Study findings suggest that perceived financial stress stemming from socioeconomic disadvantage may be a particular deleterious form of stress affecting maternal biology during the year after the birth of a child.

**Keywords**

CRP; inflammation; stress; postpartum health

Chronic stress is defined as ongoing, enduring demands that threaten to exceed the resources of an individual in areas of life such as family, marriage, parenting, work, health, housing and finances (1). Prior studies implicate chronic stressors such as unemployment, poverty, discrimination, difficult interpersonal relationships, and caregiving to increased risk of cardiovascular disease (2,3), depression (4,5), and all-cause mortality (6). Given the strong evidence that chronic stress is harmful to health, the focus has shifted to how stress affects physiological processes relevant to disease.

One of the hypothesized pathways through which chronic stress may impact morbidity and mortality is through stress-induced dysregulation of the immune system that results in chronic low-grade inflammation (7,8). Chronic low-grade inflammation refers to conditions characterized by persistently high levels of pro-inflammatory cytokines (such as IL-6, TNF-α, IL-1, IL-1ra, sTNF-R) and C-reactive protein (CRP). In particular, elevated CRP levels are associated with risk of myocardial infarction and stroke, and the development of chronic diseases including cardiovascular disease and Type II diabetes (9,10).

A significant relationship between psychological stress and inflammation has been observed in a number of investigations spanning a range of methodologies and populations (11). Experimental evidence for the inflammatory effects of stress comes from controlled laboratory studies in humans showing that acute stress exposure elicits significant increases in circulating levels of pro-inflammatory cytokines including TNF-α, IL-6, and Il-1β (12,13). Observational studies have shown significant positive associations between circulating levels of inflammatory markers including CRP, IL-6 and TNF-α and a variety of stressors including job strain (14), discrimination (15), early life stress (16,17), and interpersonal stress (18). In addition, levels of CRP also tend to be higher among individuals of lower socioeconomic status (SES; 19,20,21), who often experience higher levels of chronic stress and may have fewer resources to cope.

While stress has been linked to poor immunologic function and illness in the general adult population, the impact of stress on immune function and subsequent health outcomes has not been well explored in the context of human pregnancy (22) or during the postpartum period (23). Maternal physiology undergoes substantial changes during this time, including immunological shifts and alterations in body weight and composition. For example, pregnancy results in increases in maternal body weight, and weight gained during pregnancy may be retained through the postpartum period, potentially increasing the risk of overweight and obesity among childbearing women. In particular, women who are overweight or obese before becoming pregnant and those who gain more than recommended by the Institute of Medicine guidelines for weight gain during pregnancy (currently 25–35 lbs for women with

a normal pre-pregnancy BMI and less for those who are overweight or obese) are at risk of retaining excess weight after delivery (24–26). Excess body weight is, in turn, associated with a number of cardiovascular risk factors in the general population, including systemic inflammation. Adipocytes synthesize and secrete proinflammatory cytokines, and there is a well-established link between higher BMI and elevated levels of circulating inflammatory markers including proinflammtory cytokines and CRP (27,28).

In addition to the shifts in body weight that occur during pregnancy and postpartum, a number of adaptions in both the innate and adaptive immune systems occur during pregnancy. These include decreases in Th1 cytokines and resulting Th2 dominance, as well as the upregulation of innate immunity (29,30). Though excessive inflammation contributes to the development of pregnancy complications such as the development of preeclampsia, fetal distress and preterm birth (31,32), controlled inflammation is critical to healthy pregnancy and plays a role in processes including implantation and parturition. Prior studies have demonstrated that the immunological effects of pregnancy remain until up to a year after delivery (33), and postpartum women tend to have higher levels of pro-inflammatory cytokines (34,35).

To our knowledge, only four published studies have examined CRP during the postpartum period, and none of these studies examined associations with chronic stress or other psychological variables. A recent study of 822 women in the Philippines found higher median levels of CRP in women who had given birth in the previous year as compared to women who had given birth more than a year prior to the assessment, though associations were attenuated when adjusting for maternal adiposity (36). Another study of 181 U.S. women at 4 to 6 weeks postpartum found higher levels of CRP in comparison to 33 control participants who had not given birth; though associations of CRP with BMI were not reported in this study, there were no statistically significant differences in current BMI between groups and it is unlikely that BMI accounts for the observed differences in CRP (37). Finally, two small, recent studies have shown positive associations between CRP and maternal body mass index (BMI) during the postpartum period (38,39) which fits with the nonpregnant link between obesity and inflammation (27,28).

In sum, CRP is a potentially important risk marker for future adverse health outcomes in women following the birth of a child, but there is little available descriptive data on levels of CRP during the first postpartum year among women of diverse race/ethnicity and predominantly low socioeconomic status in the U.S. Moreover, the demographic, medical, and behavioral correlates of this key marker of systemic inflammation have not been established. To address these gaps, we utilized data collected from a large, diverse sample of women over the course of one year following the birth of a child.

