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Physical Activity, Chronic Stress, and Inflammation in Mothers during the First Postpartum Year

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by

Christine Marie Guardino

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ABSTRACT OF THE DISSERTATION

Physical Activity, Chronic Stress, and Inflammation in Mothers during the First Postpartum Year

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Chronic stress predicts a number of adverse health outcomes, including depression, cardiovascular disease, and all-cause mortality. In the context of women's reproductive health, chronic stress exposure has been associated with increased risk of adverse birth outcomes and postpartum depression. Systemic inflammation resulting from immune system dysregulation is one of the pathways through which chronic stress is thought to influence physical health outcomes. Increasingly strong evidence suggests that engaging in regular physical activity protects individuals from the health-damaging effects of stress, possibly through alterations of biological stress responses. The present research pursued two main objectives using data from a prospective investigation conducted by academic researchers and community members at five collaborating sites across the U.S. These objectives were: 1) describing the levels and correlates of physical activity at six months postpartum in mothers of three ethnic/racial groups and different levels of SES including a large proportion who are poor and; 2) developing and testing multivariate models of the combined influence of chronic stress, physical activity, and other

resilience resources on C-reactive protein (CRP), a marker of systemic inflammation that is associated with elevated risk of chronic disease.

Results indicated that African American race, Latina ethnicity, and living in a rural area were generally associated with lower levels of physical activity, whereas working outside the home was associated with high activity. In analyses predicting CRP, women with higher Financial Stress one month after the birth of child had higher CRP at both six months and one year postpartum. Notably, these subjective appraisals of Financial Stress were stronger predictors of inflammation than more objective resource indicators such as income and education. Additional analyses using structural equation modeling demonstrated that this relationship was mediated by adiposity. There was no evidence that behavioral or psychological resilience resources moderated the associations between chronic stressors and CRP. These findings advance existing knowledge by identifying correlates and predictors of important health outcomes during the first postpartum year.

The dissertation of Christine Marie Guardino is approved.

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Dedicated to my husband James with love and gratitude. Without your patient support, I would have given up long ago. This achievement belongs to us both.

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Chapter One: Introduction

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 (Dunkel Schetter & Dolbier, 2011). Strong empirical evidence links high levels of chronic stress with poorer health outcomes including increased risk of cardiovascular disease (Krantz & McCeney, 2002; Rozanski, Blumenthal, & Kaplan, 1999), depression (Hammen, 2005; Kessler, 1997) and all-cause mortality (Nielsen, Kristensen, Schnohr, & Grønbæk, 2008; Schulz & Beach, 1999). The pathways through which chronic stress and other psychosocial variables influence disease processes are complex and not fully understood, but systemic inflammation resulting from immune system dysregulation may play an important role (Steptoe, Hamer, & Chida, 2007; Vaccarino et al., 2007; Whooley et al., 2007). In contrast to the health-damaging effects of chronic stress, habitual physical activity has been associated with a number of health benefits, including reduced risk of cardiovascular disease (Oguma & Shinoda-Tagawa, 2004) and Type II diabetes (Bassuk & Manson, 2005; Jeon, Lokken, Hu, & Van Dam, 2007; Penedo & Dahn, 2005). Current understanding of the mechanisms underlying the health benefits of physical activity is also incomplete, but accumulating evidence suggests that inflammatory processes may play a role in these effects as well (Kasapis & Thompson, 2005; Pischon, Hankinson, Hotamisligil, Rifai, & Rimm, 2012).

Both high levels of chronic stress and low levels of physical activity have been associated with adverse health effects, but there is a need for multivariate models of the combined influence of chronic stress, physical activity and other resilience resource on biological markers of disease risk. Some studies have explored physical activity as a *mediator* of the effects of stress on health, and shown that the lower levels of physical activity among highly stressed individuals partially

accounts for the relationship between stress and adverse health outcomes (e.g. Hamer, Molloy, & Stamatakis, 2008; Kershaw, Mezuk, Abdou, Rafferty, & Jackson, 2010). Other studies have examined physical activity as a *moderator* of the effects of stress on health, and suggest that physical activity may prevent or ameliorate stress-related physiological damage and thus offer even greater benefits for individuals who experience high levels of stress (e.g. Krueger & Chang, 2008; Puterman, Adler, Matthews, & Epel, 2012; Rueggeberg, Wrosch, & Miller, 2012). Moreover, some evidence suggests that the stress-moderating effects of physical activity are explained by more adaptive physiological stress responses in fit individuals (Forcier et al., 2006; Hamer, Taylor, & Steptoe, 2006; Jackson & Dishman, 2006) while other studies have shown that physical activity is associated with personal resilience resources that enable individuals to better manage the stress in their lives (DiLorenzo et al., 1999; Moses, Steptoe, Mathews, & Edwards, 1989; Steptoe, Edwards, Moses, & Mathews, 1989). Thus, there is a need for a better understanding of the combined influence of chronic stress, physical activity, and other resilience resources on physical health outcomes.

Review of the Literature

This review has several sections designed to provide a context for the present research. First, it provides a brief summary of the larger literature relating chronic stress to health outcomes and inflammation in the general population followed by more detail regarding the specific studies that have been conducted during the postpartum period. This section is followed by an overview of the correlates and sequelae of physical activity, with consideration of some of the methodological issues involved in this line of inquiry. Finally, this review describes the theoretical rationale and existing empirical support, including evidence from animal models as well as human laboratory and observational studies, for the stress-moderating effects of physical

activity on physiology and health outcomes. Because an exhaustive review of each of these literatures is beyond the scope of this review, the most methodologically rigorous studies (e.g. longitudinal studies with nationally representative samples, randomized controlled trials) and those of greatest relevance to this study of postpartum women are reviewed in greatest detail. The reader is referred in the following sections to the most recent and comprehensive relevant reviews for additional evidence.

Linking Chronic Stress and Health

Stress is commonly defined as external demands appraised as exceeding an organism's ability to cope (Lazarus & Folkman, 1984). This general definition encompasses both acute and chronic stressors. Acute stressors include time-limited events with a discrete beginning and ending, such as death of a loved one, divorce, and physical assault, whereas chronic stressors include 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 (Dunkel Schetter & Dolbier, 2011). Strong epidemiological evidence links chronic stressors such as unemployment, poverty, discrimination, difficult interpersonal relationships, and caregiving to increased risk of cardiovascular disease (Krantz & McCeney, 2002; Rozanski et al., 1999), depression (Hammen, 2005; Kessler, 1997) and all-cause mortality (Nielsen et al., 2008; Schulz & Beach, 1999). For example, the Whitehall II longitudinal study of 10,308 British civil servants has been critical in establishing a relationship between job strain and adverse health outcomes. The study findings show prospective relationships between high job strain and increased risk of cardiovascular disease, metabolic syndrome, and psychiatric disorders (Chandola, Brunner, & Marmot, 2006; Marmot, Bosma, Hemingway, Brunner, & Stansfeld, 1997; Stansfeld, Fuhrer,

Head, & Ferrie, 1997). Given the strong evidence that chronic stress is harmful to health, the next step is to determine how stress affects physiological processes relevant to disease.

Effects of stress on inflammation. One of the 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 (Steptoe et al., 2007; Vaccarino et al., 2007; Whooley et al., 2007). Chronic low-grade inflammation is a term for conditions characterized by high levels of pro-inflammatory cytokines (such as IL-6, TNF-α, IL-1, IL-1ra, sTNF-R) and C-reactive protein (CRP). CRP is an acute phase reactant synthesized in the liver, and levels are associated with risk of myocardial infarction and stroke, and the development of chronic diseases including cardiovascular disease and Type II diabetes (Pradhan, Manson, Rifai, Buring, & Ridker, 2001; Ridker, Rifai, Rose, Buring, & Cook, 2002).

A significant relationship between psychological stress and inflammation has been observed in a number of investigations spanning a range of methodologies. Experimental evidence for the inflammatory effects of stress comes from controlled laboratory studies showing that acute stress exposure elicits significant increases in circulating levels of pro-inflammatory cytokines including TNF-α, IL-6, and Il-1β (Dickerson, Gable, Irwin, Aziz, & Kemeny, 2009; Steptoe et al., 2007). 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 (Emeny et al., 2012), discrimination (Friedman, Williams, Singer, & Ryff, 2009), and early life stress (Taylor, Lehman, Kiefe, & Seeman, 2006). For example, a recent cross-sectional study of 53 caregivers and 77 non-caregiving controls found that individuals who experience multiple stressors on a daily basis have higher baseline levels of serum IL-6 and CRP (Gouin, Glaser, Malarkey, Beversdorf, & Kiecolt-Glaser, 2012). Other

studies have documented associations between exposure to interpersonal stress and elevated inflammatory activity (Chiang, Eisenberger, Seeman, & Taylor, 2012; Fuligni et al., 2009; Kiecolt-Glaser et al., 2005).

In addition, levels of CRP also tend to be higher among demographic groups that experience the highest levels of chronic stress. On average women have higher levels of CRP than men (Beasley et al., 2009; Khera et al., 2005; Lakoski et al., 2006); Whites have lower CRP levels than Blacks or Hispanics (Beasley et al., 2009; Gruenewald, Cohen, Matthews, Tracy, & Seeman, 2009; McDade, Lindau, & Wroblewski, 2011; Nazmi & Victora, 2007); and elevated CRP is more prevalent in low SES individuals (Alley et al., 2006; Brummett et al., 2013; Friedman & Herd, 2010; Gallo et al., 2012; Gruenewald et al., 2009; Kershaw, Mezuk, Abdou, Rafferty, & Jackson, 2010; Loucks et al., 2006; McDade, Hawkley, & Cacioppo, 2006; Nazmi & Victora, 2007; Ranjit et al., 2007).

The mechanisms through which stress exposure contributes to systemic inflammation and related co-morbidities are not yet clear. Miller, Cohen, and Ritchey (2002) have proposed the glucocorticoid resistance model as a means of understanding how chronic stress leads to increased risk of inflammatory diseases. This model posits that chronic stress impairs the immune system's sensitivity to glucocorticoid hormones that impede inflammatory processes under normal conditions. Under chronically stressful conditions that elicit elevated activity of the hypothalamic-pituitary-adrenal (HPA) and sympathetic adrenal medullary (SAM) axes, white blood cells are continually exposed to high concentrations of stress hormones and mount a counter-regulatory response characterized by downregulation of glucocorticoid-binding receptors. The downregulation of the expression and/or function of these receptors impairs the immune system's response to the anti-inflammatory effects of cortisol. Thus, the development of

glucocorticoid resistance allows inflammation and related disease-processes to flourish unchecked. Miller and colleagues have found evidence to support the glucocorticoid resistance model in studies of individuals caring for a spouse with cancer (Miller et al., 2008), parents of children with cancer (Miller et al., 2002) and among individuals who reported a recent stressful life experience associated with long-term threat (Cohen et al., 2012).

Stress and inflammation after birth. While stress has been linked to poor immunological 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 (Coussons-Read, Okun, & Simms, 2003) or during the post partum (Yim, Tanner Stapleton, Guardino, Hahn-Holbrook, & Dunkel Schetter, 2014). A number of normal changes in the immune system and inflammatory processes occur during pregnancy and postpartum. Throughout the lifespan, the primary function of the immune system is to defend the body against pathogens by identifying and destroying foreign proteins. In order to perform this function, the immune system must distinguish between "self" and "non-self." During the gestational period, the maternal immune system must adapt to accommodate the fetus which shares half of its genomic complement with the father while also continuing to defend the mother against pathogens. This unique immunological situation has led some researchers to describe normal pregnancy as an "immunological balancing act" (Wadhwa, Culhane, Rauh, & Barve, 2001). Over the course of pregnancy, the ability of lymphocytes to proliferate in response to mitogenic stimuli declines, reflecting general immune suppression, and the predominant immune responses shift from inflammatory, cell-mediated responses to anti-inflammatory, humoral responses (Gehrz et al., 1981; Lin, Mosmann, Guilbert, Tuntipopipat, & Wegmann, 1993). The shift from Th1 to Th2 immunity that occurs during pregnancy is reflected in shifting maternal

cytokine levels; pro-inflammatory cytokines (including IL-6, IL-8, and TNF-α) tend to decrease during pregnancy, while levels of the anti-inflammatory cytokines IL-3, GN-CSF, TGF-β, IL-4, and IL-10 tend to increase (Gennaro & Hennessy, 2003). Within hours of the delivery of the infant and placenta, the anti-inflammatory milieu of pregnancy abruptly shifts to a pro-inflammatory state (Corwin, Bozoky, Pugh, & Johnston, 2003; Hebisch, Neumaier-Wagner, Huch, & von Mandach, 2004; Oestensen et al., 2005; Vassiliadis, Ranella, Papadimitriou, Makrygiannakis, & Athanassakis, 1998).

During pregnancy, higher levels of maternal stress during the second trimester have been associated with elevated serum CRP throughout pregnancy (Coussons-Read et al., 2007). High levels of maternal stress during pregnancy have also been shown to alter levels of circulating cytokines and contribute to increased maternal susceptibility to infection (Coussons-Read, Okun, Schmitt, & Giese, 2005; Ruiz, Fullerton, & Dudley, 2003). In turn, elevated levels of pro-inflammatory cytokines and infection may play a role in pregnancy complications such as the development of preeclampsia, fetal distress and premature birth (Coussons-Read, Okun, & Nettles, 2007; Munno et al., 1999). In one particularly strong study of 173 women, elevated pregnancy-specific distress, IL-6, and TNF-α were associated with shortened gestational age at birth and there was some evidence that circulating inflammatory markers mediated the stress-gestational age relationship (Coussons-Read et al., 2012).

Immune processes may also play a role in postpartum maternal mental health. Although CRP measured within the first two postnatal days was not associated with PPD in a study of more than 1,000 women (Albacar et al., 2010), a small study of 27 women at high risk for developing PPD found a positive association between CRP and atypical depressive symptoms in the first five days after birth (Scrandis et al., 2008). Higher levels of pro-inflammatory cytokines

have also been associated with postpartum depressive symptoms (Maes et al., 2000) and some researchers have hypothesized that exaggerated or prolonged inflammatory response to labor and delivery may be implicated in the etiology of postpartum depression (Corwin & Pajer, 2008). Thus, understanding the psychosocial factors that influence immune system functioning during and after pregnancy may be particularly important but relatively little work has been done in this population to date. To our knowledge, only two published studies have examined CRP during the postpartum period, and neither of these studies examined associations of systemic inflammation with 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 nulliparous women or parous women who had given birth more than a year prior to the assessment (Kuzawa, Adair, Borja, & McDade, 2013). While CRP levels tended to be lower among postpartum women who were breastfeeding than among those who were not, this difference was not statistically significant. Another smaller study of 181 U.S. women at 4 to 6 weeks postpartum found higher levels of CRP in participants who had recently given birth in comparison to 33 control participants (Groer et al., 2005). Levels of CRP did not differ between women who were exclusively breastfeeding and women who were exclusively formula feeding. In sum, CRP is a potentially important marker of risk for future adverse health outcomes, but it has not been well explored in the context of the postpartum period. Thus, there is little available evidence to characterize normal ranges of CRP over the course of the first postpartum year, and demographic, medical, and behavioral correlates of this marker of systemic inflammation have not been established.

Sources of Stress Resistance and Resilience in Mothers

Because of the demands associated with caring for an infant, most new mothers are likely

to experience at least some degree of stress during the first year of their children's lives. Evidence described above suggests that these chronic demands are likely to have effects on inflammation and other disease processes if they exceed a woman's capacity to cope. However, the impact of stress on physical health varies between individuals and many women maintain good physical and mental health during the postpartum period even when faced with high levels of stress. Some of this variability may be due to individual differences in resilience resources that moderate the effects of stressors on various indicators of health and adjustment (Dunkel Schetter & Dolbier, 2011). Resilience resources can be defined as personal, social, or community level characteristics that foster the ability to cope and function well in the context of stress (Dunkel Schetter, 2011).

In examining resilience resources that moderate the biological consequences of stress, research has emphasized psychological and/or social resources such as personality, coping styles, self-esteem, mastery, and perceived support (Antonovksy, 1987; Carver, Scheier, & Segerstrom, 2010; Uchino, 2004). For example, social support is a much-studied personal resource for new mothers that may protect against postpartum depression (Beck, 2001; Hahn-Holbrook, Dunkel Schetter, Arora, & Hobel, 2013; O'Hara, 2009; Robertson, Grace, Wallington, & Stewart, 2004; Tanner Stapleton et al., 2012). Less attention has been paid to life style factors that influence stress responses though healthy behavioral practices and physical fitness were included in two recent comprehensive inventories of resilience resources (Dunkel Schetter & Dolbier, 2011; Zautra, Hall, & Murray, 2010). Moreover, increasingly strong evidence suggests that health behaviors including smoking, alcohol use, and physical activity show such moderating effects on relationship between stress and health (Jackson, Knight, & Rafferty, 2010; Kershaw et al., 2010; Krueger & Chang, 2008; Puterman et al., 2012; Puterman et al., 2010). These health behaviors

may influence behavioral and physiological responses to stress with implications for the cumulative effects of stress over time (McEwen & Seeman, 1999). Thus, positive health behaviors may serve as resilience resources that protect against the health-damaging effects of stress while unhealthy behaviors may exacerbate these effects, but physical activity has rarely been studied in this way.

Physical Activity: Definition and Public Health Recommendations

Physical activity is defined as "any bodily movement produced by skeletal muscles that results in energy expenditure" (Caspersen, Powell, & Christenson, 1985, p. 126) and includes leisure physical activity, transportation, childcare, occupational activity, yard work and household chores. Although the term *exercise* is often used as a synonym for physical activity, exercise is a more narrowly defined as physical activity that is planned, repetitive and purposive. Exercise is a structured, leisure-time type of physical activity, whereas physical activity also arises in domestic or occupational tasks. Finally, *physical fitness* differs from both physical activity and exercise and these terms are not interchangeable. Whereas physical activity is related to the movements that people perform, physical fitness is the attribute of being able to "carry out daily tasks with vigor and alertness, without undue fatigue and with ample energy to enjoy leisure-time pursuits and to meet unforeseen emergencies" (President's Council on Physical Fitness and Sports, 1971) and includes cardio-respiratory endurance, muscular endurance, muscular strength, body composition and flexibility.

The current Centers for Disease Control and Prevention and American College of Sports Medicine recommendations for physical activity advise all healthy adults to accumulate a minimum of 30 minutes of moderate-intensity aerobic physical activity on five days each week or a minimum of 20 minutes of vigorous-intensity aerobic physical activity on three days each

week (Haskell et al., 2007). Moderate-intensity aerobic activity noticeably increases heart rate and includes activities such as brisk walking, dancing, and leisurely swimming, while vigorous-intensity activities such as jogging cause rapid breathing and a substantial increase in heart rate. The guidelines state that these levels of physical activity are adequate to elicit substantial health benefits, and thus for research purposes these criteria are often applied to classify individuals as either physically active or inactive.

Physical activity guidelines for women during and after pregnancy are slightly different than those for the general population. The American College of Obstetricians and Gynecologists recommends that pregnant women who are not experiencing medical or obstetric complications adopt the recommendation of 30 minutes or more of moderate activity on most days of the week, though women who engage in vigorous activities such as running prior to becoming pregnant can usually continue those activities during their pregnancies (Artal & O'Toole, 2003). The recommendations also promote exercise for sedentary women and those with medical or obstetric complications, but only after medical evaluation and clearance. Because many of the physiological changes of pregnancy continue past the first month postpartum, a gradual return to activity is recommended after delivery.

Measurement of Physical Activity

Physical activity can be measured directly using motion detectors or indirectly using questionnaires, records, or logs, but is most frequently assessed through retrospective self-report measures due to their low cost and ease of use (Wilcox & Ainsworth, 2008). Validity correlations between questionnaires or interviews that require participants to recall their engagement in different activities over a given duration of time (such as the past week or month) with objective measures such as doubly labeled water, accelerometers or heart rate monitoring

range from $r = .14$ to $r = .36$ (Wilcox & Ainsworth, 2008). These low to moderate correlations indicate that self-reports of physical activity may not always be reliable or accurate. Accuracy may depend on the type of activity assessed; for example, higher intensity physical activities are more accurately reported than lower-intensity activities (Sallis et al., 1985; Sallis & Saelens, 2000).

Researchers must also consider population characteristics when measuring physical activity of women and ethnic minority samples (Mâsse et al., 1998). Many popular measures of physical activity are tailored toward men rather than women in that they ask about participation in sporting or vigorous activities rather than household and other chores, which may be a more common source of physical activity among women who are caring for their children and families (Ainsworth, Leon, Richardson, Jacobs, & Paffenbarger, 1993; Eyler et al., 1998). Because many self-report measures do not include unstructured, lower-intensity activities, physical activity levels of ethnic minority individuals and women are often underestimated (Kriska, 2000; Mâsse et al., 1998). Furthermore, some categories of physical activity that are often assessed on questionnaires may not be relevant or meaningful for all participants. For example, surveys measuring leisure-time physical activity may be problematic when used among low SES or minority women who do not have any leisure time in which to formally exercise, especially if they are working outside the home (Yeager, Macera, & Merritt, 1993).

It is also unclear whether or not different categories of physical activity should be combined when studying effects on health. CDC recommendations include short bouts of housework and walking toward daily quotas of physical activity but empirical evidence of the benefits of accumulated physical activity is inconsistent. Although activity that is performed as part of job or household responsibilities is likely to provide physical health benefits because it

engages the same cardiovascular and metabolic systems as recreational exercise, it may not result in the mental health benefits that are associated with leisure-time activity. As evidence, four population surveys in the United States and Canada showed that recreational physical activities but not household chores were associated with better psychological well being (Stephens, 1988). This point is well-illustrated by a quote from a participant in a qualitative study of physical activity in minority women conducted by Eyler et al. (1998):

> I'm a fast walker [at work] because I have to get where I'm going in a hurry. So I walk a lot, but it's not like being on a tread[mill] at the rec center or fitness center. It's totally different because what you can do during that time walking, not only are you working your body, but you're working with your mind at the same time. (p. 643)

Evidence from animal models also suggests that not all physical activity is equally beneficial. Forced treadmill running or swimming does not seem to have the same benefits as voluntary wheel running. In fact, forced treadmill running (but not voluntary wheel running) results in physiological adaptations associated with chronic stress, such as declines in thymic function, adrenal hypertrophy, elevated basal corticosterone, decreased corticosterone binding globulin, or immunosuppression (Moraska & Fleshner, 2001), which suggests that involuntary physical activity produces chronic mental stress. Thus, careful consideration should be taken in utilizing standardized definitions and measurements of physical activity.

Demographic and Psychosocial Correlates of Physical Activity

Demographic characteristics including non-White race, lower levels of education, and female gender are associated with a lower likelihood of meeting the 2008 Physical Activity Guidelines for Americans (Carlson, Fulton, Schoenborn, & Loustalot, 2010). Inequalities in the built environment may contribute to disparities in physical activity by constraining the

recreational facilities that are available to individuals who live in low-income neighborhoods (Gordon-Larsen, Nelson, Page, & Popkin, 2006). Environmental and neighborhood factors such as density, street connectivity and land-use mix have also been associated with increased walking and overall physical activity (Feng, Glass, Curriero, Stewart, & Schwartz, 2010; Gordon-Larsen et al., 2006; Handy, Boarnet, Ewing, & Killingsworth, 2002). For example, a recent analysis in a sample of 136,592 participants in the Nurses' Health Study and Nurses' Health Study II found that women living in denser counties with more accessible street designs were more active and had lower body mass indices (James et al., 2013).

Patterns of physical activity also vary across the lifespan. Life events, especially changes in employment status, physical health, relationship and family structure, have been associated with decreased physical activity among adults (Allender, Hutchinson, & Foster, 2008). Marriage and having children are likely to result in lower levels of physical activity among women (Brown, Heesch, & Miller, 2009). In one study of 1442 women, participants tended to reduce moderate and vigorous physical activity during the second trimester of pregnancy and those decreases persisted at 6 months postpartum (Pereira et al., 2007). Having other children in the home, working longer hours, postpartum weight retention and lack of childcare were associated with becoming insufficiently active during or after pregnancy.

Health Benefits of Physical Activity

A large body of research supports the health benefits of physical activity among people of all ages (Bauman, 2004; Berlin & Colditz, 1990; Fox, 1999; Sallis, Prochaska, & Taylor, 2000; Vogel et al., 2009; Warburton, Nicol, & Bredin, 2006). Physical activity has been associated with reduced risk and improvement of cardiovascular disease (Hagberg, Park, & Brown, 2000; Jolliffe et al., 2001; Manson et al., 1999; Thompson et al., 2003) and improvement in

management of diabetes mellitus (Boulé, Haddad, Kenny, Wells, & Sigal, 2001; Orchard et al., 2005). It has also been associated with improvements in markers of cardiovascular disease risk including decreases in LDL cholesterol, (Kelley & Kelley, 2006; Leon & Sanchez, 2001), decreases in blood pressure (Fagard, 2001; Hagberg et al., 2000) maintenance of weight loss (Wing & Hill, 2001).

In contrast to the large body of research documenting health benefits of physical activity in the general population, the health benefits of physical activity during and after pregnancy are less clear. There is some observational evidence physical activity during and after pregnancy provides medical and psychological benefits to women and their babies (Pivarnik et al., 2006) but few methodologically rigorous randomized controlled trials of physical activity have been conducted during pregnancy (Kramer & McDonald, 2006). The clearest evidence of benefits comes from randomized controlled trials demonstrating that physical activity during pregnancy confers a reduced risk of pre-eclampsia (Meher & Duley, 2006) and excessive gestational weight gain (Gardner, Wardle, Poston, & Croker, 2011; Streuling, Beyerlein, & von Kries, 2010). Physical activity may also be beneficial in preventing and treating postpartum depression (Armstrong & Edwards, 2003; Daley, Psychol, MacArthur, & Winter, 2007; Davis & Dimidjian, 2012) and there is some evidence that women who resume physical activity after pregnancy are less likely to retain excess weight (Ohlin & Rössner, 1990; Olson, Strawderman, Hinton, & Pearson, 2003).

Inflammation as a Mediator of the Effects of Physical Activity on Health

While the mechanisms underlying the health benefits of physical activity are not completely understood, inflammation may be an important mediator of these effects (Kasapis & Thompson, 2005; Pischon et al., 2012). Interestingly, the effects of a single bout of exercise on

immune activity mirror the effects of psychological stress on the immune system and in the short term exercise produces an inflammatory effect. The immediate immune reaction to exercise is an acute phase response characterized by lymphocyte proliferation and increased white blood cell counts. During acute exercise, IL-6 is released by exercising skeletal muscle but these cytokines (known as myokines because they are produced by skeletal muscle) are thought to ultimately promote an anti-inflammatory environment by increasing expression of IL-10 and IL-1 receptor antagonist and inhibiting pro-inflammatory TNF-α (Hamer et al., 2012; Pedersen, 2011). Acute increases in pro-inflammatory cytokines in response to a bout of strenuous exercise are mirrored by increased levels of CRP 24 to 48 hours after exercise (Kasapis & Thompson, 2005).

Although there is likely to be a transient increase in serum CRP following an acute bout of exercise, chronic physical activity has been associated with lower markers of systemic inflammation in cross-sectional and prospective studies. However, evidence is not entirely consistent and associations are weaker in randomized controlled trials. Hamer (2007) reviewed 18 observational studies that examined the association of CRP and other inflammatory factors with fitness/activity levels after statistically controlling for adiposity. There was entirely consistent evidence for higher levels of CRP in less active and unfit participants, though four studies found that the difference was no longer significant after controlling for body mass index (BMI) and smoking status. However, in the nationally representative NHANES III study of 13,748 U.S. adults, amount of leisure-time physical activity was inversely associated with concurrent CRP levels even after controlling for age, gender, ethnicity, education, occupation, smoking, hypertension, BMI, waist-hip ratio, HDL cholesterol, aspirin use, disease co-morbidities, and serum insulin (Ford, 2002). Recent evidence from the Whitehall II cohort provides additional epidemiological support for this relationship. Maintenance of habitual

physical activity for 10 years predicted lower levels of CRP and IL-6 at follow-up controlling for age, sex, smoking, employment grade, BMI, and chronic illness (Hamer et al., 2012).

A smaller number of studies have examined the prospective effects of exercise training on CRP. Hamer (2007) reviewed 10 randomized controlled trials that examined the effects of exercise and weight loss on inflammatory factors. The results of these studies are mixed, with six studies showing an inverse relationship between physical activity and CRP controlling for measures of adiposity and four showing no evidence of this relationship. Overall, the evidence from randomized controlled trials does not provide consistent evidence that exercise has effects on CRP independent of weight loss. Differences in the consistency of findings of observational studies as compared to intervention studies suggest the possibility that the relationship between physical activity and CRP demonstrated by observational studies could be is confounded by other variables.

Physical Activity as a Mediator of the Relationship between Stress and Health

Building upon evidence that both stress and physical inactivity are associated with poorer health outcomes, a number of studies have examined physical activity as a mediator of the relationship between stress and health outcomes. For example, Hamer and colleagues (2008) demonstrated that physical activity explained 22% of the association between psychological distress and CVD events over a 7-year follow-up period in a sample of 6,576 adults in the UK. In a sample of 9508 U.S. adults who participated in NHANES IV, Kershaw and colleagues (2010) found that lower levels of exercise partially mediated the associations of poverty and low educational attainment with elevated CRP. Thus, there is some convincing evidence that stress affects health indirectly through lower levels of physical activity. However, these meditational models do not account for the possibility that physical activity may modify the stress-health

relationship by altering physiological, cognitive, or behavioral responses to stress. The considerable body of literature described below supports this possibility.

Physical Activity as Moderator of the Relationship between Stress and Health

Physical activity and acute stress responses. A large body of work has examined the effects of physical activity/fitness on reactivity and recovery in response to acute laboratory stressors including mental arithmetic, Stroop color-word, and cold pressor tasks. This literature includes three different types of studies: (1) studies exploring effects of single bouts of activity on stress responses, (2) studies comparing stress responses of physically fit individuals to those of unfit individuals, and (3) experimental or quasi-experimental studies in which stress responses are compared before and after an extended period of exercise training. One meta-analysis looked specifically at 15 studies of the effects of an acute bout of exercise on blood pressure responses to laboratory stress tasks and found fairly consistent evidence for moderate attenuation of the blood pressure response after exercise (Hamer et al., 2006). Studies that have examined the effects of long-term physical activity on stress responses have produced more mixed results. A meta-analysis of 73 such studies reported that physical fitness was related to greater heart rate reactivity and better recovery in response to mental challenge but effect sizes were small, heterogeneous, and influenced by study quality (Jackson & Dishman, 2006). Effect sizes were generally larger in cross-sectional studies of fit vs. unfit individuals than in randomly controlled trials of exercise training. Effect sizes were also larger in studies that estimated fitness indirectly from submaximal heart rate response to moderate exercise than in those that assessed fitness directly via peak oxygen uptake, which is considered a more accurate measure of physical fitness. Forcier et al. (2006) conducted a more selective meta-analysis of the same literature that included only studies with evidence of an exercise training effect (i.e. fit and unfit participants

differed significantly on either baseline heart rate or VO_2 max). Results indicated that fit individuals showed significantly attenuated heart rate and systolic blood pressure reactivity and a trend towards attenuated diastolic blood pressure reactivity. Fit participants also showed faster heart rate recovery, but there were no significant differences for systolic or diastolic blood pressure recovery. In summary, there is limited evidence that exercise and fitness contribute to a more adaptive stress response pattern characterized by less reactivity and faster recovery to acute stress exposure. Thus, it is unclear whether physical activity moderates the effects of stress on health by contributing to more adaptive physiological stress response patterns.

Stress, physical activity, and self-reported health. An additional group of studies has examined physical activity as a moderator of the relationship between naturally occurring stressors and self-reported health outcomes, including illness symptoms and health complaints. A systematic review of this literature identified 27 cross-sectional and longitudinal studies conducted in adolescent or adult samples (Gerber & Pühse, 2009). About half of the studies fully or partially supported the hypothesis that physical activity acts as a moderator of the stress-illness association, while the other half did not. There was a great deal of variability across studies in terms of sample characteristics, physical activity/fitness assessment, and measurement of stress, which was sometimes assessed with measures of life events, perceived stress, or daily hassles, and in other studies operationalized as a specific stressor such as unemployment, job stress, or widowhood. However, the reviewers did not detect any patterns suggesting that any methodological characteristics were associated with a greater likelihood of finding significant effects in this set of studies. Moreover, the predominance of cross-sectional designs and use of self-report measures of health in these studies limited the conclusions that could be drawn. A recent longitudinal study provides more convincing evidence that physical activity interacts with

stress to influence self-reported health outcomes. Rueggeberg et al. (2012) found that older adults with high baseline levels of perceived stress who frequently engaged in physical activity reported a reduction of perceived stress over two years and fewer increases in physical health symptoms over 4 years compared to stressed inactive participants. No such associations were found in low-stress participants, suggesting that physical activity may be beneficial in preventing adverse health outcomes associated with chronically high levels of stress.

Stress, physical activity, and biological markers of disease risk. Though the majority of studies examining the stress-moderating effects of physical activity have examined associations with self-reported health outcomes, a small number of studies have examined associations with biological markers of disease risk. No systematic or meta-analytic reviews were located to aid in characterizing this literature.

In an early study, Young (1994) examined whether physical fitness (as assessed with maximal treadmill exercise test) moderated the effects of job stress and overall perceived stress on markers of cardiovascular risk in a sample of 412 male law enforcement officers but found no evidence that fitness modified the relation of either stress measure with blood pressure or cholesterol. However, hypotheses were tested by examining patterns in partial correlation matrices and this analytic approach is not the current convention for conducting tests of moderation.

More recent studies have also explored physical activity as a moderator of the effects of stress on biomarkers. For example, Puterman et al. (2012) found that physical activity among young adults experiencing financial strain was associated with a lower risk of developing impaired fasting glucose 13 years later. This protective effect of physical activity was not apparent in participants with low financial distress. In a cross-sectional study by the same

research group, higher levels of perceived stress were associated with shorter telomere length among non-exercisers whereas perceived stress was unrelated to telomere length among exercisers (Puterman et al., 2010). These two studies suggest that physical activity protects highly stressed individuals by buffering the relationship between stress and indicators of impaired physiological functioning. Although these results demonstrating a stress-buffering effect of physical activity are intriguing, psychological stress was not measured in great detail. Future studies should move beyond subjective single-scale stress assessments to better capture the magnitude and chronicity of participant's stress exposure.

Three additional studies focused on young people have examined the interactions between physical activity, stress and components of the metabolic syndrome. In one large longitudinal study of 2,375 girls, physical activity buffered the effects of chronic stress on BMI changes from ages 10 to 19 such that chronic stress levels over the 10 year study period were less predictive of BMI growth among those who were more active compared to those who were less active (Loharuka et al., October 2012). Another study of 303 individuals between the ages of 12 and 24 found that the interaction of both personal and community stress with physical activity significantly predicted adiposity (Yin, Davis, Moore, & Treiber, 2005). In a smaller study of 38 boys between the ages of 8 and 18 years, trait anxiety was associated with higher metabolic risk scores but only among individuals who reported low levels of physical activity (Holmes, Eisenmann, Ekkekakis, & Gentile, 2008). These studies suggest that physical activity offers health-protective benefits to individuals experiencing high levels of stress during critical periods of human development. Thus, in addition to increasing energy expenditure, physical activity may also protect individuals from the health-damaging effects of chronic stress.

Stress, physical activity, and mortality. Evidence from two epidemiological studies

demonstrates that physical activity moderates the stress-mortality relationship. In a sample of 5,249 middle-aged men without cardiovascular disease, the relationship between psychological stress at work and all-cause mortality over 30 years was moderated by levels of physical fitness such that physical fitness was more strongly associated with all-cause mortality for men experiencing regular psychological pressure at work than among those who rarely experienced work-related stress (Holtermann et al., 2011). Physical inactivity also moderated the association between perceived stress and risk of death over a seven-year period in a large nationally representative sample of 40,335 U.S. adults (Krueger & Chang, 2008). Notably, this interaction was only significant among low SES participants in stratified analyses suggesting that the combination of inadequate physical activity and high perceived stress is particularly detrimental among low SES individuals. While these studies provide convincing evidence that physical activity may moderate the effects of stress on health, the brief stress measures used in these studies make it impossible to discern if physical activity is equally beneficial for stressors across different life domains and/or dependent on the chronicity of stressors.

Animal models of stress and physical activity. Animal models also provide convincing evidence to support the hypothesis that habitual physical activity counteracts the harmful effects of stress exposure on behavioral, emotional, immunologic, neural, and cellular levels (Dishman et al., 2012). Rats who are exposed to uncontrollable stress in the form of shocks later exhibit a deficit in learning to escape from escapable stress; this behavioral pattern is known as learned helplessness (Seligman and Beagley, 1975; Weiss and Glazer, 1975). However, chronic freewheel running in rats prior to uncontrollable stress exposure prevents against such behavioral responses (Dishman et al., 1997; Greenwood et al., 2003; Solberg, Horton, & Turek, 1999). Voluntary wheel running in the weeks prior to acute stress exposure has also been shown to

prevent the suppression of NK cell cytotoxicity (Dishman et al., 1995) and anti-KLH IgM and IgG2a antibodies (Moraska & Fleshner, 2001) observed in sedentary rats in response to footshock. Some evidence suggests that these stress-modulating effects occur due to activity-induced alterations in central nervous system activity, especially within the serotonergic and norepinephrine systems (Dishman et al., 2012). Though this literature does not offer a complete understanding of the physiological mechanisms involved in the stress-protective effects of physical activity and some effects may not generalize to humans, animal models suggest that regular voluntary activity can attenuate neural responses to stress that lead to elevated sympathetic activity, immunosuppression, and ultimately a variety of adverse health outcomes.

Theoretical Frameworks for the Stress-Moderating Effects of Physical Activity

A number of theories have been proposed to explain how participation in physical activity might protect individuals from the health-damaging effects of stress. These theories are often based on the assumption that the similarities between physiological responses to exercise and mental stressors means that habituation to physical activity also leads to reduced sensitivity and improved adaptation to psychological stress (Li & He, 2009; Salmon, 2001; Tsatsoulis & Fountoulakis, 2006). For example, the *cross-stressor adaptation hypothesis* asserts that the adaptations to exercise stress that occur with regular training lead to adaptations in response to other challenges (Sothmann et al., 1996). A bout of intense exercise that is intended to improve aerobic fitness usually elicits a stress response in the short term; in the long term, regular training induces adaptation in cardiorespiratory, muscular and neural responses and promotes coordination between the systems to minimize the disruption to homeostasis in response to exercise. Thus, engaging in physical activity of sufficient intensity and duration may induce

adaptive changes in the stress response system that also modify responses to future psychosocial stressors.

Another theoretical approach to understanding the role of exercise and fitness in stress research comes from Dienstbier's (1989) conception of "physiological toughness," which is characterized by a low basal rate of sympathetic nervous system and catecholamines, a rapid and robust response by these systems that allows the individual to cope with challenge, and a quick return to baseline when the challenge is over. Because modern life presents insufficient physical demands, neuroendocrine systems associated with arousal are not maintained near the optimum point of their genetic ranges. As a result of this discontinuity, individuals are not naturally toughened and thus experience a variety of negative health outcomes including weight control problems, poor muscle tone, poor endurance, and stress intolerance. To promote physiological toughening, Dienstbier advocates engagement in a program of aerobic exercise which can bring about the adaptations that are necessary for optimal responses to physical or mental stress.

Physical activity has also been hypothesized to facilitate the development of cognitive resources that influence stress appraisal and coping. The association between physical activity and self-esteem has been well-documented in cross-sectional studies (Asci, Kosar, & Isler, 2001; Dishman et al., 2006; Ensel & Lin, 2004; Fox, 2000, 2001) and there is some evidence that physical activity interventions lead to increased self-esteem in children and young adults (Dishman et al., 2006; Ekeland, Heian, Hagen, & Coren, 2005; Ekeland, Heian, Hagen, Abbott, & Nordheim, 2005; Fox, 2001) and in adults (Spence, McGannon, & Poon, 2005). Physical activity has also been associated with better perceived coping abilities and reduced anxiety (DiLorenzo et al., 1999; Moses et al., 1989; Steptoe et al., 1989). Engaging in challenging recreational activities and mastering difficult physical tasks may enhance self-esteem, self-

efficacy, and feelings of competence (Long, 1993). In addition to fostering the development of personal resources, leisure-time physical activity may enable social interactions that allow individuals to develop larger social networks and close friendships that lead to enhanced perceived social support (Coleman & Iso-Ahola, 1993). Especially when accompanied by personal and social resources, physical activity might contribute to resilience and allow women to "survive, manage, and thrive" in the context of chronic postpartum stress.

Does Physical Activity Moderate the Effects of Stress on Postpartum Health?

Although no studies examining physical activity as a moderator of relationship between stress and postpartum physical health were located, one large study of 5,000 mothers of infants in Australia found that frequency of leisure-time physical activity moderated the association between life stress and depressive symptoms. There was a weak positive association between perceived stress and levels of depressive symptoms for women with higher levels of physical activity, and a stronger positive association for women with low levels of physical activity (Craike, Coleman, & MacMahon, 2010). These results support longitudinal findings in the general population that have shown a stress-protective effect of physical activity on depression (Toker & Biron, 2012).

Additional evidence suggests that engaging in physical activity before, during, and after pregnancy may have long-term effects on maternal physical health. The first year postpartum is a point when some women do not lose their pregnancy weight and therefore become overweight or obese (Amorim, Rössner, Neovius, Lourenço, & Linné, 2012; Rooney & Schauberger, 2002; Rooney, Schauberger, & Mathiason, 2005) but women who exercise are less likely to gain too much weight during pregnancy or have trouble losing excess weight after birth (Clapp & Little, 1995). Although no studies examining the effects of physical activity on postpartum

inflammation were located, Taveras, Rifas-Shiman, Rich-Edwards, and Mantzoros (2011) found that sleeping for less than five hours per night on average during the first year postpartum was associated with elevated levels of IL-6 (but not CRP) at 3 years postpartum. Thus, research suggests that health behaviors during the postpartum may have sustained effects on maternal health.