## Methods

The sample was drawn from the larger pool of participants in the Community Child Health Network (CCHN) study, which was a five-year longitudinal, multi-site study of 2,510 mothers, and 1,436 of the fathers of their children. CCHN was funded by The Eunice Kennedy Shriver National Institutes of Child Health and Human Development (NICHD) of

NIH to investigate disparities in maternal child health and improve the health of families. The overall goal of CCHN was to gain new insights into disparities in maternal health and child development through community-based participatory methods of research (40).

The five study sites included three urban sites (Washington, DC; Baltimore, MD; Los Angeles County, CA); one suburban site (Lake County, IL); and one rural site (seven counties in eastern North Carolina). At the time of funding, each of the five sites was selected by NIH review of competitive grants in part on the basis of having presented epidemiologic evidence indicating their study sites had disparities in maternal and child health and the populations to be studied were diverse and disproportionately low or very low income. In addition, each site was required to have a community partner engaged as a co-principal investigator. These five sites were funded as an NICHD network under a U mechanism for three years to plan their research and thereafter each site was funded to participate in the collaborative five-year study that produced the data used here. With input from community partners, specific catchment areas within each site were determined for recruitment in health care settings. Women residing in the target areas were recruited and enrolled during their postpartum hospital stay following the birth of an index child (except in North Carolina where participants were recruited in clinics during pregnancy or post partum). Mothers who met the following criteria were eligible to participate: (1) between 18 and 40 years of age; (2) self-identification as either White/Caucasian, Latina/Hispanic, and/or African American/Black; (3) ability to converse in either English or Spanish; (4) residence in one of the target zip codes for at least 6 months; (5) 4 or fewer children; and (5) no plans to be surgically sterilized following the birth of the index child. The baby's father was also invited to participate in the study with the mother's permission.

Structured interviews were administered during in-home visits when index children were approximately 1 month (T1), 6 months (T2), and 12 months (T3) of age. Community members experienced and/or trained in community research or clinical service delivery conducted interviews in the participant's choice of either English or Spanish. The trained interviewers typically conducted assessments in the participant's home, with rare exceptions. Interviewers were also trained to collect biomarker data at the T2 and T3 study visits including height and weight for calculation of BMI and blood spots for CRP assays.

## Participants

Data were collected between June 2008 and December 2011. We included data from participants who completed both the T2 (24–29 weeks postpartum) and T3 (50–65 weeks post partum) study visits and also provided a usable blood spot at either timepoint. Of the 2,510 mothers in the full CCHN cohort, 1,364 participants (54%) completed both the T2 and T3 study visits. Participants were also excluded from analyses if they were pregnant at the time of the T2 or T3 visit ($n$ = 137). Of the 1,227 remaining eligible mothers, 88% provided a usable blood spot at T2 or T3 ($n$ = 1,206). Both CRP measurements were available for 1,112 of these mothers. Compared to the full CCHN cohort, these participants were less likely to be from Los Angeles (11% vs. 17%) and more likely to be from Lake County (26% vs. 23%) and North Carolina (20% vs. 17%). There were no other significant demographic differences between the full cohort and the sample included in the present analyses.

## Measures

### Stress measures.

**Financial stress.:** A financial stress index was created from five questions administered during the T1 interview. These items included the following: (1) “To what extent were worries about food, shelter, health care, and transportation stressful for you during your pregnancy?”; (2) “To what extent were money worries like paying bills stressful for you during your pregnancy?”; (3) “In the past year, did you have serious problems with money (such as a major loss of income or a debt that cannot be repaid)?”; (4) “How difficult is it for (you/your household) to meet the monthly payments on your (household’s) bills?”; (5) “How much do you worry that your total (household) income will not be enough to meet your (household’s) expenses and bills? (taken from National Survey of Families and Households).” Participants’ responses to these five items were standardized and averaged to create a composite score with a range of 1 to 4. Higher scores indicated higher levels of financial stress. Cronbach’s α for this composite score was .77.

**Chronic life stress.:** Participants completed the semi-structured CCHN Life Stress Interview (LSI; 41) at T2. Participants responded to open-ended questions regarding *neighborhood environment*, *family relationships*, *co-parenting* and *partner relationship* in the previous six months (or since the birth). Based on the objective conditions reported by the participant, interviewers assigned overall ratings in each domain. For each domain, interviewers assigned a score using a five-point Likert scale ranging from 1 (*exceptionally positive conditions*) to 5 (*exceptionally negative conditions*). Trained CCHN interviewers conducted all scoring during or immediately after administration of the interview. Interviews were also audio-recorded for later reliability and content analysis, and field stress ratings have subsequently demonstrated acceptable reliability and validity (41). Chronic stress summary scores (i.e. total LSI scores) were computed by averaging ratings over the four domains of Neighborhood, Family, Partner, and Co-parenting.

**Perceived stress.:** Perceived stress was measured at T1 and T2 using the 10-item version of the Perceived Stress Scale (PSS; 42). Responses to the 10 items were each rated on a scale ranging from 1 (*never*) to 5 (*almost always*) and summed after four positively worded items were reverse-coded. T1 and T2 scores were averaged to create a composite measure of perceived stress during the first six months after the birth of the index child, and this measure had a Cronbach’s α of .89.

**Everyday racial discrimination.:** Experiences of discrimination were measured at T1 using the Everyday Racism Scale (43). This measure assesses frequency of experiences of discrimination in everyday life such as being treated with less courtesy than others and receiving poorer service in restaurants or stores. In addition to the 9 items of the original scale, an item “being followed around the store” was added by CCHN. Responses were provided on a 6-point scale ranging from 1 (*almost everyday*) to 6 (*never*). Participants who answered “a few times a year” or more to at least one of 10 items were then asked what they thought was the main reason for these experiences (e.g., race, gender, sexual orientation). To create a racial discrimination composite score, 5 (*less than once a year*) and 6 (*never*) categories were combined, and responses to individual items were then recoded so that the

response scale ranged from 0 to 4, with higher scores indicating more frequent experiences of discrimination. The measure used in this study includes the sum of experience ratings that were attributed to race, skin color, accent or ancestry, with a possible range of 0 to 40. If the participant reported experiences of everyday discrimination but attributed those experiences to other reasons (e.g., gender, sexual orientation, age, height or weight), the racial discrimination score was set to zero. Cronbach's α for this measure was .81.

**Interpersonal violence.:** At one year postpartum, mothers were given a modified version of the HITS (for Hurt, Insult, Threaten, Screamed at; 44). The HITS asks about the frequency of physical hurt, insult, threats, and screaming occurring over the past year on a 5-point scale (never = 1 to frequently = 5). The modified form we used includes one additional item on domination or emotional control that has been included in other studies and is answered using the same scale (45): "How often does your partner/spouse restrict your actions? By actions we mean things such as spending money, visiting with family or friends, or going places that you need to go."

**C-reactive protein.:** High sensitivity C-reactive protein (hs-CRP, referred to hereafter as CRP) was measured in finger stick blood spots provided by participants at 6 months (T2) and 1 year postpartum (T3). The participant's finger was pricked with a sterile contact-activated lancet (commonly used by diabetics to test blood glucose levels) and five or more drops of blood were spotted onto blood spot collection cards purchased from Ahlstrom. The finger-stick method offers an efficient and convenient way to measure CRP in community populations because venipuncture is not required and nonmedical personnel can collect samples. Hs-CRP assayed from blood spots has shown strong correlations with serum levels of hs-CRP (46,47). After collection, cards were allowed to dry for 30 minutes, stored in plastic bags with desiccant, and then stored frozen at −30°C until analysis. ZRT laboratories (Beaverton, OR) analyzed the samples using a high-sensitivity enzyme-linked immunosorbent assay (ELISA) protocol developed for use with blood spots (46). The lower detection limit for CRP was 0.1 mg/L. Intra-assay coefficients of variation (CVs) ranged from 4.77% to 7.73% and inter-assay CVs ranged from 4.86% to 11.29%.

**BMI.:** Study staff obtained measures of height and weight at T2 and T3 home visits. Height was measured to the nearest 0.1 inch using a yellow, rigid measuring tape and weight was measured using a calibrated digital scale to the 0.01 lb unless participant was over 350 lbs. Because the maximum limit for the scales was 350 lbs, weights of participants over this limit were coded as 350 lbs ($n$ = 4 at T2 and $n$ = 2 at T3). Body mass index (BMI) was calculated by dividing weight in pounds by height in inches squared and multiplying by a conversion factor of 703 (BMI = weight (lb) / [height (in)]$^2$ x 703).

**Covariates.:** Demographic variables included participants' self-reported primary racial/ethnic identification at the time of study enrollment (African American/Black, White/Caucasian or Latina/Hispanic), years of education completed, and per capita household income (total household income from all sources before taxes divided by the number of individuals in the household), and study site. Because the distribution of per capita household income was not normally distributed (skew= 7.43, kurtosis = 90.56), included

several outliers at the upper end of the distribution, and preliminary analyses indicated a non-linear association between income and CRP, cost of living adjusted per capita household income was coded into quintiles based on the sample distribution. Education was coded as a single continuous variable (years of education) rather than as discrete categories of educational attainment because preliminary analyses revealed a linear association between education and CRP such that CRP gradually decreased with increasing educational attainment. Therefore, we elected to include a single continuous education variable rather than several dummy coded categorical variables to allow for a more parsimonious model while also capturing the full range of education within these discrete categories.

Physical activity was assessed at T2 (6 months postpartum) using the short 9-item form of the International Physical Activity Questionnaire (IPAQ). Using the instrument's well-validated scoring protocol (48,49), participants were assigned to moderately/highly active or low activity categories.Smoking status was defined as current, past, or never based on participant self-report at T2. Participants also reported alcohol use at T2 and were categorized as excessive drinkers if they reported drinking 8 or more drinks per week or 4 or more drinks per day of drinking (http://www.cdc.gov/alcohol/faqs.htm). Participants also reported their average number of hours of sleep per night in the previous month. Breastfeeding status was categorized based on participant self-report as either never breastfeed and/or breastfed for less than 6 months vs. breastfed for 6 months or more.