Health behaviors during the postpartum period may be especially important for women who are planning to have additional children as the postpartum period can also be thought of as the preconception period for the next child. Physical activity during the preconception period is important in facilitating the maintenance of healthy weight over the course of gestation. Women who exercise prior to becoming pregnant are also able to safely continue engaging in physical activity during pregnancy, and are also more likely to engage in physical activity during pregnancy. If physical fitness is an effective means of developing resilience to stress, it may be especially important for women during this time because elevated stress has been associated with a variety of adverse maternal and infant health outcomes, including postpartum depression, preterm birth, and infant developmental delays.

Understanding the triadic system of interactions between physical activity, psychosocial stress and physical health is particularly important in this population because the first year postpartum may set the stage for the adoption of lifestyle habits that could either promote or prevent long-term well-being in both new mothers and their children. Also, women who have recently given birth to a child may be particularly likely to experience high stress and low activity levels. The demanding life changes that accompany pregnancy, childbirth, and motherhood have been shown to contribute to high levels of perceived stress in this population, especially when these demands are coupled with chronic strain or other stressful life events

(Norbeck & Anderson, 1989; Ritter, Hobfoll, Lavin, Cameron, & Hulsizer, 2000). The postpartum period is also characterized by a variety of physiological adjustments including immune changes as the body transitions from a pregnant to a non-pregnant state. In addition to these social, psychological and physiological challenges, new mothers also may experience numerous barriers to physical activity, such as lack of time and inadequate access to childcare as evidenced by the declining rates of physical activity that occur during pregnancy and after childbirth (Brown et al., 2009; Pereira et al., 2007).

In sum, physical activity is potentially very important in the postpartum period for many reasons including that it may reduce effects of chronic stress and provide important longer-term health benefits. However, highly stressed individuals are also less likely to engage in health-promoting behaviors, including regular exercise (Ng & Jeffery, 2003; Steptoe et al., 1998). As noted above, non-white race, lower levels of education, and female gender are associated with a lower likelihood of meeting the recommended levels of physical activity outlined in the 2008 Physical Activity Guidelines for Americans. Unfortunately, these same groups also have the fewest socioeconomic resources and bear a disproportionate exposure to many sources of chronic strain. The current study addresses the need for further research to understand how regular physical activity and other potential sources of resilience affect stress processes during critical transitions across the lifespan, including pregnancy and motherhood.

Aims of Current Study

The current study explored associations between chronic stress, resilience resources, physical activity and inflammation in a large and relatively poor sample of African American/Black, Hispanic/Latina, and non-Hispanic White women during the year following the birth of a child. CRP is currently the only inflammatory marker available in CCHN and was

chosen as the outcome of interest in this study because it indicates elevated risk of disease that may not have manifested clinically in younger individuals but who are nonetheless vulnerable to the health-damaging effects of stress and physical inactivity.

There were two major objectives in the present study: 1) To describe the levels and correlates of physical activity at 6 months postpartum in mothers of three ethnic/racial groups and varying SES including a large proportion who are poor, and 2) To develop and test multivariate models of the combined influence of chronic stress, physical activity, and other resilience resources on CRP. In particular, this study addressed the following specific aims and hypotheses.

Specific Aim 1: Establish the demographic correlates of physical activity in community-residing women in five regions of the US at 6 months after a birth.

Hypotheses. Demographic factors including race, education, age and gender have been associated with likelihood of meeting the 2008 Physical Activity Guidelines for Americans (Carlson et al., 2010). Therefore, it was expected that African Americans and Latinas would report lower levels of physical activity than White women in this study. However, SES variables such as income and education were expected to partially explain observed ethnic group differences in physical activity. Also, women living in urban areas were expected to report higher levels of physical activity than individuals living in suburban and rural sites. Physical activity was also expected to be lower among older women, married women, employed women, and women who have multiple children.

Specific Aim 2: Determine whether chronic stress is associated with physical activity.

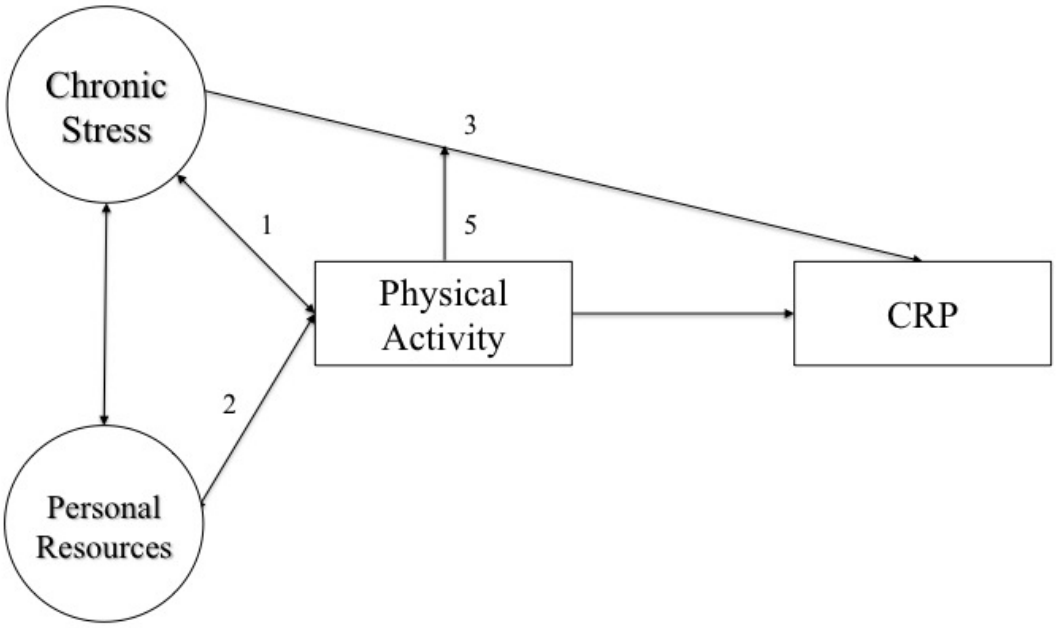
Hypotheses. Individuals experiencing high levels of chronic stress generally have fewer opportunities to engage in health-promoting behaviors. Therefore, it was expected that higher

levels of chronic stress would be associated with lower levels of physical activity among new mothers at 6 months postpartum when controlling for potential confounders including race/ethnicity, SES, type of residential area (rural, suburban or urban), parity, relationship status, employment status, and age (see Figure 1, Path 1).

Specific Aim 3: Determine whether personal resources that CCHN studied under the umbrella concept of resilience resources are associated with physical activity.

Hypotheses. Self-esteem, mastery, optimism, and perceived social support have been linked individually to higher levels of physical activity in past research. Therefore, it was expected that individuals with greater personal resources (defined here as self-esteem, mastery, optimism, and perceived social support) would report higher levels of physical activity during the first year postpartum, controlling for race/ethnicity, SES, type of residential area (rural, suburban or urban), parity, relationship status, employment status, and age (see Figure 1, Path 2).

Figure 1. Hypothesized relationships between chronic stress, personal resources, physical activity, and C-reactive protein (CRP)



Specific Aim 4: Test the association between chronic stress and elevated CRP.

Hypotheses. Psychosocial stress has been associated with physiological dysregulation in the HPA axis and immune system that results in systemic inflammation, which in turn increases risk of many chronic diseases including heart disease, diabetes, and depression. Therefore, we hypothesized that higher levels of chronic stress will be associated with higher levels of CRP controlling for race/ethnicity, SES, age, parity, health behaviors, chronic health conditions, and medication use (see Figure 1, Path 3).

Specific Aim 5: Test multivariate models of the combined influence of chronic stress, personal resilience resources, and physical activity on CRP.

Hypotheses. Based on the existing though small literature demonstrating a stress-buffering effect of physical activity on health outcomes, physical activity was hypothesized to moderate the association of chronic stress on CRP (see Figure 1, Path 5). Specifically, stronger

relationships between chronic stress and CRP were expected among physically inactive women as compared to women who were physically active. Psychological resilience resources were also explored as a potential moderator between chronic stress and CRP.

An alternative hypothesis is that chronic stress influences inflammation indirectly via lower levels of physical activity. That is, lower levels of physical activity may explain the relationship between stress and CRP. I also planned to test this model to determine whether physical activity mediates the association between chronic stress and CRP levels.

Chapter Two: Methods

Study Design

The sample includes participants in the Community Child Health Network (CCHN) study, a five-year longitudinal, multi-site study of 2,510 mothers and 1,436 of their partners or the fathers of their children. The Eunice Kennedy Shriver National Institutes of Child Health and Human Development of the National Institutes of Health funded CCHN to investigate disparities in maternal child health and improve the health of families through community-based participatory research. The overall goal of CCHN was to gain new insights into disparities in maternal health and child development. The primary aims of the network were 1) to explore how stress and resilience influence maternal allostatic load during the inter-conceptional period, and to examine whether allostatic load in turn predicts birth outcomes and child health and development in subsequent pregnancies, and 2) to evaluate the usefulness of community-partnered participatory research for conducting research on health disparities, and understanding how communities and families create contexts that influence pregnancy and early child health and development outcomes (Ramey et al., 2014).

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). Women 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 if 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), 12 months (T3), and 24 months (T5) of age. An abbreviated telephone interview was also conducted at 18 months (T4). 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 all assessments in the participant's home, and very rarely at another convenient setting. Interviewers were also trained to collect biological data at the T2 and T3 study visits including: 1) blood pressure, 2) height and weight for calculation of BMI, 3) waist and hip measurements, and 4) blood spots for CRP, HbA1c, and HDL and LDL cholesterol assays. Mothers who became pregnant again during the study period were interviewed during the second (wave referred to as P1) and third (P2) trimesters of their subsequent pregnancies, as well as 1 month after the birth of the subsequent child (P3).

Participants

The full CCHN cohort includes 2,510 mothers (54% African American, 24% Latina, and 22% non-Hispanic White) who completed the T1 interview 2 to 16 weeks after the birth of a child, or what was referred to as the "T1 catch-up interview" containing most of the needed sections of the T1 interview at the same time as the completion of the T3 interview. The cohort also includes 1,436 fathers or partners but only data collected from mothers were used in this study. Given the local demographics, Washington, DC and Baltimore sites recruited more African American participants, while Los Angeles and Lake County recruited more Latino/a participants into the study. The CCHN sample intentionally over represents families living below, at, or just slightly above the federal poverty level. All participants were recruited between 2008 and 2010.

Due to attrition over the course of the study, the sample used for the present analyses varied depending on the outcome of interest. Characteristics of the full cohort and sample used in each set of analyses are provided below in Table 1. The first set of analyses (Study 1) addresses research questions regarding the demographic and psychological correlates of physical activity (i.e., analyses in which physical activity was the criterion variable) and excludes 822 participants who did not complete the T2 interview containing the physical activity measure at 24-39 weeks postpartum. A total of 74 participants were also excluded if they were pregnant at the time of T2 study visit, or if they had insufficient data on the physical activity measure to categorize their physical activity levels (IPAQ) (n = 33). The final sample of 1,581 mothers included in physical activity analyses did not differ significantly from the full cohort with respect to income, education, age, relationship status, employment status, or parity. In comparison to the full CCHN cohort, participants in this analysis sample were less likely to be Latina (22% vs. 24% in the full

cohort), less likely to have been recruited from the Los Angeles site (12% vs. 17% in the full cohort), and more likely to have been recruited from the Lake County site (25% vs. 23% in the full cohort) or the North Carolina site (19% vs. 17%).

CRP is the criterion variable in the second set of analyses (Study 2). These analyses include data from a smaller subset of participants who completed both the T2 (24-29 weeks post partum) and T3 (50-65 weeks post partum) study visits and also provided a usable blood spot at either T2 or T3. Of the 2,510 mothers in the full CCHN cohort, 1,364 participants (54.3%) completed both the T2 and T3 study visits. Participants were also excluded from Study 2 analyses if they were pregnant at the time of the T2 or T3 visit (n = 137). Of the 1,227 eligible mothers with complete study visits, 88% provided a usable blood spot at T2 and/or T3 (n = 1,206). Repeated CRP measurements were available for 1,112 of these mothers. An additional 37 participants had CRP values only at T2 and 57 participants had CRP values only at T3. Reasons that participants with complete study visits were missing CRP data include participant refusal due to contraindications with current medications or health conditions (n = 5) or refusal for unspecified reasons (n = 4). An additional 21 participants provided blood spots but the lab determined there was an insufficient quantity for the assay. Compared to the full CCHN cohort, participants in the Study 2 sample were less likely to be from Los Angeles (11% vs. 17%) and more likely to be from the Lake County (26% vs. 23%) and North Carolina sites (20% vs. 17%). There were no other significant demographic differences between the full cohort and the sample included in CRP analyses.

Table 1. Sample Characteristics

	Full Cohort (*n* = 2,510)		PA Sample (*n* = 1,581)		CRP sample (*n* = 1,206)	
Categorical Variables	*n*	(%)	*n*	(%)	*n*	(%)
Race/ethnicity						
African American/Black	1349	(53.8)	861	(54.5)	651	(54.0)
White/Caucasian	554	(22.1)	368	(23.3)	279	(23.1)
Hispanic/Latina	607	(24.2)	352	(22.2)	276	(22.9)
Household income quintiles						
Q1 (lowest)	504	(20.1)	309	(19.5)	244	(20.2)
Q2	500	(19.9)	310	(19.6)	236	(19.6)
Q3	478	(19.0)	305	(19.3)	245	(20.3)
Q4	540	(21.5)	332	(21.0)	240	(19.9)
Q5	488	(19.4)	325	(20.6)	241	(20.0)
Education						
Less than HS	469	(18.9)	277	(17.7)	208	(17.3)
HS or equivalent	1075	(43.4)	677	(43.2)	520	(43.1)
Some college	564	(22.8)	367	(23.4)	282	(23.4)
4 year degree or higher	371	(15.0)	245	(15.6)	190	(15.8)
Relationship status						
Not married or cohabitating	1013	(40.4)	684	(43.3)	511	(42.4)
Cohabitating, not married	704	(28.1)	396	(25.1)	309	(25.6)
Married	792	(31.6)	501	(31.7)	386	(32.0)
Site						
Baltimore	554	(22.1)	363	(25.1)	259	(21.5)
Lake County, IL	579	(23.1)	396	(12.1)	317	(26.3)
Los Angeles County	431	(17.2)	191	(19.4)	127	(10.5)
Eastern North Carolina	437	(17.4)	306	(20.6)	243	(20.2)
Washington, DC	509	(20.3)	325	(25.1)	260	(21.6)
Type of Residential Area						
Urban	1494	(59.5)	879	(55.6)	646	(53.6)
Suburban	579	(23.1)	396	(25.1)	317	(26.3)
Rural	437	(17.4)	306	(19.4)	243	(20.2)
Multiparity	1289	(54.6)	807	(54.5)	616	(54.5)
Continuous variables	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)
Age	25.7	(5.7)	25.8	(5.7)	25.8	(5.7)
Education (years)	12.9	(2.8)	13.0	(2.8)	13.0	(2.8)
Per capita household income[a]	10,800	(11,120)	10,960	(11,070)	10,700	(10,900)

[a]Adjusted for cost of living.

Demographic Measures

Race/ethnicity. Participants self-reported their primary racial/ethnic identification at the time of study enrollment as African American/Black, White/Caucasian or Latina/Hispanic.

SES. Participant education and household income were included as indicators of socioeconomic status in this study and were recorded during the T1 interview. Participants reported the number of years of education they had completed, which resulted in a continuous education variable with a range of 4 to 23 years. Participants were also asked to report the highest degree they had completed, from no education to a doctoral or professional degree, and responses were recoded into four categories: less than high school, high school diploma or equivalent, some college, and 4-year degree or higher.

During the T1 interview, participants were asked about their pretax household income in the previous calendar year using ordered categories. The midpoint value for each category was assigned to create a continuous gross household income variable. Per capita household income was computed by dividing gross household income by the number of household members. Due to variability in the cost of living across the five study sites, per capita household income was adjusted using cost of living indices available from the US Census Bureau. Because the distribution of per capita household income was not normally distributed (skew= 7.43, kurtosis = 90.56) and included several outliers at the upper end of the distribution, this variable was winsorized at the top 5% of the distribution (i.e., per capita household incomes greater than $40,180 per capita were recoded to equal $40,180). This resulted in a more normally distributed distribution (skew = 1.33, kurtosis = 3.83). To explore possible non-linear effects of income, cost of living adjusted per capita household income was also coded into quintiles based on the sample distribution.

Stress Measures

Financial Stress. A Financial Stress index was created from five questions administered during the T1 interview. This measure differs slightly from the Financial Stress index used in previous CCHN publications (Dunkel Schetter et al., 2013). Table 2 lists each item and its response scale. Participants' responses to these five items were averaged to create a composite score with a range of 1 to 4. Higher scores indicated higher levels of Financial Stress.

Table 2. Individual Items and Response Scales Used to Calculate Financial Stress Composite Score

Item	Response scale
To what extent were worries about food, shelter, health care, and transportation stressful for you during your pregnancy?	1= no stress 2 = some stress 3 = moderate stress 4 = severe stress
To what extent were money worries like paying bills stressful for you during your pregnancy? Would you say	1= no stress 2 = some stress 3 = moderate stress 4 = severe stress
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)? IF YES: How was this experience for <u>you</u> personally?	1 = not endorsed or endorsed but was not rated as aversive 2 = endorsed and rated as slightly negative or undesirable 3 = endorsed and rated as somewhat negative or undesirable 4 = endorsed and rated as very negative or undesirable
How difficult is it for (you/your household) to meet the monthly payments on your (household's) bills?	1 = not difficult at all 2 = slightly difficult 3 = somewhat difficult 4 = very or extremely difficult
How much do you worry that your total (household) income will not be enough to meet your (household's) expenses and bills?	1 = not at all 2 = a little 3 = a lot 4 = a great deal

During the T1 interview, Participants also responded to two additional items assessing food insecurity. While these items were not included in the Financial Stress index score, they

were used as an additional indicator of financial stress in the structural equation model tested in Study 2. Participants were asked to report how often the statement "The food that (I/we) bought just didn't last, and (I/we) didn't have money to get more" was true in their households in the last 12 months on a scale ranging from 1 (*often true*) to 3 (*never true*). Participants also answered yes (coded as 1) or no (coded as 0) to the question "In the last 12 months, were you ever hungry but didn't eat because you couldn't afford enough food?". Responses to the first item were reverse scored and then the two items were summed to create a food insecurity index ranging from 1 to 4, with higher scores indicating greater food insecurity.

Chronic life stress. Participants completed the CCHN Life Stress Interview (LSI), a semi-structured interview based on the UCLA LSI (Hammen et al., 1987; Hammen, Marks, Mayol, & DeMayo, 1985) at T2. Participants responded to open-ended questions regarding *neighborhood environment*, *family relationships*, *co-parenting* and *partner relationships* with a focus on the previous six months (since the birth of the index child). Based on objective conditions reported by the participant, interviewers assigned overall ratings in each domain, as well as for more specific subdomains within each major domain (see Table 3 for a list of subscores within each domain). For each domain and subdomain, interviewers assigned a score using a five-point Likert scale ranging from 1 (*exceptionally positive conditions*) to 5 (*exceptionally negative conditions*). The overall domain ratings were a summary judgment made by the interviewer rather than a mathematical average of the subdomain ratings. 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 (Tanner Stapleton, Dooley, Paek, Huynh, & Dunkel Schetter, 2014). Chronic stress summary scores (i.e. total LSI

Table 3. Life Stress Interview: Major Domains and Subdomains

Domain	Scores
Neighborhood Environment	Safety Noise Familiarity/Problems with Neighbors Overall Rating
Family Relationships	Availability/Contact Closeness/Trust/Openness Acceptance Support Conflict/Resolution Overall Rating
Partner Relationships [For Participants Who Have One]	Commitment/Stability Closeness/Trust/Confiding Support/Dependability Conflict/Resolution Overall Rating
Partner Relationships [For Those Not Currently in a Committed Relationship]	Pressure to Have Partner Single Parenting Strain Overall Rating
Co-Parenting with Baby's Father	Overall Rating
Co-Parenting with Partner	Overall Rating

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, T2, and T3 using the 10-item brief version of the Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). This scale measures the perceived unpredictability and uncontrollability of general life stress using items such as "how often have you felt you were effectively coping with important changes that were occurring in your life?" and "how often have you been upset because of something that happened unexpectedly?" At T1 and T2, participants were asked to indicate how frequently they had felt each way since the baby was born on a scale ranging from 1 (*never*) to 5 (*almost always*). Responses to the 10 items were 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.

Everyday racial discrimination. Experiences of racial discrimination were measured at T1 using the Everyday Racism Scale (Forman, Williams, & Jackson, 1997). This measure assesses frequency of experiences of discrimination in everyday life such as being treated with less courtesy or respect than others, receiving poorer services in restaurants or stores, and other chronic, routine and less overt experiences of unfair treatment. 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 asked what they thought was the main reason for these experiences. 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.