Physical health status was coded using data obtained through abstraction of participant's hospital charts at the time of study enrollment, and by participant self report during the T2 interview. Presence or absence of the following cardiovascular-related health conditions were of interest in the present analyses: high blood pressure or hypertension ($n$ = 268), high cholesterol ($n$ = 79), heart problems ($n$ =81), and diabetes ($n$ = 82). Participants were classified as having a particular condition if their charts indicated a history of that condition during the current pregnancy, or if they reported during the T2 interview that a doctor or nurse had ever said that they had the condition. Information about medication usage was also collected at T2. Dummy-coded variables (0 = *no*, 1 = *yes*) indicating self-reported use of anti-hypertensive, cholesterol-lowering, antidepressant, and oral contraceptive medications were included in analyses.

## Data Analytic Plan

First, frequencies and descriptive statistics were used to summarize data on study variables including demographic covariates, health behaviors, stress variables, BMI, and CRP. Correlational analyses and ANOVA were used to test bivariate relationships between study variables in preliminary analyses. Multivariate analyses examined the relationships of the stress variables with continuous log-transformed CRP values using a series of hierarchical linear regression models. Separate models were run for each of stress variables, and the stress variable was entered in Step 1. Bivariate associations between the stress variables and CRP were tested in the first model. The second model added adjustment for demographic and socioeconomic factors, and the third added health behaviors and health status indicators. The fourth added BMI to determine whether stress was associated with inflammation net of adiposity, or whether adiposity could partially explain any associations. All continuous

variables were mean-centered prior to use in regression analyses, and significance was set at $\alpha = .05$. Statistical analyses were conducted using Stata 13.

### Missing Data

To facilitate consistency with other publications using the CCHN data, the current study applies some of the approaches to missing data handling that were decided upon by the CCHN's central Data Coordination and Analyses Center (DCAC). For example, when items that were needed to compute stress scale scores were missing, mean replacement was used if at least 70% of items were complete (see 50). In regression analyses, missing values for other covariates (per capita household income, education, parity, BMI, health conditions, medication use, alcohol use, sleep, breastfeeding, and smoking) were imputed using multiple imputation (*mi*) procedures in Stata 13. The covariates with the largest percentage of missing data were per capita household income (17%), parity (6%) and participant report of recent illness at T2 (5%) and T3 (4%). All other covariates were missing for less than 3% of the sample. Ten imputations were generated using chained equations procedures (*mi impute*). Results across the ten imputed data sets were averaged, and the standard errors adjusted using the *mi estimate* procedure in Stata 13. Sensitivity analyses also examined results from participants with only complete data, and there were no major differences from results obtained using imputed data. Missing values for CRP were not imputed for regression analyses.

## Results

Characteristics of the sample of 1,206 mothers of newborns included in the present analyses are provided in Table 1. Women in the sample identified themselves as African American/Black (54%), Latino/Hispanic (23%), and non-Hispanic White (23%). Participants had completed an average of 13 years of education, and 60% of the sample had a high school degree or less. The median per capita household income was $6,599, and 70% of the sample had household incomes near or below the federal poverty level ($21,954 for a family of 4 in 2009). These figures diverge from many other studies of pregnant/postpartum women in which samples are typically predominantly White, highly educated and less likely to be poor.

Table 2 provides descriptive statistics for CRP levels at Time 2 and Time 3. CRP levels ranged from <.1 mg/L to over 36 mg/L and there was marked positive skew in the distributions at T2 and T3. Prior to analyses, values of CRP were natural-log transformed to normalize the distributions.

To further characterize levels of inflammation in the current sample, CRP was categorized according to the CDC/American Heart Association criteria for cardiovascular disease risk (51). The percentage of participants in each CRP category were as follows at T2 and T3, respectively: 53% and 54% had CRP of less than 3 mg/L; 39% and 35% were between 3 to 10 mg/L; and 8% and 12% had CRP levels of 10 or higher mg/L. Forty-eight percent of the sample showed evidence of elevated CRP at T2 and 46.4% were elevated at T3. Among the 1,112 participants in the sample who had CRP measurements at both T2 and T3, which were six months apart, there was a moderate correlation ($r = .67$, $p < .001$). There was no

statistically significant overall change in CRP from T2 to T3 ($M$ = .03, paired $t$ = 0.88, $p$ = .38). In terms of categorical outcomes, 35% had high CRP ( ≥ 3 mg/L) at both T2 and T3, 40% of participants had consistently "normal" CRP (< 3 mg/L) at both T2 and T3, and 25% had high CRP at just one of the two study visits.