Resilience Measures

Mastery. Mastery was assessed at T1 using the 7-item Mastery Scale (Pearlin & Schooler, 1978). This scale was modified slightly for use in CCHN. Items were embedded with six other items assessing self-esteem, and the instructions were changed to be appropriate for use with all items. The instruction set stated: "The next set of questions concern general feelings about yourself. For each item, please tell me if you strongly agree, agree, feel neutral, disagree or strongly disagree." Responses were given on a scale ranging from 1 (*strongly disagree*) to 5

(*strongly agree*). Two negatively worded items were reverse coded and then all items were summed to create a total mastery score, such that higher scores reflect greater mastery.

Self-esteem. A 6-item modified version of the Rosenberg Self-Esteem Scale was used to assess trait self-esteem at T1 (Rosenberg, 1965). The instruction set and response scale used to assess self-esteem were identical to those described above for the Mastery scale. Four of the original items from the Rosenberg scale were omitted and slight changes were made to the remaining six questions to aid in participant understanding of the items. Negatively worded items were reverse scored and then all items were summed to generate a total self-esteem score such that higher scores on this scale reflect greater self-esteem.

Optimism. Dispositional optimism was measured at T2 with a modified version of the 8 item Life Orientation Test (Scheier & Carver, 1985). The instruction set used in CCHN omitted a sentence found in the original instructions asking participants to be honest and accurate, and were also edited to include an introduction to the section of the interview. The instructions stated" I have a few statements now about your attitude toward life. There are no correct or incorrect answers. Please answer according to your own feelings, rather than how you think 'most people' would answer." The response scale ranged from 1 (*I agree a lot*) to 5 (*I disagree a lot*). Four filler items were embedded in the scale. All positively worded items were reverse coded and all non-filler items were summed to generate a total optimism score such that higher scores indicate greater optimism.

Perceived social support. Social support was measured during the T1 interview using the 19-item Medical Outcomes Study Social Support Survey (Sherbourne & Stewart, 1991). This measure assesses perceived availability of emotional/informational support (8 items), tangible support (4 items), affectionate support (3 items), and positive social interaction (3 items). One

additional item asks about the availability of someone that can help get the participant's mind off of things. Participants were asked to rate how often they each of these support mechanisms available, ranging from 0 (*none of the time*) to 5 (*all of the time*). Subscale scores are obtained by calculating the average score from each item in the subscale, and an overall functional support index is calculated by averaging the scores for all items. Higher scores (for the index as well as subscales) indicate greater perceived availability of support. In this study, the scaled index score was transformed into a normative score (0-100) by using this equation: 100 X (observed score – minimum possible score)/(maximum possible score – minimum possible score).

Health Behavior Measures

Physical activity. Physical activity was assessed at T2 (6 months postpartum) using the short 9-item form of the International Physical Activity Questionnaire (IPAQ). The IPAQ short has acceptable test-retest reliability and demonstrates fair to moderate associations with accelerometry (Ainsworth et al., 2000; Ainsworth et al., 2006; Hagstromer, Oja, & Sjostrom, 2006). Participants were asked to report activities performed for at least 10 minutes during the last seven days. The short version of the IPAQ assesses time spent in physical activity across work, housework, transportation, and leisure domains at 3 different intensities: vigorous, moderate, and walking. Participants were provided with examples of activities that represent each intensity (e.g. "vigorous physical activities like heavy lifting, aerobics, or fast bicycling") and then asked to report the number of days and number of hours and/or minutes per day they had spent doing each activity for a minimum of ten minutes over the previous 7 days. Total weekly physical activity energy expenditure was calculated by weighting time spent in each activity intensity with its estimated metabolic equivalent (MET). Using the instrument's well-validated scoring protocol, participants were then classified into categories representing low,

moderate, and high levels of physical activity. Briefly, participants were classified as moderately active if they reported 3 or more days of vigorous activity of at least 20 minutes per day, 5 or more days of moderate-intensity activity and/or walking of at least 30 minutes per day, or 5 or more days of any intensity activity totaling at least 600 MET-minutes per week. Participants were classified as highly active if they reported vigorous activity on at least 3 days and achieving at least 1500 MET-minutes/week or 7 or more days of any intensity activity accumulating at least 3000 MET-minutes/week. Participants who reported no activity or some activity but less than the moderate criteria were assigned to the low activity category.

Other health behaviors. 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 non-drinkers if they reported no alcohol use, 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), or as moderate drinkers if reported alcohol use but did not meet CDC criteria for excessive drinking. Finally, participants reported their average number of hours of sleep per night in the previous month.

Biomarker Measures

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). In this study, 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 (Kapur, Kapur, & Zava, 2008; McDade, Burhop, & Dohnal, 2004). 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 (Kapur et al., 2008). 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%

Adiposity. Study personnel obtained measures of height, weight, and waist circumference at T2 and T3. Participants were asked to remove shoes and heavy clothing. 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. 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). Waist circumference was measured at the navel using a cloth measuring tape and recorded to the nearest cm. The measurement was then repeated and the average of the two measurements was used.

Health-Related Measures

Health conditions. 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 health conditions were of interest in the present analyses: high blood pressure or hypertension, anemia and other blood

problems, high cholesterol, heart problems, diabetes, kidney problems, liver problems/hepatitis, cancer, thyroid problems, asthma/reactive airway disease/bronchitis, epilepsy, periodontal/gum disease, tuberculosis, HIV/AIDS, and any other disease, major health problems, or chronic medical conditions. Participants were classified as having a particular condition if their charts indicated a history of that infection or illness 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. Given the large number of possible health conditions, a summary count of the total number health conditions was used in all analyses.

Medication. Information about medication usage was also collected at T2. Medications including anti-hypertensives, cholesterol-lowering, and antidepressants have anti-inflammatory properties (Jaim & Ridker, 2005; Kenis & Maes, 2002; Pradhan et al., 2001; Ridker, Hennekens, Rifai, Buring & Manson, 1999). On the other hand, use of oral contraceptives has been associated with higher CRP (Buchbinder et al., 2008; Dreon, Slavin, & Phinney, 2003; Kluft, Gevers Leuven, Helmerhorst, & Krans, 2002; Williams, Williams, Milne, Hancox, & Poulton, 2004). Dummy-coded variables (0 = *no*, 1 = *yes*) indicating self-reported use of these medications were included in analyses.

Overview of Analytic Strategy

Data analysis included univariate, bivariate, and multivariate techniques. First, frequencies and descriptive statistics were used to summarize data on study variables including demographic covariates, physical activity, stress and resilience variables, BMI, and CRP. Correlational analyses and ANOVA were used to test bivariate relationships between study variables in preliminary analyses. Multinomial logistic regressions were used to test for significant independent correlates of physical activity. Multivariate linear regression and logistic

regressions were used to test for significant predictors of CRP. Finally, structural equation modeling was used to model associations between stress, adiposity, and CRP. Specific analytic procedures are described in detail before each set of results. All continuous variables were mean-centered prior to use in regression analyses, and significance was set at $\alpha = .05$. Statistical analyses were primarily conducted using Stata 13, and EQS 6.2 (Bentler, 2006) was used for structural equation modeling.

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, there was a large percentage of missing data for per capita household income because 15% of mothers reported that they did not know their total household income. An additional 1% refused to report their total household income. The CCHN DCAC imputed missing data for cost-of-living adjusted per capita household income, and imputed values were used for 17% of the sample in the present analyses. When items that were needed to compute stress and resilience scale scores were missing, mean replacement was used if at least 70% of items were complete (see Dunkel Schetter et al., 2013).

In regression analyses, missing values for other covariates (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 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. Five imputations were generated using chained equations procedures (*mi impute*). Results across the five imputed data

sets were averaged, and the standard errors adjusted using the *mi estimate* procedure in Stata. 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. Full-information maximum likelihood (FIML) procedures were used in testing structural equation models.

Assumptions

Frequency distributions for all continuous measures were visually examined using histograms, and normality assumptions were also assessed by examining skewness and kurtosis coefficients. Racial discrimination and interpersonal violence had highly skewed response distributions because a minority of participants endorsed any experience of racism or interpersonal violence. These values were log-transformed prior to conducting analyses, but descriptive statistics for untransformed scores are reported to facilitate interpretation. The distribution of CRP was also markedly positively skewed and there were several outliers at the upper end of the distribution (details described below under Study 2 Results). Prior to analyses, values of CRP were natural-log transformed to normalize the distributions and extreme values (≥3 SDs above the mean) were excluded.

For all multivariate regression analyses, we examined cases with high leverage (greater than twice the mean leverage; Belsley, Kuh, & Welsch, 2005) and identified cases with large residuals by inspecting scatterplots of residuals and by assessing Cook's D (Cook, 1977). Participants whose data were influential due to high leverage and large residuals were excluded from the analysis.

Chapter Three: Study 1 Results

Preliminary Analyses: Demographics

To better characterize the current sample prior to hypothesis testing, demographic characteristics for the total sample and within subgroups by race/ethnicity are presented in Table 4. ANOVAs for continuous variables and chi-square tests for categorical variables tested whether demographic characteristics of the sample differed based on race/ethnicity. *F* statistics that were significant at the $p < .05$ level were followed up with pairwise t-tests with Bonferroni correction for multiple comparisons, unless otherwise noted. As shown in Table 4, the race/ethnic groups were significantly different in age, income (continuous and quintiles), education, relationship status, employment status, type of residential area, and parity (*p*'s < .05). These differences are described in detail below.

Age. The mean age of participants differed by race/ethnicity. African Americans were significantly younger than Whites (*M* difference = -5.6 years; t (1227) = -16.96, p = <.001) and Latinas (*M* difference = -1.5 years; t (1211) = -4.43, p = <.001), and Whites were significantly older than Latinas (*M* difference = 4.1 years; t (718) = 10.41, p < .001).

Income. Per capita household income (adjusted for cost of living) also differed by race/ethnicity. Posthoc Tamhane comparisons, which were used because of unequal variances across groups, revealed that Whites in the current sample had a higher mean per capita household income of than African Americans (*M* difference = $9,830; p = <.001) and Latinas (*M* difference = $10,580; p = <.001). White participants were also less likely to be in the lowest income quintile (7%) than African Americans (25%) or Latinas (20%), and more likely to be in the highest income quintile (55%) than African Americans (18%) or Latinas (15%).

Table 4. Demographic Characteristics, for the Total Sample and for each Race/Ethnicity

		Race/Ethnicity			
	Total (*n* = 1,581)	Blacks (*n* = 861)	Whites (*n* = 368)	Latinas (*n* = 352)	Test of group differences
Categorical variables	n (%)	n (%)	n (%)	n (%)	*p*
Household income (quintiles)					<.001
Q1	309 (19.5)	211 (24.5)	27 (7.3)	71 (20.2)	
Q2	310 (19.6)	182 (21.1)	38 (10.3)	90 (25.6)	
Q3	305 (19.3)	160 (18.6)	46 (12.5)	99 (28.1)	
Q4	332 (21.0)	195 (22.7)	88 (23.9)	49 (13.9)	
Q5	325 (20.6)	113 (13.1)	169 (45.9)	43 (12.2)	
Educational attainment					<.001
Less than high school	277 (17.7)	136 (15.9)	19 (5.2)	122 (35.5)	
High school or equiv.	677 (43.2)	424 (49.6)	94 (25.6)	159 (46.2)	
Some college	367 (23.4)	236 (27.6)	87 (23.7)	44 (12.8)	
4 year degree or higher	245 (15.6)	59 (6.9)	167 (45.5)	19 (5.5)	
Relationship Status					<.001
Not cohabitating	684 (43.3)	548 (63.7)	59 (16.0)	77 (21.9)	
Cohabitating, not married	396 (25.1)	198 (23.0)	61 (16.6)	137 (38.9)	
Married	501 (31.7)	115 (13.4)	248 (67.4)	138 (39.2)	
T2 employment					<.001
Working part-time	250 (16.1)	110 (13.0)	78 (21.7)	62 (17.8)	
Working full-time	445 (28.7)	223 (26.4)	128 (35.7)	94 (27.0)	
Not working by choice	416 (26.8)	161 (19.1)	108 (30.1)	147 (42.2)	
Unemployed	440 (28.4)	350 (41.5)	45 (12.5)	45 (12.9)	
Residential Area					<.001
Urban	879 (55.6)	588 (68.3)	123 (33.4)	168 (47.7)	
Suburban	396 (25.1)	64 (7.4)	152 (41.3)	180 (51.1)	
Rural	306 (19.4)	209 (24.3)	93 (25.3)	4 (1.1)	
Multiparity (% yes)	807 (54.4)	415 (53.1)	181 (51.6)	211 (60.5)	0.03
Continuous variables	*M (SD)*	*M (SD)*	*M (SD)*	*M (SD)*	
Age	25.8 (5.7)	24.2 (4.9)	29.7 (5.9)	25.6 (5.3)	<.001
Education (years)	13.0 (2.8)	12.8 (2.0)	15.1 (2.9)	11.2 (2.8)	<.001
Per capita household income[a]	10,960 (11,070)	8,850 (9,500)	18,680 (12,800)	8,100 (8,880)	<.001

[a]Adjusted for cost of living

Education. On average, African Americans had completed significantly fewer years of education than Whites (*M* difference = 2.4 years; $t(1227) = 15.72$, $p = < .001$). Latinas had fewer years of education than both African Americans (*M* difference = 1.6 years; $t(1211) = 10.39$, $p = < .001$), and Whites (*M* difference = 4.0 years; $t(718) = 21.94$, $p = < .001$). A majority of African

American (66%) and Latina (82%) participants had completed a high school education or less, whereas most White women had attended at least some college or received a college degree (70%).

Relationship status. Among African Americans, 64% of mothers were not married to or living with the baby's father as compared to 16% of Whites and 22% of Latinas. White participants were more likely to be married to the baby's father than (67%) than African Americans (13%) or Latinas (39%).

Residential area. Given the local demographics, the North Carolina, Washington, DC and Baltimore sites recruited significantly more African American participants, while Los Angeles and Lake County recruited more Latino participants into the study. As a result, 68% of African American participants were living in urban areas (Washington, DC, Baltimore or Los Angeles) in comparison to 33% of Whites and 47% of Latinas. Very few Latinas (1%) were living in the one rural site (North Carolina), whereas 24% of African Americans and 25% of Whites were recruited from there.

Employment status. Employment status also differed significantly by race/ethnicity. A greater percentage of White participants were working either full or part time (57%), in comparison to 40% of African Americans and 45% of Latinas. Although the percentages of working African Americans and Latinas were similar, most non-working African Americans were unemployed (68%) whereas most non-working Latinas were not working by choice (77%).

Summary of Race/Ethnicity Demographic Differences

In sum, there were significant differences by race/ethnicity for all of the demographic variables. On average, White mothers were generally older, more educated, more likely to be married and employed, and had higher incomes as compared to African American and Latina

mothers. Latina mothers were less likely to be living in rural areas than White women or African American women and more likely to have given birth before their most recent pregnancy. African American mothers were least likely to be married to or living with the baby's father. These differences are noted because they illustrate racial/ethnic differences on key demographic variables, and this is important to keep in mind when comparing outcomes across groups. That is, differences between race/ethnic groups on physical activity or CRP may be confounded by demographic differences between the groups.

Preliminary Descriptives: Chronic Stress and Resilience

Descriptive statistics for all stress and resilience measures for the Study 1 sample are provided in Table 5. Data on each of these measures was missing for less than 6% of the sample, with the exception of Interpersonal Violence at Time 1, which was missing for about 20% of the sample.

Table 5. Descriptive Statistics for Stress Measures in Study Sample (n = 1,581)

Variable	*n*	*M*	*SD*	Min	Max	Skew	Kurtosis	Cronbach α	% missing
Stress Variables									
T1 Financial Stress	1576	1.88	0.71	1	4	0.79	2.93	.77	0.0
T2 Life Stress Interview									
Neighborhood Stress	1548	2.02	0.76	1	5	1.04	4.16	.80	2.1
Family Stress	1548	1.69	0.69	1	5	1.75	6.74	.88	2.1
Co-Parenting Stress	1492	2.09	1.31	1	5	1.16	3.11	n/a	5.6
Partner Relationship	1523	1.65	0.72	1	5	1.67	5.93	.90[a]/.76[b]	3.7
Total Life Stress	1526	1.97	0.67	1	5	0.91	3.63		3.5
T1/T2 PSS Composite	1579	13.26	5.91	0	34	0.27	2.70	.89	0.1
T1 Discrimination	1558	2.45	4.57	0	31	2.63	11.31	.88	1.5
T1 Interpersonal Violence	1273	6.38	2.35	5	24	2.83	14.13	.81	19.5
Resilience Variables									
T1 Mastery	1581	28.03	3.94	14	35	-0.31	2.94	.71	0.0
T1 Self-Esteem	1581	25.82	3.10	9	30	-0.81	4.62	.80	0.0
T2 Optimism	1581	31.03	5.23	10	40	-0.46	3.18	.71	0.0
T1 Perceived Support	1578	83.37	18.22	2.63	100	-1.42	4.80	.96	0.2

Note. PSS = Perceived Stress Scale
[a]Cronbach's α for participants currently in a romantic relationship (*n* = 1,241)
[b]Cronbach's α for participants not currently in a romantic relationship (*n* = 282)

Specific Aim 1: *Describe the levels and demographic correlates of physical activity at 6 months postpartum in mothers of three ethnic/racial groups and varying SES including a large proportion who are poor.*

Levels of Physical Activity

Table 6 provides descriptive statistics for number of days per week, minutes per week, and energy expenditure (MET-mins/week) participants spent engaging in each type of physical activity. Most participants (67%) reported no vigorous activity in the previous week and only 10% reported 3 or more days of activities like heavy lifting, aerobics or fast bicycling. Forty-three percent of the sample reported no moderate activity while 28% reported 3 or more days and 18% reported 5 or more days of activities such as like carrying light loads or bicycling at a regular pace. Walking was reported more frequently than moderate or vigorous activity: 70% of the sample reported walking on 3 or more days, and 38% reported 5 or more days.

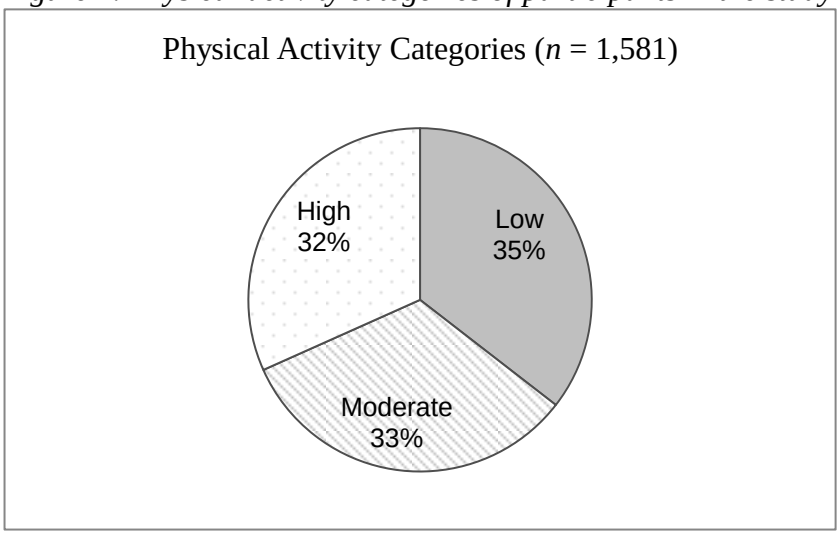
Table 6. Physical Activity Descriptives by Type (n = 1,581)

Variable	*M*	(*SD*)	*Mdn*	Min	Max	Skew	Kurtosis
Vigorous							
Days/week	0.98	(1.76)	0	0	7	1.98	6.20
Minutes/day	24.80	(47.57)	0	0	180	2.17	6.86
MET-mins/week	659.91	(1648.81)	0	0	10080	3.68	17.80
Moderate							
Days/week	2.29	(2.62)	1	0	7	0.79	2.13
Minutes/day	47.25	(62.22)	20	0	180	1.19	2.97
MET-mins/week	834.27	(1369.97)	180	0	5040	1.92	5.70
Walking							
Days/week	4.20	(2.59)	5	0	7	-0.31	1.66
Minutes/day	62.77	(62.24)	30	0	180	0.96	2.47
MET-mins/week	1089.68	(1292.16)	495	0	4158	1.34	3.52
Total							
Minutes/day	177.52	(196.95)	105	0	960	1.62	5.20
MET-mins/week	2573.98	(2890.46)	1476	0	19278	1.80	6.78

Note. MET = Metabolic equivalent

Scores on the continuous physical activity variables were used to assign participants to one of three Physical Activity categories using the IPAQ scoring criteria as described above in the Measures section. As shown in Figure 2, 35% of the sample fell into the Low Activity category, 33% were moderately active, and 32% were highly active.

Figure 2. Physical activity categories of participants in the study sample



Correlates of Physical Activity

Bivariate associations. Table 7 reports demographic characteristics of participants by Physical Activity level. For categorical variables, chi-square tests tested whether each variable was associated with Physical Activity level. To examine group differences on continuous age, income, and education variables, ANOVAs were conducted and significant main effects of Physical Activity level were followed up with pairwise t-tests with Bonferroni correction for multiple comparisons. These bivariate analyses showed significant associations of Physical Activity with race/ethnicity, income, and education. Mean per capita household income was higher among participants in the Moderate Activity group than those in the Low (*M* difference = $1,986; *t*(1078) = 2.96, *p* = .01) and High activity groups (*M* difference = $2,331; *t*(1018) = 3.38, *p* = <.01). Participants in the Moderate Activity group had more years of education on average

Table 7. Socio-demographic Characteristics, by Physical Activity Level (n = 1,581)

	IPAQ Physical Activity (T2)						
	Low (*n* = 561)		Moderate (*n* = 519)		High (*n* = 501)		
Categorical variables	*n*	(%)	*n*	(%)	*n*	(%)	*p*
Race/ethnicity							<.001
African American/Black	345	(40.1)	253	(29.4)	263	(30.6)	
White/Caucasian	95	(25.8)	152	(41.3)	121	(32.9)	
Hispanic/Latina	121	(34.4)	114	(32.4)	117	(33.2)	
Education							<.001
less than high school	101	(36.5)	79	(28.5)	97	(35.0)	
High school or equivalent	245	(36.2)	212	(31.3)	220	(32.5)	
Some college	130	(35.4)	113	(30.8)	124	(33.8)	
4 year degree or more	77	(31.4)	111	(45.3)	57	(23.3)	
Other, no information	8	(53.3)	4	(26.7)	3	(20.0)	
Household income (quintiles)							<.001
Q1 (lowest)	120	(38.8)	93	(30.1)	96	(31.1)	
Q2	110	(35.5)	79	(25.5)	121	(39.0)	
Q3	96	(31.5)	107	(35.1)	102	(33.4)	
Q4	131	(39.5)	110	(33.1)	91	(27.4)	
Q5	104	(32.0)	130	(40.0)	91	(28.0)	
T2 Relationship Status							.01
Not cohabitating	256	(37.4)	203	(29.7)	225	(32.9)	
Cohabitating	147	(37.1)	121	(30.6)	128	(32.3)	
Married	158	(31.5)	195	(38.9)	148	(29.5)	
T2 employment							<.001
Working part-time	75	(30.0)	85	(34.0)	90	(36.0)	
Working full-time	130	(29.2)	160	(36.0)	155	(34.8)	
Not working by choice	157	(37.7)	138	(33.2)	121	(29.1)	
Unemployed	186	(42.3)	131	(29.8)	123	(28.0)	
Residential Area							<.001
Urban	286	(32.5)	315	(35.8)	278	(31.6)	
Suburban	137	(34.6)	124	(31.3)	135	(34.1)	
Rural	138	(45.1)	80	(26.1)	88	(28.8)	
Multiparity	293	(36.3)	243	(30.1)	271	(33.6)	.04
Continuous variables	M	(SD)	M	(SD)	M	(SD)	
Age	25.35_a	(5.51)	26.65_b	(6.19)	25.39_a	(5.32)	<.001
Education (years)	12.80_a	(2.62)	13.36_b	(3.08)	12.77_a	(2.56)	.001
Per capita household income[a]	$10,420_a$	(10,570)	$12,410_b$	(11,810)	$10,080_a$	(10,700)	.001

Note. Differing subscripts across groups indicate that groups differ at $p < .05$.
[a]Adjusted for cost of living.

than participants in the Low (*M* difference = .55 years; $t(1078) = 3.27$, $p = <.01$) and High Activity groups (*M* difference = .59 years; $t(1018) = 3.39$, $p <.01$). All other pairwise comparisons were nonsignificant ($p > .05$).

To examine possible non-linear relationships, associations of Physical Activity with income quintiles and education categories were also examined. Results of these analyses demonstrated a possible curvilinear relationship between Physical Activity and income, such that participants in the fourth quintile were more likely to be in the low Physical Activity category than those with both lower and higher incomes, and participants in the second income quintile were more likely to be highly active than those with both lower and higher incomes. Income quintiles were used in subsequent analyses. There was no evidence of a non-linear relationship between Physical Activity level and education, and the continuous years of education variable is used in subsequent multivariate models.

Patterns of Physical Activity level also varied significantly by maternal age, type of residential area, relationship status, employment status, and parity in bivariate analyses, as shown in Table 6. Women who were in the Moderate activity category were significantly older than women in both the Low (*M* difference = 1.30 years; $t(1078) = 3.76$, $p = .001$) and High (*M* difference = 1.26 years; $t(1060) = 3.76$, $p = .001$) activity groups. Women living in rural areas were less likely than those living in urban or suburban areas to be moderately or highly active. Women with more than one child were less likely than women who had recently given birth to their first child to be moderately active. Women who were married were more likely to be moderately active than women who were not married, regardless of whether or not they were cohabitating with the baby's father. Women who were unemployed were less likely to be moderately or highly active than women who were employed either full or part-time. Women

who were not working by choice were less likely to be highly active than women who were working either full or part-time.

Additional analyses explored associations between Physical Activity and BMI at T2 and T3. Results of ANOVAs indicated that there were no significant differences in T2 or T3 BMI values by Physical Activity category (p's > .50). Chi square analyses were also run with BMI categories (underweight/normal vs. overweight vs. obese) and there were no statistically significant differences in Physical Activity at either time point (p's > .20).

In sum, bivariate analyses demonstrated associations of Physical Activity with race/ethnicity, education, income, relationship status, employment status, type of residential area, parity, and age. These analyses were conducted as a preliminary step before completing the multivariate analyses described below to determine which variables were uniquely predictive of Physical Activity.

Hypothesis 1a. *African Americans and Latinas will report lower levels of physical activity than White women.*

This hypothesis was tested using multinomial logistic regression. The multinomial regression models estimated the likelihood of being in the Moderate and High Activity categories relative to the Low Activity category as a function of race/ethnicity as well as other demographic correlates in subsequent analyses. This approach was used rather than ordinal logistic regression because the assumption of proportional odds required for fitting ordinal models was not satisfied (i.e., the associations between a given predictor and Physical Activity differed across categories of Physical Activity). The initial model included two dummy-coded variables for race/ethnicity with Whites serving as the referent group. Additional demographic correlates were added to subsequent multivariate models to determine whether SES and other

demographic differences explained variation in physical activity levels among the racial/ethnic groups.

The hypothesis that African Americans and Latinas would report lower levels of Physical Activity as compared to White women was supported in the unadjusted multinomial logistic regression analysis. African American women and Latina women were less likely than White women to be moderately active relative to low active, and African American women were less likely than White women to be highly active (Table 8). There were no significant differences between African American women and Latina women (p's >.10).

Table 8. Multinomial Logistic Regression Models Predicting Physical Activity Categories from Race/Ethnicity (n = 1,581)

	OR for moderate activity vs. low activity		OR for high activity vs. low activity	
	OR	(95 % CI)	OR	(95 % CI)
Race/Ethnicity				
White	Ref.			
Black	0.46***	(0.34-0.62)	0.60***	(0.44-0.82)
Latina	0.59***	(0.41-0.85)	0.76	(0.52-1.10)

* $p < 0.05$; ** $p < 0.01$; ***$p < .001$.

Hypothesis 1b. *SES variables such as income and education are expected to partially explain observed ethnic group differences in physical activity.*

Income and education were entered along with race/ethnicity into a multinomial logistic regression to examine whether SES explained observed racial/ethnic differences in Physical Activity. Results are shown in Table 9. After including income and education in the model, African American women were still less likely than White women to be moderately or highly active. Marginally significant differences emerged between Latinas and African Americans for both Moderate ($p = .08$) and High Activity ($p = .06$) relative to Low Activity, such that African

American women were less likely than Latinas to be moderately or highly active. Income and education were not significant predictors of Physical Activity in this model.

Table 9. Multinomial Logistic Regression Models Predicting Physical Activity Categories from Race/Ethnicity and SES

	OR for moderate activity vs. low activity		OR for high activity vs. low activity	
Predictor	OR	(95 % CI)	OR	(95 % CI)
Race/Ethnicity				
White	Ref.			
Black	0.52***	(0.37-0.73)	0.54***	(0.39-0.77)
Latina	0.69	(0.45-1.04)	0.65*	(0.43-1.01)
Per capita household income				
Q1	Ref.			
Q2	0.88	(0.59-1.32)	1.35	(0.93-1.87)
Q3	1.33	(0.90-1.98)	1.31	(0.88-1.22)
Q4	0.91	(0.62-1.34)	0.82	(0.55-1.28)
Q5	1.13	(0.74-1.72)	0.92	(0.60-1.42)
Education (years completed)	1.04	(0.98-1.10)	0.99	(0.94-1.05)

* $p < 0.05$; ** $p < 0.01$; ***$p < .001$

Hypothesis 1c. *Individuals living in urban areas will report higher levels of physical activity than individuals living in nonurban sites. Physical activity is also expected to be lower among older women, married women, employed women, and women who have multiple children*.

As shown in Table 10, five of the eight demographic variables achieved statistical significance when entered into a simultaneous multinomial logistic regression, overall model $\chi 2(30, N = 1{,}581) = 101.45$, $p < .001$. African American and Latina women were less likely to be moderately or highly active in comparison to White women. Women residing in rural areas were less likely to be moderately or highly active than women living in urban or suburban areas. Having more than one child was associated with lower likelihood of Moderate Activity, but not with likelihood of High Activity. Women who were unemployed or not working by choice were less likely to be highly active than women who were working full or part time. Finally, greater maternal age was associated with lower likelihood of being highly active. Notably, the

association of race/ethnicity with physical activity remained significant even after adjustment for income, education, type of residential area, multiparity, relationship status, employment status, and maternal age. Relationship status was not associated with Physical Activity in this multivariate model.

Table 10. Multivariate Multinomial Logistic Regression Models Predicting Physical Activity Categories from Demographic Variables

	OR for moderate activity vs. low activity		OR for high activity vs. low activity	
	OR	(95 % CI)	OR	(95 % CI)
Race/Ethnicity				
White	Ref.			
Black	0.44***	(0.30-0.67)	0.45***	(0.30-0.69)
Latina	0.58*	(0.37-0.92)	0.58*	(0.37-0.91)
Per capita household income				
Q1	Ref.			
Q2	0.86	(0.56-1.31)	1.36	(0.90-1.87)
Q3	1.16	(0.76-1.77)	1.21	(0.54-1.16)
Q4	0.78	(0.51-1.18)	0.77	(0.57-1.36)
Q5	0.95	(0.59-1.53)	0.96	(0.59-1.56)
Education (years completed)	1.01	(0.95-1.07)	1.01	(0.94-1.07)
Residential Area				
Urban	Ref.			
Suburban	0.61**	(0.44-0.85)	0.83	(0.59-1.17)
Rural	0.43***	(0.29-0.62)	0.49***	(0.34-0.70)
Multiparity	0.75*	(0.56-0.99)	1.21	(0.91-1.62)
Relationship Status				
Not married or cohabitating	Ref.			
Cohabitating, Not Married	0.83	(0.58-1.16)	0.79	(0.56-1.10)
Married	1.08	(0.73-1.60)	0.84	(0.56-1.24)
T2 employment				
Not working by choice	Ref.			
Working part-time	1.21	(0.80-1.82)	1.55*	(1.03-2.34)
Working full-time	1.26	(0.88-1.80)	1.75**	(1.22-2.52)
Unemployed	0.93	(0.65-1.34)	0.94	(0.65-1.36)
Age	1.01	(0.98-1.04)	0.97*	(0.94-1.00)

* $p < 0.05$; ** $p < 0.01$; ***$p < .001$

Summary of Demographic Correlates of Physical Activity

In summary, Aim 1 tested hypothesized associations of race/ethnicity, and other demographic and background variables with Physical Activity. In multivariate analyses including all demographic predictors, several effects were revealed: First, African American race and Latina ethnicity, living in a rural area, and multiparity were associated lower odds of Moderate Activity as compared to Low Activity. Second, African American and Latina race/ethnicity, rural residence, unemployment or not working by choice, and higher age were associated with lower odds of High Activity as compared to Low Activity. Notably, neither of the two indicators of SES (income, education) was a significant predictor of Physical Activity, nor was relationship status related to Physical Activity in multivariate analyses.

Specific Aim 2: *Determine whether chronic stress is associated with physical activity.*

Hypothesis. *Physical activity will be negatively associated with chronic stress.*

Chronic Stress and Physical Activity

Means for each of the dimensions of chronic stress are presented by Physical Activity level. As shown in Table 11, Financial Stress was higher in the High Activity group than in the Low and Moderate Activity groups. There were no significant differences between groups on any of the other stress measures.

Table 11. Descriptive Statistics for Stress Variables, by Physical Activity Level

	Physical Activity (T2)						
	Low		Moderate		High		
Variables	M	(SD)	M	(SD)	M	(SD)	*p*
Financial Stress	1.80_{a}	(0.67)	1.82_{a}	(0.68)	1.98_{b}	(0.72)	<.001
Life Stress							
Neighborhood Stress	2.12	(0.88)	2.13	(0.87)	2.14	(0.89)	.91
Family	1.86	(0.80)	1.83	(0.81)	1.94	(0.87)	.09
Co-parenting	2.17	(1.36)	2.01	(1.28)	2.16	(1.29)	.08
Partner relationship	1.82	(0.83)	1.77	(0.86)	1.84	(0.85)	.41
Total	1.99	(0.64)	1.93	(0.70)	2.02	(0.67)	.10
Discrimination	2.39	(4.47)	2.42	(4.60)	2.37	(4.44)	.98
Interpersonal Violence	6.32_{ab}	(2.11)	6.17_{a}	(2.14)	6.56_{b}	(2.57)	.05
Perceived Stress	13.29	(6.00)	12.83	(5.58)	13.6	(5.67)	.10

Each measure of chronic stress was then entered along with covariates into a separate multinomial logistic regression model with Physical Activity as the criterion variable. As shown in Table 12, Financial Stress was associated with greater likelihood of High Activity such that each one unit increase in Financial Stress was associated with 1.4 times the odds of High Activity even after controlling for race/ethnicity, income, multiparity, type of residential area, employment, and age. Financial Stress was not significantly associated with odds of Moderate Activity.

Table 12. Multinomial Logistic Regression Model Testing Financial Stress as a Predictor of Physical Activity Categories

	OR for Moderate Activity vs. low activity		OR for High Activity vs. low activity	
Variable	OR	(95 % CI)	OR	(95 % CI)
Unadjusted				
Financial Stress	1.02	(0.85-1.22)	1.42***	(1.20-1.69)
Adjusted				
Financial Stress	1.05	(0.89-1.39)	1.40***	(1.15-1.70)
Race/Ethnicity				
White	Ref.			
Black	0.47***	(0.31-0.75)	0.48***	(0.32-0.72)
Latina	0.50**	(0.31-0.81)	0.53**	(0.34-0.82)
Per capita household income				
Q1	Ref.			
Q2	0.95	(0.59-1.55)	1.38	(0.77-1.94)
Q3	1.34	(0.84-2.14)	1.20	(0.65-1.69)
Q4	0.88	(0.54-1.44)	0.81	(0.52-1.39)
Q5	1.04	(0.60-1.79)	1.10	(0.50-1.55)
Residential area				
Urban	Ref.			
Suburban	0.76**	(0.43-1.85)	0.78	(0.57-1.27)
Rural	0.44***	(0.30-0.64)	0.49***	(0.30-0.71)
Multiparity	0.76	(0.60-1.00)	1.18	(0.89-1.56)
T2 employment				
Not working by choice	Ref.			
Working part-time	1.17	(0.60-1.15)	1.55*	(1.03-2.35)
Working full-time	1.17	(0.74-1.86)	1.73**	(1.20-2.49)
Unemployed	0.82	(0.55-1.24)	0.93	(0.65-1.34)
Age	1.01	(0.98-1.04)	0.96**	(0.93-0.99)

* $p < 0.05$; ** $p < 0.01$; ***$p < .001$

In sum, only one of the nine stress measures was significantly associated with physical activity and the association was contrary to what was hypothesized. Financial Stress was associated with significantly greater odds of High Activity.

Specific Aim 3: *Determine whether personal resilience resources that CCHN studied under the umbrella concept of resilience resources are associated with physical activity.*

Hypothesis. *Individuals with greater personal resilience resources (self-esteem, mastery, optimism, and perceived social support) will report higher levels of physical activity, controlling for race/ethnicity, SES, type of residential area, parity, relationship status, employment status, and age.*

Personal Resilience Resources and Physical Activity

Means and standard deviations for personal resilience resource variables (mastery, self-esteem, optimism, and perceived social support) by physical activity level are shown in Table 13. Group differences on these variables were tested using ANOVA. Only optimism differed significantly across groups, such that participants who were moderately active had higher mean optimism scores than those who were highly active (*M* difference = 0.93 years; $t(718) = 2.85$, $p = .01$). Each measure of resilience was entered along with covariates into a separate multinomial logistic regression model with Physical Activity as the criterion variable. Optimism was not a significant predictor of Physical Activity after controlling for race/ethnicity, income, multiparity, type of residential area, employment, and age, $\chi2(2) = 3.61$, $p = .16$. None of the resilience measures emerged as significant predictors of Physical Activity after statistical control for covariates.

Table 13. Descriptive Statistics for Resilience Variables, by Physical Activity Level (n = 1,581)

	Physical Activity (T2)						
	Low		Moderate		High		
Variable	*M*	(*SD*)	*M*	(*SD*)	*M*	(*SD*)	p
Resilience resource							
T1 Mastery	27.94	(4.06)	28.38	(3.99)	28.01	(3.85)	.15
T1 Self-Esteem	25.90	(2.98)	26.10	(2.97)	25.86	(3.25)	.38
T1 Social Support	83.40	(18.33)	84.73	(17.99)	83.20	(17.44)	.33
T2 Optimism	31.12_{ab}	(4.96)	31.56_{a}	(5.19)	30.63_{b}	(5.42)	.02

Note. Differing subscripts across groups indicate that groups differ at $p < .05$.

Summary of Study 1

This study examined levels and correlates of physical activity in a diverse sample of 1,581 women who were interviewed approximately six months after the birth of a child. Although most participants did not report engaging in regular vigorous or moderate activity, 70% reported walking 3 times per week or more and 65% met the IPAQ criteria for Moderate or High Activity. A number of demographic variables were associated with physical activity level with race/ethnicity, type of residential area, parity, employment status, and age emerging as significant predictors in multivariate analyses.

Contrary to hypotheses, there was little evidence that psychosocial factors contributed to physical activity levels. Of the nine stress variables examined in this study, only Financial Stress was associated with physical activity and the direction of this effect ran counter to hypotheses such that Financial Stress was associated with *greater* likelihood of High Activity. Resilience resources including mastery, self-esteem, optimism, and perceived support were not associated with physical activity and depressive symptoms measured at three time points over the first postpartum year also showed no significant relationship.

Chapter Four: Study 2 Results

Prior to reporting results of analyses testing study hypotheses, descriptive statistics and tests of bivariate associations for key study variables are provided as context for more complex multivariate models.

Sample Characteristics

Demographic characteristics of the sample for these analyses *(n* = 1,206) were previously reported in Table 1. To further characterize the Study 2 sample, Table 14 includes frequencies and descriptive statistics for health status variables including frequencies for clinically diagnosed medical conditions, medication use, and complications of the most recent pregnancy and birth. The most commonly reported medical condition was anemia (35%), followed by hypertension (22%), and asthma (20%). The most frequently used medication was hormonal birth control, which was reported by 39% of participants. Aspirin, statins, antihypertensives and SSRIs were used infrequently in this sample. In the most recent pregnancy, many participants had experienced complications including preterm birth (14%) and gestational hypertension (15%). Thirty-six percent of participants had gained more than 35 pounds in the most recent pregnancy.

Table 15 shows the frequency of various health behaviors in the current sample, including breastfeeding, physical activity, smoking, alcohol use, and sleep. At the time of the second study visit, 21% of the sample was breastfeeding and another 51% had breastfed at some point but had since stopped before their child was 6 months old. At Time 2, most participants were moderately (33%) or highly active (32%), and about 19% percent were current smokers. In addition, 10% were classified as excessive drinkers based on meeting CDC criteria for heavy or binge drinking. Nearly half of the sample (48%) reported sleeping less than 7 hours per night on average during the previous month.