Epidemiological studies of systemic inflammation have conventionally excluded individuals with CRP values over 10 mg/L in the analysis stage under the assumption that these high values reflect acute inflammation due to recent infection or injury (52). However, recent evidence suggests that CRP is clinically useful in predicting CVD risk across a full range of values (53,54) and patterns in the current sample also suggest that very high CRP values may be indicative of chronic rather than acute inflammation. Of the 97 women who had CRP values ≥ 10 mg/L at T2, 81% also had elevated CRP (≥ 3 mg/L) at T3. Thus, applying conservative criteria and excluding participants with CRP values ≥ 10 mg/L could lead to the loss of meaningful variance in the outcome variable. Therefore, sample-specific criteria were used to classify and exclude outliers. For preliminary descriptive analyses, CRP values that were more than three standard deviations from the sample mean were excluded. There were 8 individuals with T2 CRP values that were greater than three standard deviations above the mean (15.5 to 36.6 mg/L) and 11 individuals with T3 CRP values that were more than 3 standard deviations above the mean (CRP > 17.23 to 36.4 mg/L). These participants were excluded from analyses.

Table 2 displays bivariate correlations of T2 and T3 CRP and BMI with chronic stress variables. There were significant positive correlations between financial stress and CRP at T2 and T3. Neighborhood stress, family relationship stress, co-parenting stress, partner relationship stress, total life stress, discrimination, interpersonal violence, and perceived stress were not associated with continuous CRP values at either time point. In regression models, no significant associations of these stress variables with T2 or T3 CRP emerged after statistical adjustment for covariates (all $p$'s > .05). Financial stress was the only one of the nine stress variables that showed a significant unadjusted association with T2 and T3 CRP.

Table 3 displays results for the unadjusted and adjusted regression models using financial stress to predict T2 and T3 CRP. Financial stress remained significantly associated with T2 and T3 CRP after adjusting for race/ethnicity, income, education and age in Model 2 and health-related covariates in Model 3. Further adjustment for BMI attenuated the coefficient to non-significance at T2 and T3. In the final model predicting T2 CRP, Latino ethnicity, cardiovascular risk conditions, higher BMI, and use of birth control were associated with higher CRP and smoking was associated with lower CRP. In the final model predicting T3 CRP, residence in eastern North Carolina, higher BMI, recent illness and use of birth control were associated with higher CRP and African American race, breastfeeding and smoking were associated with lower CRP.

Because of the strong relationship between BMI and CRP that emerged in our regression analyses, we conducted a series of analyses to further probe the associations between these variables over time and test whether changes in CRP tracked with changes in BMI. First, we tested the correlation between change in BMI and change in CRP from T2 to T3. This

correlation was not significant, $r$ (1074) = 0.04, $p$ = .20. We also tested whether change in BMI from T2 to T3 was associated with T3 CRP, and this correlation was not significant, $r$ (1074) = 0.01, p =.70. To further probe for any association, we divided participants into three groups: those who had lost at least 5 percent of their body weight between T2 and T3 ($n$ = 155), those with relatively stable weight ($n$ = 810) and those who had weight increases of 5% or more ($n$ = 130). We found no significant differences in T3 CRP or CRP changes from T2 to T3 depending on whether participants had gained weight, lost weight, or remained roughly the same weight.

## Discussion

The present study examined associations of several chronic stressors with CRP, which is an acute phase protein that serves as a useful marker of low-grade systemic inflammation. We expected that higher levels of chronic stress would be associated with higher levels of CRP. Overall, results did not provide strong evidence for these hypothesized associations, although financial stress emerged as a significant predictor of CRP at 6 months and 1 year postpartum.

This study is the first to report detailed descriptive information about levels of CRP at two time points during the year after a birth of a child in a diverse sample of predominately low SES women from five regions of the U.S. We found that a striking number of participants had clinically elevated levels of CRP (47 % at 6 months and 46% at 12 months postpartum). Also notable is the high frequency (35%) of consistent CRP values at both T2 and T3 that exceeded the clinical cutpoint of 3 mg/L. Systemic inflammation may also be related to a recent pregnancy and childbirth, given that this period involves a number of pronounced shifts in immune system function and changes in distribution of body fat (33,37,55). Because little is known about the normal range of CRP during the first postpartum year, it is unclear whether the apparent high rates of elevated CRP in this sample are fairly typical for women in the year following birth of a child or reflective of a particularly at-risk sample though we suspect the latter.