Table 14. Health Status of Sample (n = 1,206)

Variable	*n*	(%)
Self-reported recent illness (% yes)		
T2	153	(13.2)
T3	173	(15.0)
Medical diagnoses (% yes)		
Anemia	417	(34.9)
Hypertension	268	(22.3)
Asthma	241	(20.0)
Type I or II diabetes	82	(6.8)
High cholesterol	79	(6.8)
Kidney problems	69	(5.8)
Heart problems	57	(4.7)
Periodontal/Gum Disease	57	(4.7)
Thyroid problems	56	(4.7)
Tuberculosis	42	(3.5)
Liver disease/hepatitis[a]	23	(1.9)
History of cancer	14	(1.2)
Epilepsy	14	(1.2)
HIV	4	(0.3)
Number of health conditions		
0	500	(30.1)
1	412	(34.2)
2 or more	430	(35.7)
Medication Use (% yes)		
Hormonal Birth Control	458	(38.5)
SSRI or SNRI	49	(4.1)
Steroid/asthma medication	44	(3.7)
Blood pressure medications	29	(2.4)
NSAIDs	27	(2.2)
Diabetes medications	11	(0.9)
Aspirin	6	(0.5)
Statins	2	(0.2)
Complications in most recent pregnancy (% yes)		
Weight gain >35 lbs	298	(36.2)
Indicated/emergency C-section	277	(24.3)
Preterm Birth (<37 weeks)	163	(13.5)
Gestational Hypertension	178	(14.9)
Gestational Diabetes	79	(6.6)
Preeclampsia	76	(7.0)

Table 15. Frequencies for Various Health Behaviors at T2

Variable	*n*	(%)
Breastfeeding (T2)		
Never breastfed	335	(27.9)
Breastfed < 6 mos.	613	(51.0)
Breastfed ≥ 6 mos.	254	(21.1)
Physical Activity		
Low	422	(35.6)
Moderate	390	(32.9)
High	374	(31.5)
Smoking		
Non-smoker	936	(77.7)
Former smoker	45	(16.9)
Current smoker	223	(18.5)
Alcohol use		
Non-drinker	604	(50.2)
Moderate drinker	477	(39.6)
Heavy or binge drinker	144	(10.2)
Hours of sleep per night		
5 or less hours	260	(21.7)
6 hours	317	(26.5)
7 hours	273	(22.8)
8 hours	248	(20.7)
9 or more hours	99	(8.3)
Sleep hours/night *M* (*SD*)	6.64	(1.57)

Adiposity Descriptives

Markers of adiposity including BMI and waist circumference are strongly associated with elevated levels of inflammatory markers including CRP, and detailed descriptive statistics for these variables are reported in Table 16. The mean pre-pregnancy BMI was 27.72 (*SD* = 8.12) for the 860 women who had chart data on pre-pregnancy height and weight. The mean BMI was 30.13 (*SD* = 8.18) at Time 2, and 29.89 (*SD* = 8.12) at Time 3. Notably, a majority of participants were either overweight (26%) or obese (43%) at Time 2, with similar proportions at Time 3. The mean waist circumference was 95.44 cm (*SD* = 17.45) at Time 2 and 95.14 cm (*SD* = 17.93) at Time 3.

Table 16. Anthropometric Characteristics of the Sample

Continuous variables	*M*	(*SD*)	Min	Max	Skew	Kurtosis
BMI (kg/m^2)						
Pre-pregnancy (*n* = 860)	27.72	(7.52)	15.78	63.06	1.25	4.76
T2 (*n* = 1171)	30.01	(8.18)	13.61	61.50	0.96	3.80
T3 (*n* = 1177)	29.89	(8.12)	15.57	58.20	0.92	3.63
Waist circumference (cm)						
T2 (*n* = 1169)	95.44	(17.45)	55.00	185.00	0.66	3.53
T3 (*n* = 1174)	95.15	(17.93)	58.00	185.50	0.65	3.30

Categorical Variables	*n*	(%)
T2 BMI categories		
Normal/underweight (< 25)	368	(31.4)
Overweight (25-29.9)	304	(26.0)
Obese Class I (30-34.9)	229	(19.6)
Obese Class II (35-39.9)	129	(11.0)
Obese Class III (≥40)	141	(12.0)
T3 BMI categories		
Normal/underweight (< 25)	374	(31.8)
Overweight (25-29.9)	302	(25.7)
Obese Class I (30-34.9)	229	(19.5)
Obese Class II (35-39.9)	131	(11.1)
Obese Class III (≥40)	141	(12.0)

Note. BMI = body mass index

CRP Descriptives

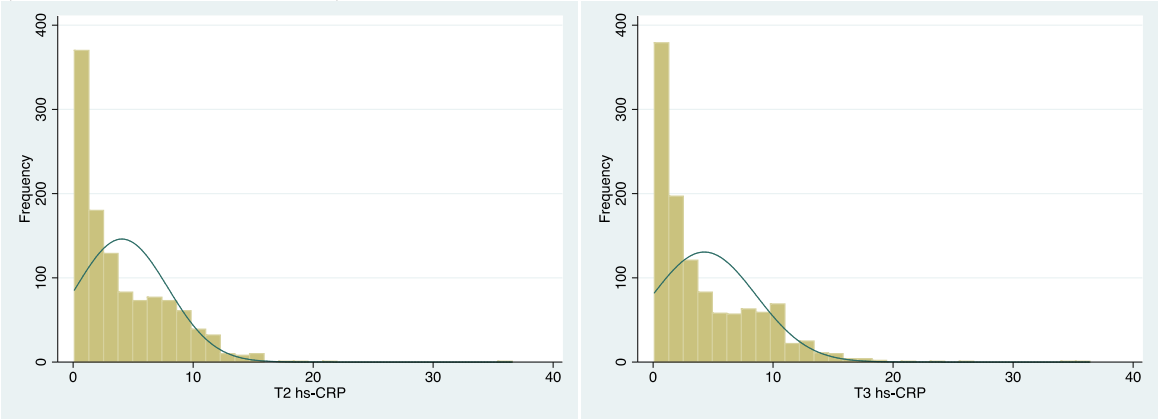
Table 17 provides descriptive statistics for continuous raw and log-transformed ("log CRP") CRP levels at Time 2 and Time 3. As shown in Figure 3, CRP levels ranged from <.1 mg/L to over 36 mg/L and there was marked positive skew in the distributions at T2 and T3. Prior to analyses, values of CRP were natural-log transformed to normalize the distributions.

Table 17. Descriptive Statistics for hs-CRP and Log Transformed hs-CRP at T2 and T3 (n = 1,206)

Variable	*n*	*M*	(*SD*)	*Mdn*	Interquartile range	Min	Max	Skew	Kurtosis
Time 2									
CRP (mg/L)	1149	4.06	(3.82)	2.70	(1.00-6.50)	0.10	36.60	1.48	7.67
CRP (log transformed)	1149	0.83	(1.23)	0.99	(0.00-1.87)	-2.30	3.60	-0.59	2.64
Time 3									
CRP (mg/L)	1169	4.27	(4.32)	2.60	(0.90-6.70)	0.10	36.40	1.67	8.06
CRP (log transformed)	1169	0.80	(1.31)	0.96	(-0.11-1.90)	-2.30	3.59	-0.52	2.43

Note. CRP = C-reactive protein

Figure 3. Histograms showing distribution of T2 and T3 CRP for participants in study sample (T2 n = 1,149; T3 n = 1,169)



Clinical categories. 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 (Pearson, 2003). Table 18 presents the percentage of participants who fell into clinical CRP categories of less than 3 mg/L, 3 to 10 mg/L, and 10 or higher mg/L at each

timepoint. Forty-eight percent of the sample showed evidence of elevated CRP at T2 and 46.4% were elevated at T3.

Table 18. CRP Categories at T2 and T3 (n = 1,206)

	T2 (*n* = 1,149)		T3 (*n* = 1,169)	
Category	*n*	%	*n*	%
CRP < 3	604	52.6	626	53.6
CRP ≥ 3 to 10	448	39.0	406	34.7
CRP ≥ 10	97	8.40	137	11.7

Note. CRP = C-reactive protein

Stability from T2 to 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 ($r = .67$, $p < .001$). The association was slightly stronger for the 858 participants who did not report being ill at either T2 or T3 ($r = .72$, $p < .001$) and smaller for those 261 participants who reported being ill at either one or both time points ($r = .49$, $p < .001$). 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.

Outliers. 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 (Ridker, 2003). However, recent evidence suggests that CRP is clinically useful in predicting CVD risk across a full range of values (Muir, Weir, Alwan, Squire, & Lees, 1999; Ridker & Cook, 2004). Moreover, longitudinal studies have shown that CRP values over 10 often indicate persistent

systemic inflammation and thus may reflect long-term processes rather than transient increases due to acute infection or injury (Hamer, Chida, & Stamatakis, 2010; Ishii et al., 2012).

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. In addition, CRP levels in postpartum women are slightly elevated in comparison to nulliparae (Burlingame, Ahn, & Tang, 2013; Groer et al., 2005; Kuzawa et al., 2013) and "normal" CRP levels for the current population (women of diverse race/ethnicity and predominantly low SES during the first year postpartum) have not been established. 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 predicting continuous CRP outcomes.

Summary of Descriptive Analyses

Several potentially important descriptive findings emerged in examining characteristics of the study sample. First, there were several indicators of poor health status, including diagnoses of chronic conditions or health problems and complications in the prior pregnancy. Second, a number of behavioral risk factors were present including breastfeeding for less than the WHO recommended 6 months (79%), low physical activity (36%), past or current smoking (35%), excessive alcohol use (10%), and inadequate sleep (48%). Third, most participants were either

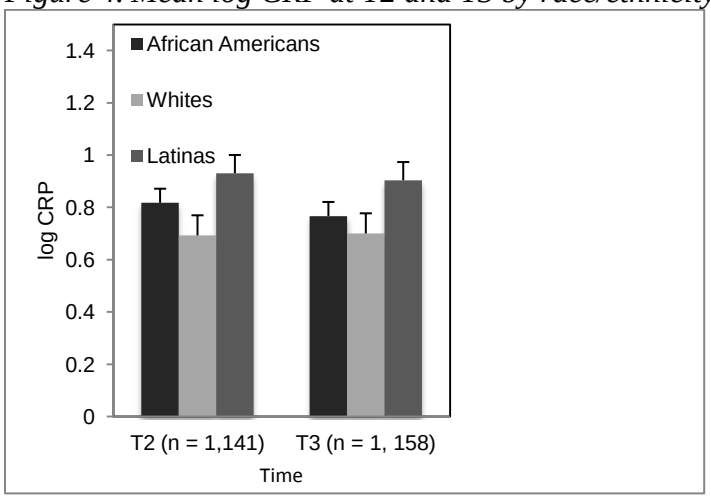
overweight (26%) or obese (43%). Finally, approximately 35% of the sample had CRP concentrations consistent high risk of cardiovascular disease at both T2 and T3. Each of these descriptive findings suggests heightened risk of future health problems in this sample of mothers.

Examining Covariates

In these initial analyses, we examined levels of CRP across a number of covariates including demographic factors, health behaviors, and health status indicators that have well-documented links to markers of inflammation (O'Connor et al., 2009). Demographic variables included race/ethnicity, SES as indexed by income and education, parity, and age. A number of health behaviors including physical activity, sleep, smoking, alcohol consumption, and breastfeeding were considered. Finally, associations of CRP with health status indicators (participant self-report of recent illness, BMI, chronic health conditions, and medication usage) were considered. Relationships between CRP and each of these covariates are described below.

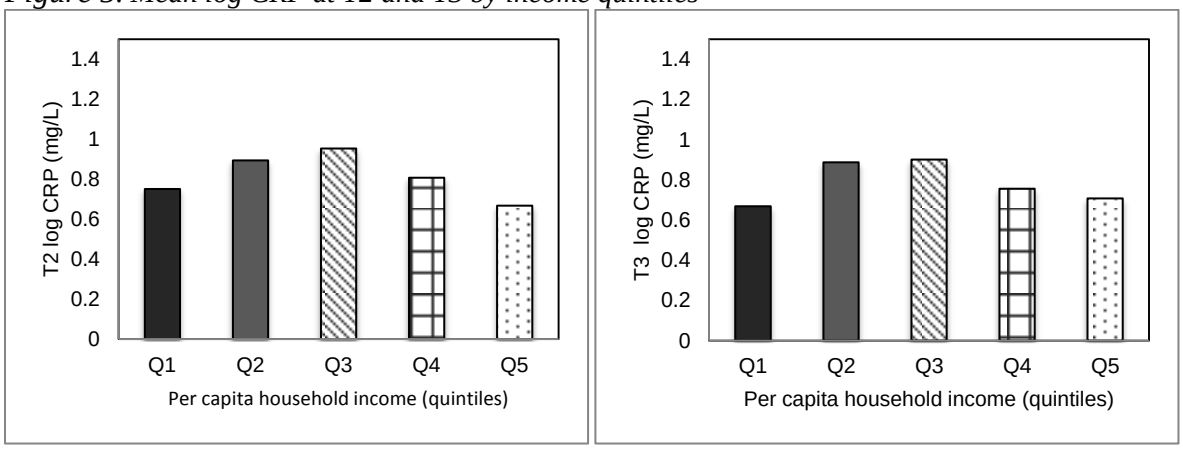
Race/Ethnicity and CRP. Means for T2 and T3 CRP by race/ethnicity are shown in Figure 3. Results of ANOVAs indicated that there was no significant differences in T3 CRP values by race/ethnicity, and the effect was marginally significant at T2, $F(2, 1137) = 2.53$, $p = .08$. Posthoc Tamhane comparisons, which were used because of unequal variances across groups, revealed marginally higher log CRP scores among Latinas ($M = 0.93$, $SD = 1.07$) than White women ($M = 0.69$. $SD = 1.26$), $p = .07$). Chi square analyses were also run with categorical CRP outcome variables (CRP<3 mg/L vs. CRP ≥ 3 mg/L) and there were no statistically significant differences in elevated CRP by race/ethnicity at either time point (all p's > .25).

Figure 4. Mean log CRP at T2 and T3 by race/ethnicity



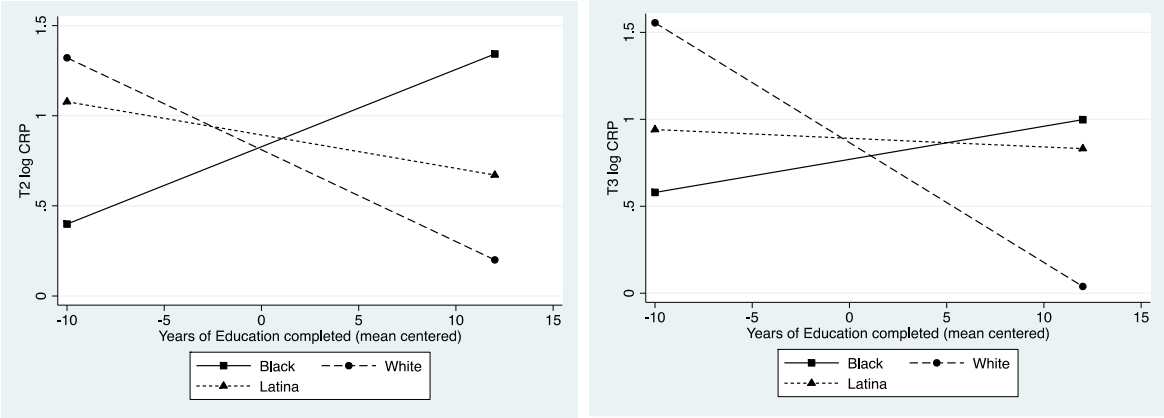
SES and CRP. Associations between SES and log CRP at T2 and T3 were first tested using continuous measures of per capita household income (cost of living adjusted) and years of education completed. The distribution of per capita income had a marked positive skew due to the presence of outliers at the upper end of the distribution, and was winsorized at the top five percent of the distribution. Pearson correlations revealed no significant associations of either income or education with T2 CRP or T3 CRP, though there was a marginally significant negative association between income and T2 CRP (r = -.05, p = .06). To examine possible non-linear effects of SES, associations between CRP and categorical SES indicators (educational attainment and income quintiles) were tested using factorial ANOVA. Results showed evidence of a possible non-linear association between per capita household income and CRP at both time points (see Figures 4), though overall effects did not achieve statistical significance (p's < .15). There were no significant effects of the categorical educational attainment variable on CRP at either time (p's > .50) or no evidence of a non-linear relationship. The continuous variable (years of education completed) was used in all subsequent analyses.

Figure 5. Mean log CRP at T2 and T3 by income quintiles



Interaction terms were entered into multiple regression analyses to determine whether race/ethnicity moderated associations of SES indicators with CRP. Results indicated that associations of income with CRP did not vary significantly by race/ethnicity (*p*'s >.20). There were significant race/ethnicity X education interactions at T2, F (2, 1128) = 3.87, p =.02) and T3, F (2, 1146) = 3.17, p = .04, indicating that education was not uniformly associated with T2 and T3 CRP. As shown in Figures 6 and 7, more education was associated with lower T2 CRP (b = -.05, SE = .03, p < .05) and T3 CRP (b = -.07, SE = .03, p < .01) for White mothers only. Simple slopes for Latinas and African Americans were not significant, though there was a marginally significant association of more education with higher CRP at T2 among African Americans (b = .04, SE = .04, p = .07).

Figure 6. Interaction between education and race/ethnicity predicting T2 and T3 CRP



Adiposity and CRP. Table 19 shows correlations of CRP values with measures of adiposity (BMI and waist circumference) at each time point. Each measure of adiposity was positively correlated with T2 and T3 CRP, and these correlations were highly significant. Moreover, BMI was largely stable between T2 and T3 and correlations between BMI and waist circumference were also strong.

Table 19. Correlations of CRP with Adiposity Measures

	(1)	(2)	(3)	(4)	(5)	(6)
(1) T2 CRP (log)	1					
(2) T3 CRP (log)	.67***	1				
(3) T2 BMI	.52***	.52***	1			
(4) T3 BMI	.51***	.52***	.97***	1		
(5) T2 Waist Circumference	.51***	.52***	.86***	.83***	1	
(6) T3 Waist Circumference	.52***	.53***	.86***	.88***	.87***	1

Notes. CRP = C-reactive protein; BMI = body mass index (kg/m^2)
*$p < 0.05$; ** $p < 0.01$; ***$p < .001$
Pearson *r* values shown

Physical Activity and CRP. In comparing individuals falling into each of the three physical activity categories (low, moderate, and high), ANOVAs indicated no significant differences in mean CRP levels at T2 or T3 (p's > .60). Table 20 shows correlations of CRP values with weekly energy expenditure for vigorous, moderate, walking, and total physical activity. There were no significant correlations (all p's > .30).

Table 20. Correlations of CRP with METs Expenditures for Physical Activity Intensities

	(1)	(2)	(3)	(4)	(5)	(6)
(1) T2 CRP (log)	1					
(2) T3 CRP (log)	.67***	1				
(3) Vigorous METs/week	.00	-.01	1			
(4) Moderate METs/week	.01	-.01	.17***	1		
(5) Walking METs/week	-.01	-.01	.17***	.23***	1	
(6) Total METs/week	-.01	-.03	.71***	.63***	.63***	1

Notes. CRP = C-reactive protein; METs = Metabolic equivalents
*$p < 0.05$; ** $p < 0.01$; ***$p < .001$
Pearson *r* values shown

Other health behaviors and CRP. Separate ANOVAs were used to examine associations of CRP with smoking status (coded as never smoker, former smoker, and current smoker), alcohol use (coded as non-drinker, moderate drinker, and heavy/binge drinker), and breastfeeding status (coded as never breastfed, breastfed but stopped before 6 months post partum, and currently breastfeeding). Smoking status and alcohol use were not associated with T2 CRP or T3 CRP (all *p*'s > .30). Mean log CRP at T3 was lower among women who breastfed for 6 or more months than for those who had never breastfed or who had breastfed for less than 6 months. There were no significant differences between women who had never breastfed and those who had breastfed but stopped before 6 months post partum.

Summary of Relationships Between CRP and Potential Covariates

In sum, relationships between CRP and variables that have been correlated with inflammation in previous studies of different populations than this were examined in the present sample of women during the year following a birth. Greater BMI was associated with higher T2 and T3 CRP in univariate analyses, whereas breastfeeding for at least 6 months was associated with lower CRP. However, race/ethnicity, income, education, and physical activity did not contribute significantly to the prediction of T2 or T3 CRP. Although these variables were not related to CRP in the present sample, they were included in subsequent regression analyses to be conservative and facilitate comparison with other studies that have included these variables as covariates.

Specific Aim 4: *Test the association between chronic stress and elevated CRP.*

Hypothesis*. Higher levels of chronic stress will be associated with higher levels of CRP controlling for income, education, maternal age, parity, health behaviors, medication usage and health conditions.*

Chronic Stress and CRP

Table 21 displays bivariate correlations of T2 and T3 CRP 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.

Table 21. Correlations of T2 and T3 CRP with Stress Measures

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

Notes. Pearson *r* values shown. CRP = C-reactive Protein
*$p < 0.05$; ** $p < 0.01$; ***$p < .001$

The first set of 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.

Multivariate analyses predicting continuous CRP outcomes. Table 22 summarizes the results from multiple linear regression models with T2 CRP as a continuous outcome. There were no significant unadjusted effects of Neighborhood Stress, Family Relationship Stress, Co-Parenting Stress, Partner Relationship Stress, total life stress, perceived stress, discrimination, or interpersonal violence on T2 (all p's > .05) and no significant associations emerged after statistical control 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 CRP.

Table 23 summarizes the results from multiple linear regression models with T3 CRP as a continuous outcome. Results were largely consistent with those described above for T2 CRP. There were no significant unadjusted effects of Neighborhood Stress, Family Relationship Stress, Co-Parenting Stress, Partner Relationship Stress, total life stress, perceived stress, discrimination, or interpersonal violence on T2 (all p's > .20) and no significant associations emerged after statistical control for covariates (all p's > .05). Financial Stress was once again the only one of the nine stress variables that showed a significant unadjusted association with CRP.

Table 22. Summary of Linear Regression Models Predicting T2 C-Reactive Protein

	Unadjusted				Demographics Adjusted				Health Adjusted				Fully adjusted			
Predictor	β	b	SE	p	β	b	SE	p	β	b	SE	p	β	b	SE	p
Financial Stress	.10***	0.17	(0.05)	<.001	.08***	0.15	(0.05)	<.01	.08**	0.14	(0.05)	<.01	.03	0.06	(0.05)	.20
Neighborhood Stress	.02	0.03	(0.05)	.60	.00	0.00	(0.05)	.96	-.01	-0.01	(0.05)	.79	-.01	-0.02	(0.04)	.68
Family Stress	.05	0.09	(0.05)	.94	.05	0.08	(0.05)	.12	.04	0.07	(0.06)	.18	.02	0.03	(0.05)	.54
Co-Parenting Stress	.01	0.01	(0.03)	.72	.01	0.01	(0.03)	.65	.00	0.00	(0.03)	.95	.00	0.00	(0.02)	.88
Partner Relationship Stress	.03	0.05	(0.05)	.29	.03	0.05	(0.05)	.37	.03	0.05	(0.05)	.37	.02	0.04	(0.04)	.41
Total Life Stress	.03	0.06	(0.05)	.28	.03	0.06	(0.06)	.30	.02	0.04	(0.06)	.46	.01	0.01	(0.05)	.85
Perceived Stress	-.02	0.00	(0.01)	.53	-.02	0.00	(0.01)	.51	-.02	0.00	(0.01)	.47	-.02	0.00	(0.01)	.56
Discrimination	-.01	-0.02	(0.08)	.84	-.03	-0.07	(0.08)	.37	-.04	-0.09	(0.08)	.25	-.01	-0.02	(0.07)	.77
IPV	.06	0.15	(0.08)	.06	.06	0.14	(0.08)	.06	.05	0.12	(0.08)	.11	.04	0.09	(0.07)	.16

*$p < 0.05$; ** $p < 0.01$; ***$p < .001$

Table 23. Summary of Linear Regression Models Predicting T3 C-Reactive Protein

	Unadjusted				Demographics Adjusted				Health Adjusted				Fully adjusted			
	β	b	SE	p	β	b	SE	p	β	b	SE	p	β	b	SE	p
Financial Stress	.09***	0.18	(0.06)	<.001	.10***	0.18	(0.06)	<.001	.09**	0.17	(0.06)	<.01	.05*	0.10	(0.05)	<.05
Neighborhood Stress	.00	0.00	(0.05)	.93	.00	-0.01	(0.05)	.92	.00	-0.01	(0.05)	.90	.00	0.00	(0.04)	.94
Family Stress	.03	0.05	(0.06)	.36	.03	0.06	(0.06)	.29	.02	0.03	(0.06)	.55	-.01	-0.02	(0.05)	.72
Co-Parenting Stress	.01	0.01	(0.03)	.66	.02	0.02	(0.03)	.59	.00	0.00	(0.03)	.99	.02	-0.01	(0.03)	.74
Partner Relationship Stress	.04	0.06	(0.05)	.24	.03	0.06	(0.05)	.26	.03	0.05	(0.05)	.33	.02	0.03	(0.05)	.46
Total Life Stress	.03	0.05	(0.06)	.39	.03	0.06	(0.06)	.36	.02	0.03	(0.06)	.62	.03	-0.01	(0.05)	.77
Perceived Stress	-.03	-0.01	(0.01)	.33	-.03	-0.01	(0.01)	.31	-.05	-0.01	(0.01)	.13	-.05	-0.01	(0.01)	.07
Discrimination	.00	-0.05	(0.08)	.49	-.04	-0.10	(0.08)	.22	-.02	-0.12	(0.08)	.15	-.02	-0.06	(0.07)	.38
Interpersonal Violence	.02	0.06	(0.08)	.44	.02	0.05	(0.08)	.50	.01	0.03	(0.08)	.76	.00	-0.01	(0.07)	.89

*$p < 0.05$; ** $p < 0.01$; ***$p < .001$

Table 24 displays results for the unadjusted and adjusted regression models using Financial Stress to predict T2 CRP. Financial Stress remained significantly associated with T2 CRP after controlling 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. In the final model, Latino ethnicity, higher BMI, and use of birth control were associated with higher CRP and smoking was associated with lower CRP.

Table 24. Standardized Regression Coefficients of Multivariate Regressions Predicting T2 Log CRP (n = 1,139)

Variables	Model 1	Model 2	Model 3	Model 4
Financial Stress	0.10**	0.08**	0.08**	0.04
Race/ethnicity				
African American race		0.07	0.03	-0.04
Latino ethnicity		0.07	0.06	0.08*
Household Income (Quintiles)				
Q2		0.04	0.04	0.01
Q3		0.06	0.06	0.03
Q4		0.01	0.01	-0.02
Q5		-0.03	-0.03	-0.01
Education		-0.03	-0.03	0.03
Age		0.11**	0.12**	0.05
Parity		-0.07	-0.09	-0.05
Moderately/Highly active			-0.05	-0.05
Sleep (avg. hours)			-0.02	-0.02
Current smoker			-0.09**	-0.07**
Breastfed > 6 mos.			-0.10**	-0.04
Excessive drinker			0.01	0.00
Recent Illness			0.02	0.03
Health conditions			0.08*	0.04
Medications (1 = yes)				
Hormonal Birth Control			0.02	0.06*
Antihypertensives			0.06	0.04
Antidepressants			-0.03	-0.03
Steroids			0.00	-0.01
NSAIDS			-0.00	-0.03
Current BMI				0.53***
Adjusted R^2	0.01	0.03	0.06	0.31

* $p<0.05$; ** $p<0.01$

Table 25 displays results for the unadjusted and adjusted regression models using Financial Stress to predict T3 CRP. Financial Stress remained significantly associated with higher T3 CRP after controlling for race/ethnicity, income, education and age in Model 2, and after controlling for health-related covariates in Model 3. Further adjustment for BMI attenuated the coefficient but the association remained significant ($p < .05$). In the final model, higher BMI and use of birth control and antihypertensive medications were associated with higher CRP and African American race and smoking were associated with lower CRP.

Table 25. Standardized Regression Coefficients of Multivariate Regressions Predicting T3 Log CRP (n = 1,156)

Variables	Model 1	Model 2	Model 3	Model 4
Financial Stress	0.09**	0.10**	0.10**	0.04*
Race/ethnicity				
African American race		0.02	0.01	-0.09**
Latino ethnicity		0.04	0.06	0.07
Household Income (Quintiles)				
Q2		0.05	0.04	0.01
Q3		0.08*	0.07	0.04
Q4		0.04	0.02	0.01
Q5		0.04	0.03	0.06
Education		-0.03	-0.01	0.03
Age		0.02	0.04	-0.04
Multiparity		-0.08*	-0.07*	-0.04
Moderate/High Activity			-0.04	-0.04
Sleep (avg. hours)			-0.03	-0.04
Current smoker			-0.06	-0.07*
Breastfed > 6 mos.			-0.16**	-0.10**
Excessive drinker			-0.00	-0.00
Recent Illness			0.11**	0.14**
Health conditions			0.03	0.00
Medications (1 = yes)				
Hormonal Birth Control			0.02	0.06*
Antihypertensives			0.09**	0.06*
Antidepressants			0.00	-0.01
Steroids			0.02	0.01
NSAIDS			0.01	-0.01
Current BMI				0.56**
Adjusted R^2	0.01	0.02	0.07	0.35

* $p<0.05$; ** $p<0.01$

Multivariate analyses predicting chronic inflammation. Next, we sought to better characterize the 35% of women who demonstrated chronically elevated levels of CRP (≥3 mg/L at both time points). A series of logistic regression models tested unadjusted and adjusted associations between each of the stress variables and chronic inflammation, defined as having CRP concentrations ≥ 3 at both T2 and T3. As shown in Table 26, Financial Stress, Family Stress, and Total Life Stress were associated with significantly greater odds of chronically elevated CRP in the unadjusted model (Model 1). After adding demographic covariates (Model 2), only Financial Stress was significantly associated with greater odds of chronic CRP elevation. The statistical significance of this association was maintained after controlling for health-related covariates in Model 3, but was attenuated to non-significance after T2 and T3 BMI were entered in Model 4. Tables 27, 28, and 29 show all unadjusted and adjusted regression models for the three stress variables that showed significant unadjusted associations with chronic inflammation.

Table 26. Summary of Logistic Regression Models Predicting Chronic CRP Elevation (n = 1,112)

	Model 1[a]		Model 2[b]		Model 3[c]		Model 4[d]	
	T2 CRP mg/L		T2 CRP mg/L		T2 CRP mg/L		T2 CRP mg/L	
	OR	95% CI	OR	95% CI	OR	95% CI	3-10	≥ 10
Financial Stress	1.32**	(1.11- 1.57)	1.31**	(1.10- 1.57)	1.32**	(1.09- 1.60)	1.20	(0.96- 1.50)
Neighborhood Stress	1.04	(0.88- 1.23)	0.99	(0.83- 1.18)	0.98	(0.82- 1.17)	0.98	(0.79- 1.21)
Family Stress	1.22*	(1.02- 1.46)	1.19	(0.99- 1.43)	1.15	(0.95- 1.40)	1.09	(0.87- 1.37)
Co-Parenting Stress	1.10	(1.00- 1.21)	1.08	(0.98- 1.19)	1.06	(0.96- 1.17)	1.06	(0.94- 1.20)
Partner Relationship Stress	1.12	(0.94- 1.32)	1.09	(0.91- 1.30)	1.08	(0.90- 1.30)	1.07	(0.87- 1.32)
Total Life Stress	1.25*	(1.04- 1.50)	1.20	(0.98- 1.46)	1.18	(0.96- 1.44)	1.11	(0.87- 1.42)
Perceived Stress	1.00	(0.98- 1.02)	1.00	(0.97- 1.02)	1.00	(0.97- 1.02)	1.00	(0.97- 1.02)
Discrimination	0.86	(0.67- 1.12)	0.78	(0.59- 1.02)	0.76	(0.58- 1.01)	0.84	(0.61- 1.16)
Interpersonal Violence	1.20	(0.93- 1.55)	1.19	(0.92- 1.55)	1.13	(0.86- 1.48)	1.10	(0.80- 1.51)

Note: Chronic CRP elevation defined as CRP values ≥3 mg/L at both T2 and T3.
[a]Adjusted for demographic covariates (race/ethnicity, income, education, current age, and parity)
[b]Adjusted for demographic covariates + health behaviors (physical activity, smoking, sleep, breastfeeding, and alcohol use)
[c]Adjusted for demographic covariates + health behaviors + health conditions (recent illness, chronic conditions, and medication)
[d]Adjusted for demographic covariates + health behaviors + health conditions + T2 BMI + current BMI
* $p<0.05$; ** $p<0.01$

As shown in Table 27, greater Financial Stress remained significantly associated with increased odds of chronic inflammation after controlling for race/ethnicity, SES, age and parity in Model 2, and after controlling for health-related covariates in Model 3. Further adjustment for BMI attenuated the coefficient to non-significance. In the final model, multiparous women and women who had breastfed for at least 6 months had lower odds of chronic CRP elevation, whereas chronic health conditions and higher BMI were associated with greater odds.

Table 27. Logistic Regressions Using Financial Stress to Predict Chronic CRP Elevation (n = 1,112)

	Odds Ratio			
	Model 1	Model 2	Model 3	Model 4
Financial Stress	1.32**	1.31**	1.32**	1.20
Race/ethnicity				
African American race		1.33	1.17	0.63
Latino ethnicity		0.95	0.92	0.93
Household Income (Quintiles)				
Q2		1.15	1.07	0.98
Q3		1.34	1.33	1.05
Q4		0.82	0.82	0.60
Q5		0.78	0.74	0.80
Education		0.97	0.98	1.04
Age		1.03*	1.04*	1.01
Parity		0.60**	0.60**	0.58**
Moderately/Highly active			0.86	0.93
Sleep (avg. hours)			1.01	0.99
Current smoker			0.73	0.63
Breastfed > 6 mos.			0.42**	0.49**
Excessive drinker			1.05	0.93
Recent Illness (T2)			1.07	1.21
Recent Illness (T3)			1.05	1.21
Health conditions			1.28**	1.30*
Medications (1 = yes)				
Hormonal Birth Control			0.92	1.19
Antihypertensives			2.68*	2.04
Antidepressants			0.88	0.76
Steroids			0.76	0.60
NSAIDS			1.72	1.35
T2 BMI				1.13**
T3 BMI				1.06

Note: Chronic CRP elevation defined as CRP values ≥3 mg/L at both T2 and T3.
* $p<0.05$; ** $p<0.01$

As shown in Table 28, Total Life Stress score was no longer significantly associated with likelihood of chronic inflammation after controlling for race/ethnicity, SES, age and parity in Model 2. In the final model, African American women, multiparous women and women who had breastfed for 6 months or more had lower odds of chronic inflammation, whereas chronic health conditions and higher BMI were associated with greater odds of chronic inflammation.

Table 28. Logistic Regressions Using Total Life Stress to Predict Chronic CRP Elevation (n = 1,073)

	Odds Ratio (95% CI)			
	Model 1	Model 2	Model 3	Model 4
Total Life Stress	1.25*	1.20	1.18	1.11
Race/ethnicity				
African American race		1.28	1.03	0.60*
Latino ethnicity		0.97	0.91	0.95
Household Income (Quintiles)				
Q2		1.20	1.18	1.04
Q3		1.35	1.25	1.04
Q4		0.84	0.75	0.59
Q5		0.72	0.68	0.72
Education		0.98	1.00	1.04
Age		1.04*	1.03	1.01
Parity		0.63**	0.61**	0.58**
Moderately/Highly active			0.91	0.93
Sleep (avg. hours)			1.00	0.99
Current smoker			0.74	0.63
Breastfed > 6 mos.			0.39**	0.49**
Excessive drinker			0.99	0.93
Recent Illness (T2)			1.05	1.21
Recent Illness (T3)			1.07	1.21
Health conditions			1.30**	1.30*
Medications (1 = yes)				
Hormonal Birth Control			0.91	1.19
Antihypertensives			2.52*	2.04
Antidepressants			0.92	0.76
Steroids			0.73	0.60
NSAIDS			1.81	1.35
T2 BMI				1.13**
T3 BMI				1.06

Note: Chronic CRP elevation defined as CRP values ≥3 mg/L at both T2 and T3.
* $p<0.05$; ** $p<0.01$

As shown in Table 29, Family Stress was no longer significantly associated with likelihood of chronic inflammation after controlling for race/ethnicity, income, education, age and parity in Model 2. In the final model, African American women, multiparous women and women who had breastfed for 6 months or more had lower odds of chronic inflammation, whereas chronic health conditions and higher BMI were associated with greater odds of chronic inflammation.

Table 29. Logistic Regressions Using Family Stress to Predict Chronic CRP Elevation (n = 1,086)

	Odds Ratio (95% CI)			
	Model 1	Model 2	Model 3	Model 4
Family Stress	1.22*	1.19	1.15	1.09
Race/ethnicity				
African American		1.28	1.04	0.60*
Latina		1.00	0.95	1.00
Household Income (Quintiles)				
Q2		1.18	1.17	1.03
Q3		1.33	1.25	1.05
Q4		0.81	0.72	0.60
Q5		0.72	0.70	0.76
Education		0.98	1.00	1.04
Age		1.03*	1.03	1.00
Parity		0.63**	0.61**	0.58**
Moderately/Highly active			0.90	0.93
Sleep (avg. hours)			0.99	0.99
Current smoker			0.76	0.62*
Breastfed > 6 mos.			0.42**	0.52**
Excessive drinker			1.01	0.98
Recent Illness (T2)			1.10	1.26
Recent Illness (T3)			1.09	1.24
Health conditions			1.30**	1.28*
Medications (1 = yes)				
Hormonal Birth Control			0.93	1.20
Antihypertensives			2.57*	2.05
Antidepressants			0.90	0.75
Steroids			0.73	0.61
NSAIDS			1.77	1.31
T2 BMI				1.13**
T3 BMI				1.05

Note: Chronic CRP elevation defined as CRP values ≥3 mg/L at both T2 and T3.
* $p<0.05$; ** $p<0.01$

Summary of Results for Chronic Stress and CRP

The purpose of these analyses was to examine associations of several indicators of chronic stress with continuous CRP levels at T2 and T3, as well as presence or absence of chronic inflammation. In multivariate analyses, Financial Stress was the only one of the nine stress variables associated with these outcomes. Financial Stress was related to higher CRP and greater likelihood of chronic inflammation even after controlling for a number of demographic and health-related covariates, but the effect was not robust to the inclusion of BMI in models which was a powerful predictor of CRP.

Specific Aim 5: *Test multivariate models of the combined influence of chronic stress, personal resilience resources, and physical activity on CRP.*

Hypothesis 5a: *Chronic stress will be more strongly related to higher levels of CRP among inactive women but weakly or not associated with CRP among women who are physically active. Psychological resilience resources will also be explored as a potential moderator of the association between chronic stress and CRP.*

Moderation Analyses

Although most of the stress variables were not related to CRP outcomes, additional analyses explored the possibility that stress is associated with CRP in certain vulnerable individuals. In particular, study hypotheses proposed that stress would be associated with inflammation only among individuals who were physically inactive. A series of multiple regression analyses tested whether the effects of the stress variables on CRP were moderated by physical activity level. Each regression model included a set of two terms coding the interaction between the three-level physical activity categorical variable (represented by two dummy coded variables) and a given linear stress variable. *F*-change statistics were then computed to test the statistical significance of the overall interaction step in predicting either T2 log CRP, T3 log CRP, or chronic inflammation.

Table 30 shows correlations between the stress variables and CRP at T2 and T3 for each physical activity level. Although the pattern of correlations suggests that the association between Financial Stress and CRP was strongest among those who were moderately or highly active, the interaction was not statistically significant in multiple regression analyses predicting T2 CRP, T3 CRP, or chronic inflammation (all p's > .50). There were no statistically significant interactions between physical activity level and any of the stress variables.

Table 30. Zero-Order Correlations Between Stress Variables and Log Transformed CRP According to Physical Activity Level

	T2 log CRP			T3 log CRP		
	Low	Moderate	High	Low	Moderate	High
	(n = 401)	(n = 386)	(n = 366)	(n = 411)	(n = 373)	(n = 354)
Financial Stress	.03	.15**	.12*	.07	.12**	.10
Neighborhood Stress	.04	.03	-.01	.03	.04	-.05
Family Stress	.03	.09	.05	.07	.05	-.01
Co-Parenting Stress	.00	.03	.00	.05	.06	-.06
Partner Relationship Stress	-.01	.06	.06	.04	.04	.04
Total Life Stress	.02	.07	.03	.06	.08	-.05
Perceived Stress	-.05	.02	-.02	-.03	-.03	-.03
Discrimination	.07	-.05	-.05	.03	-.03	-.08
Interpersonal Violence	.05	.09	.08	-.02	.08	.02

*p < 0.05; ** p < 0.01; ***p < .001
Pearson r values shown

Another series of multiple regression analyses examined whether personal resilience resources moderated associations between stress variables and CRP outcomes. Before running these models, we assessed if it was empirically justifiable to combine the correlated resilience resource variables in order to reduce the number of interactions tested and avoid Type I error inflation. Based on published research (Rini, Dunkel Schetter, Wadhwa, & Sandman, 1999; Taylor et al., 2006) and theory (Dunkel Schetter and Dolbier, 2011), a confirmatory factor analysis was conducted with mastery, self-esteem, optimism, and perceived support scores. The measurement model provided a good fit to the data, and each of the four factor loadings was positive and statistically significant. Thus, a Personal Resources

Composite score was constructed by standardizing and summing across the four components and this composite score was used in testing the interaction models. Table 30 summarizes these regression analyses by providing coefficients and significance levels for the interaction terms entered into each regression models. There were no significant interactions between the resilience resources composite and any of the nine stress variables.

Table 31. Summary of Stress and Personal Resilience Resource Effects on T2 and T3 log CRP and Chronic Inflammation

	Predicting T2 log CRP			Predicting T3 log CRP			Predicting Chronic Inflammation		
	b	*se*	*p*	*b*	*se*	*p*	*b*	*se*	*p*
Financial Stress x Resilience	0.03	0.03	.17	0.04	0.03	.18	0.08	0.06	.20
Neighborhood Stress x Resilience	0.02	0.02	.54	0.00	0.03	.87	-0.01	0.06	.87
Family Stress x Resilience	0.02	0.02	.35	-0.01	0.03	.78	-0.03	0.06	.53
Co-Parenting Stress x Resilience	0.00	0.02	.89	0.00	0.02	.97	0.02	0.03	.35
Partner Relationship Stress x Resilience	0.00	0.02	.99	-0.01	0.03	.76	0.05	0.04	.30
Total Life Stress x Resilience	0.00	0.03	.98	0.00	0.03	.90	0.00	0.01	.63
Perceived Stress x Resilience	0.00	0.00	.77	0.00	0.00	.69	0.00	0.01	.63
Discrimination x Resilience	-0.02	0.04	.69	0.02	0.04	.73	0.02	0.07	.81
Interpersonal Violence x Resilience	0.01	0.04	.85	-0.06	0.05	.82	-0.05	0.07	.47

Note: Chronic CRP elevation defined as CRP values ≥3 mg/L at both T2 and T3. CRP = C-reactive protein

Hypothesis 5b. *An alternative hypothesis is that chronic stress influences inflammation indirectly via lower levels of physical activity. That is, lower levels of physical activity may explain the relationship between stress and CRP. I will also test this model to determine whether physical activity mediates the association between chronic stress and CRP levels, and compare this mediation model to the stress moderation model.*

As indicated above, the only positive predictive association between stress and the outcome variables in multivariate analyses was that reported for Financial Stress and CRP. Physical activity was not associated with CRP in bivariate analyses or multivariate analyses. Moreover, none of the stress variables were independently related to physical activity. Thus, the proposed mediation model was not tested. However, because the associations of Financial Stress and CRP (T2, T3, and chronic) were

attenuated with the addition of BMI to linear and logistic models, additional analyes were performed to examine adiposity as a potential mediator between Financial Stress and CRP.