Several other potentially important descriptive findings emerged in examining characteristics of the study sample. First, there were several indicators of poor health status in these women, including diagnoses of chronic health conditions or problems and high rates of complications in the recent pregnancy. Second, a number of behavioral risk factors were present including breastfeeding for less than 6 months (79%), low physical activity (36%), past or current smoking (35%), excessive alcohol use (10%), and inadequate sleep (48%). Third, a majority of participants were either overweight (26%) or obese (43%). Each of these descriptive findings suggests adverse health conditions in the sample and portends of heightened risk of future health problems for these women. In particular, higher BMI and breastfeeding for less than 6 months were associated with higher levels of CRP at 6 months and 1 year postpartum. Postpartum BMI can be thought of as an aggregate of three different risk factors: 1) overweight/obesity and accompanying metabolic dysregulation prior to pregnancy, 2) excessive gestational weight gain, and 3) postpartum weight retention, in some cases due to lack of breastfeeding. Prior work in this sample showed that over 75% of women were heavier at 1 year postpartum than they were before becoming pregnant, and

nearly half retained more than 10 lbs (26). Future work may address whether controlling weight gain during pregnancy and promoting breastfeeding facilitate returning to a healthy weight and reduce inflammation after birth.

In contrast with our expectations, eight of the nine stress variables included in this study were not associated with higher levels of CRP at T2 or T3. When each of these chronic stressors was examined, only financial stress emerged as a significant predictor of T2 CRP and T3 CRP. Notably, we also tested for interactions between race/ethnicity and each of the additional stress variables examined as predictors of T2 and T3 CRP and none were significant (all *p*'s >.15; results not shown), indicating that relationships between the stress variables and CRP did not vary by race/ethnicity.

How is financial stress different from other domains of chronic stress, and why was this type of stress uniquely associated with CRP? Because it touches on concerns related to the most fundamental resources necessary for survival, financial stress may also be more likely than other forms of chronic stress to elicit exaggerated stress responses, producing physiological dysregulation, and ultimately, poorer health outcomes. Moreover, worries about food, shelter, transportation, and other basic necessities may be especially distressing after the birth of a child, and these persistent negative thoughts may contribute to prolonged stress responses among financially strained women. Additional research is needed to better understand why financial stress as measured here has a stronger association with biological markers of disease risk than other forms of chronic stress. Multivariate analyses indicated that financial stress positively predicted CRP at T2 and T3, and this relationship was not accounted for by race/ethnicity, income, education, parity, health behaviors, or health conditions. However, it was notable that results were attenuated to non-significance when BMI was included in the regression model. Consistent with evidence that visceral adipose tissue serves as a source of pro-inflammatory cytokines, BMI was highly correlated with CRP at both time points, and was also associated with financial stress. This pattern of findings suggests that financial stress does not affect CRP levels through direct immodulation of immune functioning, but rather indirectly through increased adiposity.

These results contribute to a growing body of literature demonstrating associations of financial strain with adverse health outcomes including elevated blood pressure, allostatic load, and risk of cardiac events (56–58). To our knowledge, this is the first study linking financial stress to increased adiposity and CRP in women during the first year postpartum though previous studies have also linked financial stress and increased weight (59,60). We note that household income and education were not significantly associated with CRP in this sample, though prior studies have shown links between lower SES and higher levels of inflammatory markers (19–21,61). One possible explanation for this discrepancy is the limited number of participants at the higher end of the SES spectrum, which may have reduced our ability to detect an association. In addition, the financial stress measure used in this study captures worries about finances, which are typically conceptualized as more proximal to physiological stress responses than stress exposures. Financial stress was negatively correlated with income in this sample as we would expect, but there was a good deal of variability at different income levels in financial stress which is likely due to individual differences in stress appraisals that are a function in part of resources for coping

with stress. For example, even among the participants in the full CCHN cohort who were living below the poverty level, only 30% reported that money worries were moderately or severely stressful during their pregnancies and less than 30% reported that they had serious problems with money during the previous year. These somewhat surprising findings may be attributable at least in part to the high levels of resilience resources reported by participants such as high optimism, mastery, self-esteem, and perceived support in this sample. Regardless of the reason, the results suggest that having a low income must be appraised through the lens of a panoply of personal, social and other resources in order to determine effects on physiology and health outcomes (42,62).

Several limitations deserve consideration. First, the measure of financial stress included in this study is based on various existing measures but is not standardized. However, it was designed through community-based participatory methods and showed evidence of acceptable psychometric properties, including acceptable internal consistency and evidence of good fit in a confirmatory factor analysis. The timing of the study was coincident with a major economic recession in the U.S., and thus examining financial stress among participants was particularly important to our research network. Second, because of the observational nature of the data, causal relationships between study variables cannot be inferred. For example, it is possible, though perhaps less likely, that high levels of inflammation related to chronic illness contribute to financial stress by limiting employment opportunities or generating additional healthcare expenses. Third variables like poverty could contribute to both financial strain and CRP but those were included in our regression models. Ideally, future studies should assess financial stress, CRP, and other indicators at multiple time points to explore the directionality of effects. Finally, additional issues may have reduced our ability to detect a significant association between chronic stress and inflammation; for example, participants with current infections or injuries were not excluded from blood sampling, though we attempted to adjust for recent illness as a covariate. Nonetheless the extent of data collection on infectious illness was limited to minimal self-report.

In addition to limitations, this study had several methodological strengths. First, this study also included a diverse sample of participants from populations that are often underrepresented in research on stress and health, including a large proportion of very low SES individuals and women who identified as African American/Black or Hispanic/Latina. Second, the longitudinal design allowed for multiple assessments over the course of the first year postpartum including two measures of CRP 6 months apart for a relatively large group of women. Third, the use of community-based participatory research methods led to the inclusion of a multiple carefully designed chronic stress measures which were tailored to the study population and rare in stress research. In addition, the use of the innovative CCHN LSI provided a more objective measure of chronic stress in the domains of neighborhood, family relationships, partner relationship, and co-parenting. In contrast, financial stress was assessed directly from participants and reflected perceptions or worries, rather than being inferred from conditions such as low income which was measured and tested too.

In conclusion, our findings suggest that financial stress stemming from socioeconomic disadvantage may be a particularly deleterious form of stress with respect to physical health

and specifically systemic inflammation. It is difficult to propose practical intervention strategies in light of the fact that financial strain among families living in poverty stems from larger societal issues such as income inequality and inadequate allocation of resources to economic assistance programs, though social programs providing financial assistance or job placement for families with young children may ameliorate the problem. As has been suggested elsewhere, policy changes that target these fundamental social and economic problems may improve the health of the population by reducing the prevalence of financial strain and its associated long-range health ramifications.

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## Glossary

| | |
|---|---|
| **BMI** | body mass index |
| **CCHN** | Community Child Health Network |
| **CRP** | C-reactive protein |
| **LSI** | Life Stress Interview |
| **PSS** | Perceived Stress Scale |
| **SES** | socioeconomic status |

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**Table 1.**

*Sample Characteristics* (*n* = 1,206)

| **Categorical Variables** | ***n*** | **(%)** | | |
|---|---|---|---|---|
| Race/ethnicity | | | | |
| African American/Black | 651 | (54.0) | | |
| White/Caucasian | 279 | (23.1) | | |
| Hispanic/Latina | 276 | (22.9) | | |
| Per capita household income quintiles | | | | |
| Q1 ($0–2,083) | 244 | (20.2) | | |
| Q2 ($2,100–4,875) | 236 | (19.6) | | |
| Q3 ($5,167–9,375) | 245 | (20.3) | | |
| Q4 ($9,500–20,833) | 240 | (19.9) | | |
| Q5 ($21,250–500,000) | 241 | (20.0) | | |
| Site | | | | |
| Baltimore | 259 | (21.5) | | |
| Lake County, IL | 317 | (26.3) | | |
| Los Angeles County | 127 | (10.5) | | |
| Eastern North Carolina | 243 | (20.2) | | |
| Washington, DC | 260 | (21.6) | | |
| Multiparity | 616 | (54.5) | | |
| Current smoker | 223 | (18.5) | | |
| Breastfed ≥ 6 mos. | 254 | (21.1) | | |
| Excessive drinker | 144 | (10.2) | | |
| T2 Recent Illness | 153 | (13.2) | | |
| T3 Recent Illness | 173 | (15.0) | | |
| Medication use (% yes) | | | | |
| Hormonal Birth Control | 458 | (38.5) | | |
| Antihypertensives | 29 | (2.4) | | |
| Antidepressants | 49 | (4.1) | | |
| Steroids | 44 | (3.7) | | |
| NSAIDS | 27 | (2.2) | | |
| Moderately/highly active | 764 | (64.4) | | |
| **Continuous variables** | ***M*** | **(*SD*)** | **Range** | |
| Education (years) | 12.98 | (2.78) | 4 | 23 |
| Age (years) | 25.82 | (5.74) | 18.01 | 41.69 |
| Sleep (average hours) | 6.67 | (1.25) | 5 | 9 |
| Number of CV health conditions | 0.40 | (0.64) | 0 | 4 |
| Time 2 CRP (mg/L) | 4.06 | (3.82) | 0.10 | 36.60 |
| Time 3 CRP (mg/L) | 4.27 | (4.32) | 0.10 | 36.40 |
| Time 2 BMI | 30.01 | (8.18) | 13.61 | 61.50 |
| Time 3 BMI | 29.89 | (8.12) | 15.57 | 58.20 |

*Note*. NSAIDS = non-steroidal anti-inflammatories; CV= cardiovascular; CRP = C-reactive protein; BMI = Body Mass Index

**Table 2.**

Correlations of T2 and T3 CRP and BMI with Stress Measures

| | | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) | (11) | (12) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| (1) | T2 CRP (log) | 1 | | | | | | | | | | | |
| (2) | T3 CRP (log) | .67*** | 1 | | | | | | | | | | |
| (3) | T2 BMI | .52*** | .50*** | 1 | | | | | | | | | |
| (4) | T3 BMI | .51*** | .49*** | .97*** | 1 | | | | | | | | |
| (5) | Financial stress | .10*** | .09** | .11*** | .10*** | 1 | | | | | | | |
| (6) | Total Life Stress | .03 | .02 | .09** | .10** | .26*** | 1 | | | | | | |
| (7) | Neighborhood Stress | .02 | .02 | .05 | .02 | .21*** | .57*** | 1 | | | | | |
| (8) | Family Stress | .05 | .05 | .09** | .11*** | .24*** | .64*** | .32*** | 1 | | | | |
| (9) | Co-Parenting Stress | .01 | .00 | .05 | .06* | .18*** | .75*** | .18*** | .27*** | 1 | | | |
| (10) | Partner Relationship | .03 | .03 | .04 | .04 | .16*** | .70*** | .26*** | .41*** | .39*** | 1 | | |
| (11) | Perceived Stress | −.02 | −.03 | .01 | .03 | .42*** | .40*** | .26*** | .34*** | .26*** | .30*** | 1 | |
| (12) | Discrimination | .00 | −.02 | .04 | .06 | .21*** | .13*** | .15*** | .09*** | .06* | .08*** | .14*** | 1 |
| (13) | Interpersonal Violence | .06* | .03 | .03 | .03 | .23*** | .24*** | .17*** | .19*** | .12*** | .28*** | .30*** | .11*** |

*Notes*. Pearson *r* values shown. CRP = C-reactive Protein; BMI = Body Mass Index

* $p < 0.05$;

** $p < 0.01$;

*** $p < .001$

**Table 3.**

Standardized Regression Coefficients of Multivariate Regressions Predicting T2 and T3 Log CRP

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|---|---|---|---|---|---|---|---|---|
| **Variables** | **T2** | **T3** | **T2** | **T3** | **T2** | **T3** | **T2** | **T3** |
| Financial stress | 0.10** | 0.09** | 0.08** | 0.09** | 0.09** | 0.08** | 0.04 | 0.04 |
| Race/ethnicity | | | | | | | | |
| African American race | | | 0.12** | 0.06 | 0.06 | 0.02 | −0.02 | −0.08* |
| Latino ethnicity | | | 0.12** | 0.08 | 0.09 | 0.08 | 0.08* | 0.07 |
| Household Income (Quintiles) | | | | | | | | |
| Q2 | | | 0.04 | 0.06 | 0.04 | 0.05 | 0.01 | 0.03 |
| Q3 | | | 0.04 | 0.05 | 0.04 | 0.04 | 0.04 | 0.04 |
| Q4 | | | 0.04 | 0.04 | 0.03 | 0.03 | −0.02 | 0.03 |
| Q5 | | | −0.02 | 0.01 | −0.04 | −0.01 | −0.01 | 0.04 |
| Education | | | −0.03 | −0.03 | −0.04 | −0.03 | 0.01 | 0.02 |
| Study Site | | | | | | | | |
| Lake County, IL | | | 0.03 | 0.05 | 0.01 | 0.02 | 0.03 | 0.05 |
| Los Angeles | | | −0.05 | −0.01 | −0.05 | −0.01 | −0.02 | 0.03 |
| North Carolina | | | 0.07 | 0.11** | 0.04 | 0.09* | 0.02 | 0.06* |
| Washington, DC | | | −0.08* | −0.03 | −0.09* | −0.03 | −0.06 | 0.01 |
| Age | | | 0.13** | 0.05 | 0.11** | 0.05 | 0.04 | −0.03 |
| Multiparity | | | −0.06 | −0.07* | −0.06 | −0.07* | −0.02 | −0.03 |
| Moderately/Highly active | | | | | −0.04 | −0.00 | −0.04 | −0.01 |
| Sleep (avg. hours) | | | | | −0.03 | −0.03 | −0.03 | −0.03 |
| Current smoker | | | | | −0.10** | −0.07* | −0.09** | −0.07* |
| Breastfed > 6 mos. | | | | | −0.06* | −0.12** | −0.01 | −0.10** |
| Excessive drinker | | | | | 0.01 | −0.00 | 0.00 | −0.00 |
| Recent Illness | | | | | 0.01 | 0.11*** | 0.02 | 0.12*** |
| Cardiovascular conditions | | | | | 0.14** | 0.09*** | 0.06* | 0.03 |

| | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
|---|---|---|---|---|---|---|---|---|
| **Variables** | **T2** | **T3** | **T2** | **T3** | **T2** | **T3** | **T2** | **T3** |
| Medications (1 = yes) | | | | | | | | |
| Hormonal Birth Control | | | | | 0.02 | 0.02 | 0.06* | 0.06* |
| Antihypertensives | | | | | 0.03 | 0.05 | 0.02 | 0.04 |
| Antidepressants | | | | | −0.01 | 0.02 | −0.01 | −0.01 |
| Steroids | | | | | 0.01 | −0.01 | −0.01 | −0.01 |
| NSAIDS | | | | | −0.02 | 0.00 | −0.04 | −0.01 |
| Current BMI | | | | | | | 0.51*** | 0.53*** |
| *Adjusted* $R^2$ | 0.01 | 0.01 | 0.03 | 0.02 | 0.08 | 0.06 | 0.29 | 0.31 |

* $p<0.05$;

** $p<0.01$