Structural Equation Modeling

Structural equation modeling using EQS 6.2 was undertaken to further evaluate the relationships between resilience resources, financial stress, adiposity, and inflammation. In the hypothesized model, greater resilience resources are associated with lower financial stress, which in turn predicts more adiposity, which then leads to chronic CRP elevation. Four latent variables were developed: resilience resources, financial stress, adiposity, and CRP. The structural model included a bidirectional path between resilience resources and financial stress, which were treated as exogenous variables. The model also included a directional path leading from financial stress to adiposity, and a directional path leading from adiposity to CRP. Table 32 shows intercorrelations among the indicators of each of the factors as well as covariates.

Because of missing data for several stress variables and covariates, full information maximum likelihood estimation was used to test the hypothesized model (Bentler, 2006). To evaluate model fit, three fit indices were computed and examined: χ^2, root mean squared error of approximation (RMSEA), and the comparative fit index (CFI). The χ^2 statistic provides a global test of exact fit, with good fit indicated by a non-significant χ^2 (or a signficance level between 0.01 and 0.05 for acceptable fit; Schermelleh-Engel, Moosbrugger, & Müller, 2003) and smaller χ^2 values and larger p-values demonstrating better model fit. The RMSEA provides an estimate of the average size of the residual, adjusted for degrees of freedom; good fit is indicated by a RMSEA of 0.06 or smaller (Hu & Bentler, 1999). The CFI is an index of relative fit as compared to the null model and values, which range from 0 to 1, with good fit demonstrated at values of 0.95 or greater (Hu & Bentler, 1999). Modification indices (the Wald and Lagrange multiplier tests; Chou & Bentler, 1990) were consulted for suggested modifications to improve model fit.

Examination of Mardia's coefficient demonstrated violation of the assumption of multivariate normality in this sample. Values of Mardia's coefficient greater than 3 indicate nontrivial positive kurtosis and modeling statistics may be affected at values greater than 5, 6, or more (Bentler, 2006). Because the value for Mardia's coefficient for this analysis was 10.31, cases with the largest contributions to normalized multivariate kurtosis were examined. There were a number of cases that were much larger than the normalized estimate and no recording errors were detected in these cases. Therefore, all subsequent analyses were conducted using maximum likelihood and robust methods (Method=FIML, ROBUST) and values for the Yuan-Bentler scaled χ^2, which performs well under non-normal conditions, are reported below.

Measurement model. An initial confirmatory factor analysis was conducted with the four latent contructs. Perceived support, mastery, and optimism served as indicators of resilience resources. Four sets of variables were treated as indicators of financial stress. The five items used to compute the previously described Financial Stress index were grouped into three parcels by combining items with similar instruction sets and/or response scales. Pregnancy financial stress was scored as the mean of the two items that assessed worries about basic needs and money during pregnancy. Episodic financial stress was measured with a single item assessing the occurrence and impact of serious problems with money over the past year. Financial difficulties/worries includes the mean of the two items assessing difficulty with paying bills and worries about expenses and bills exceeding income. An additional 2-item measure of food insecurity was also included as an indicator of financial stress. T2 BMI and waist circumference were grouped to form an adiposity factor, and T2 and T3 CRP values served as indicators of the CRP factor variable.

This measurement model fit the data well, Yuan-Bentler $\chi^2(38) = 84.71$, $p < .001$, CFI = 0.98, RMSEA = 0.033 (90% confidence interval [CI] 0.024-0.042). As shown in Figure 7, standardized factor loadings for the observed variables on their respective latent constructs were all greater than .50

and highly significant (p's < .0001). There was a significant negative correlation between Resilience Resources and Financial Stress (r = -.56, p <.001) and positive correlations between Financial Stress and Adiposity (r = .12, p = .001), Financial Stress and CRP (p < .01), and CRP and Adiposity (r = .68, p < .001). The Resilience Resources factor was not correlated with Adiposity or CRP.

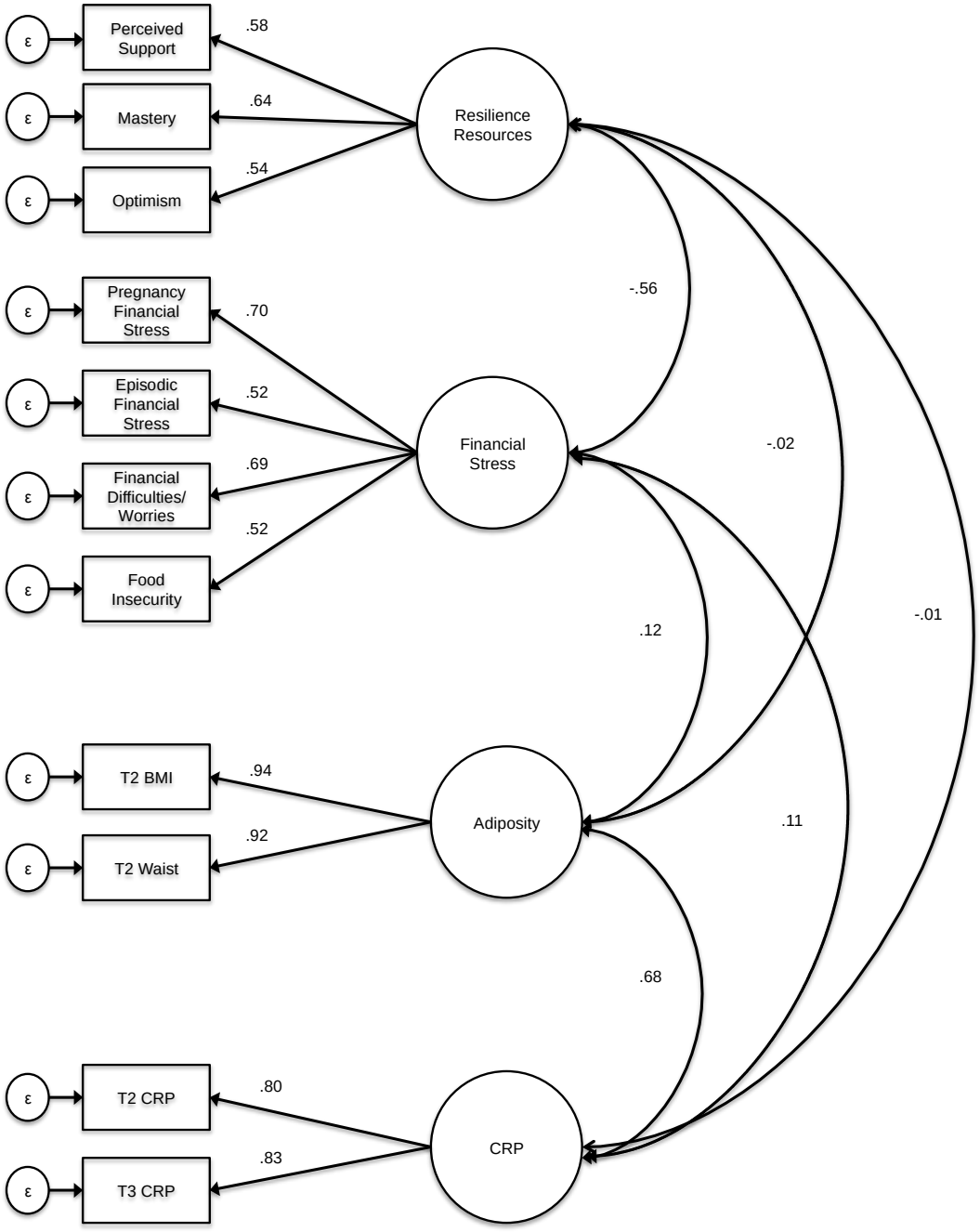
Table 32. Intercorrelations of Study Variables in Structural Equation Model

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)
Resilience																				
(1) Mastery	1																			
(2) Optimism	.38***	1																		
(3) Social Support	.36***	.29***	1																	
Financial Stress																				
(4) Pregnancy Stress	-.25***	-.20***	-.29***	1																
(5) Money Problems	-.08**	-.12***	-.15***	.39***	1															
(6) Financial Worries	-.24***	-.18***	-.23***	.46***	.38***	1														
(7) Food Insecurity	-.18***	-.18***	-.25***	.36***	.22***	.38***	1													
Adiposity																				
(8) T2 BMI	.01	-.02	-.02	.09**	.12***	.05	.06*	1												
(9) T2 Waist	-.02	-.01	-.03	.08**	.07**	.04	.05	.86***	1											
CRP																				
(10) T2 CRP (log)	.01	-.03	-.03	.09**	.08**	.07*	-.01	.51***	.50***	.51***	1									
(11) T3 CRP (log)	.03	-.02	.00	.07*	.06*	.09**	-.01	.53***	.52***	.53***	.67***	1								
Covariates																				
(12) African American Race	.10***	-.05	-.03	-.03	.10***	-.13***	.11***	.18***	.08**	.01	-.01	1								
(13) Latina Ethnicity	-.17***	.02	-.09**	.07*	-.04	.19***	.00	-.09**	-.03	.05	.05	-.59***	1							
(14) Income	.17***	.16***	.10***	-.19***	-.11***	-.17***	-.22***	-.10***	-.08**	-.02	-.01	-.20***	-.12***	1						
(15) Education (years)	.27***	.22***	.17***	-.07*	.01	-.12***	-.18***	-.09***	-.06*	-.03	-.04	-.08**	-.36***	.45***						
(16) Age	.08**	.17***	.02	-.01	.02	.07**	-.11***	-.01	.04	.05	-.01	-.29***	-.04	.40***	.46***	1				
(17) Multiparity	-.09**	-.03	-.04	.04	.07*	.13***	.05	-.01	.00	-.03	-.06	-.02	.04	-.11***	-.05	.28***	1			
(18) Breastfeeding	-.02	.10***	-.01	-.01	-.03	.09***	-.03	-.14***	-.12***	-.06*	-.12***	-.25***	.11***	.16***	.16***	.26***	.07*	1		
(19) Smoking	-.08**	-.18***	-.06*	.14***	.08**	.05	.13***	.07	.06**	-.06**	-.03	.12***	-.16***	-.16***	-.13***	-.12***	-.04	-.16***	1	
(20) Chronic Conditions	.02	-.09***	-.03	.10***	.11***	.06	.05	.09**	.06*	.08**	.04	.12***	-.18***	.03	.07	.05	.05	-.08***	.11***	
(21) Birth Control	.02	-.02	.05	-.03	-.01	-.01	-.02	-.06	-.09**	.02	.05	.10***	-.03	-.04	-.08*	-.14***	-.05	-.13***	-.04	-.01

*$p < 0.05$; ** $p < 0.01$; ***$p < .001$

Pearson *r* values shown

Figure 7. Measurement model for latent factors: Resilience Resources, Financial Stress, Adiposity and CRP. Standardized coefficients are presented.



Test of hypothesized structural equation model. The hypothesized model was first tested without inclusion of covariates. This resulted in a model with acceptable fit with the exception of the χ^2 test, which indicated misspecification, χ^2 (41) = 87.90, $p < 0.001$, CFI = 0.986, RMSEA = 0.034 (90% CI 0.025-0.042). However, even a model with very good fit to the data can be expected to result in a statistically significant chi-square given the large sample size. Nonetheless, Lagrange Multiplier tests were consulted to determine if there were any additional paths that should be added to the model, but none of the suggested paths was theoretically justifiable.

As shown in Figure 8, there was a significant positive association between resilience resources and financial stress. Greater financial stress predicted greater adiposity, which in turn predicted greater CRP. Overall, the model explained 1% of the variance in adiposity, and 46% of the variance in CRP. Estimated parameters were decomposed into direct and indirect effects to assess the influence of Financial Stress on CRP via Adiposity. As hypothesized, financial stress had a significant indirect effect on CRP via Adiposity ($\beta = .15$, $p = .002$).

Estimates were recalculated to examine whether inclusion of covariates would alter the findings. Race/ethnicity, income, education, age, parity, chronic conditions, smoking, and breastfeeding were added as predictors of adiposity and CRP. The fit of the resulting model was acceptable, Yuan-Bentler χ^2 (104) = 389.90, $p < 0.001$, CFI = 0.939, RMSEA = 0.048 (90% CI 0.043-0.053). There were a number of non-significant paths from covariates, and the Wald test was used to identify paths that could be dropped from the model to improve fit. Paths from several covariates (Latina ethnicity, income, multiparity, smoking, and chronic conditions) to adiposity were dropped. In addition, paths from Latina ethnicity, income, education, age, multiparity, breastfeeding and chronic conditions to Inflammation were dropped. The model was re-estimated, resulting in a model with slightly better fit, Yuan-Bentler χ^2 (87) = 349.42, $p < 0.001$, CFI=0.942, RMSEA=0.05 (90% CI 0.045-0.056).

The final trimmed model is shown in Figure 9. In this model, there was a significant positive association between resilience resources and financial stress. Greater Financial Stress predicted greater adiposity, net of race/ethnicity, income, education, parity, hormonal birth control use, breastfeeding, smoking, and chronic conditions. Adiposity in turn predicted greater inflammation net of covariates. The indirect effect of financial stress on CRP via adiposity remained significant after controlling for covariates, ($\beta = .14$, $p = .003$).

Figure 8. Structural equation model predicting chronic inflammation (n = 1,206). All path coefficients are standardized. Yuan-Bentler scale χ^2 (41)=87.90, p<0.001, CFI=0.986, RMSEA=0.034 (90% CI 0.025-0.042)

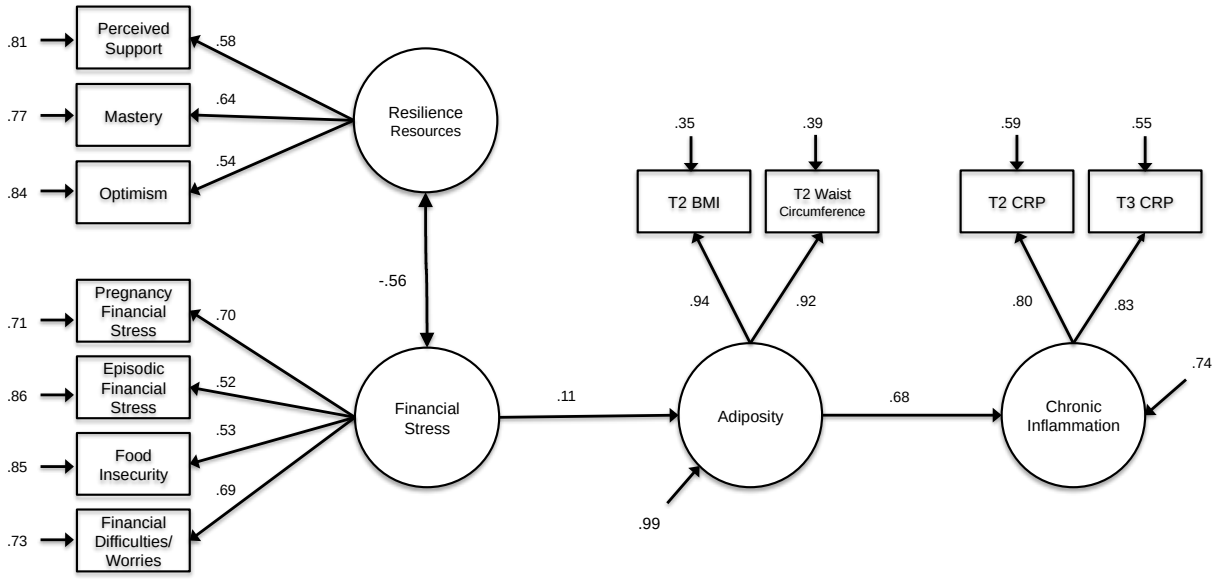
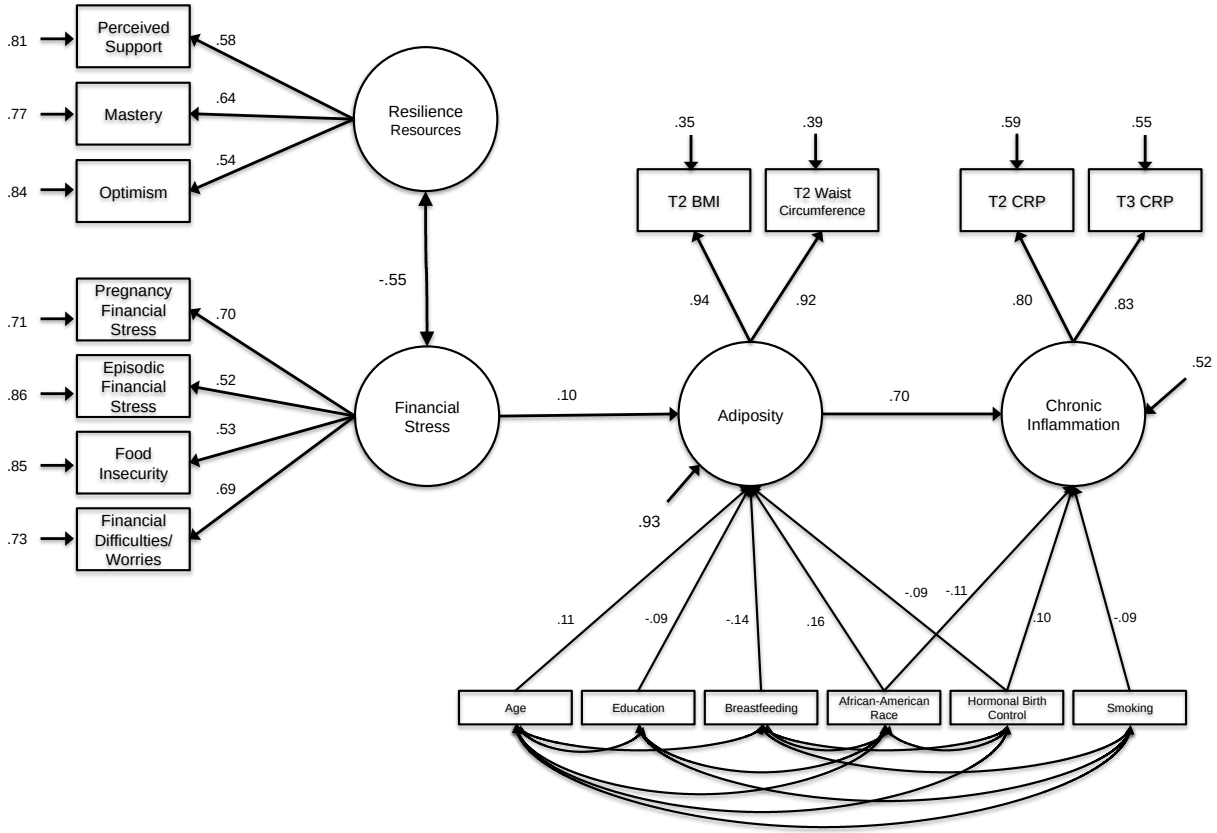


Figure 9. Structural equation model predicting chronic inflammation with significant covariates included (n = 1,206). All path coefficients are standardized. Yuan-Bentler scale χ^2 (87)=349.42, p<0.001, CFI=0.942, RMSEA=0.050 (90% CI 0.045-0.056)



Chapter Five: Discussion

Summary of Findings

The primary aims of this study were to: 1) Examine levels and correlates of physical activity in a diverse sample of women during the year after the birth of a child, and 2) Test associations of chronic life stress in several domains with CRP, a marker of systemic inflammation, at 6 months and 1 year postpartum. These aims were addressed using data from the CCHN study, a five-site, longitudinal study of mothers during the first year after the birth of a child. The sample included a large proportion of Black and Latino mothers, and individuals of very low socioeconomic were over-represented. We expected that higher levels of chronic stress would be associated with lower levels of physical activity, and higher levels of CRP.

Overall, results did not provide strong evidence for these hypothesized associations, although financial stress emerged as a significant predictor of both physical activity and CRP. Associations with physical activity ran contrary to hypotheses, such that individuals in the highest physical activity category had greater financial stress than less active participants. On the other hand, greater financial stress was associated with a higher CRP at both study visits, as well as a greater likelihood of chronic CRP elevation as hypothesized. Using structural equation modeling, we found that increased adiposity served as a mechanism for this association. Secondary aims were to explore whether behavioral and psychological resilience resources moderated relationships between chronic stress and CRP. There was no evidence that physical activity or resilience resources moderated these associations.

Results are reviewed in detail below, beginning with a discussion of results related to correlates of physical activity. We then consider findings on predictors of CRP, followed by overall strengths and limitations of the study methodology. Finally, implications and future directions are discussed.

Levels of Physical Activity in the Study Sample

In the present sample, close to two-thirds of all participants reported engaging in physical activity at frequencies and intensities that met IPAQ scoring criteria for moderate or high activity. This was somewhat unexpected because previous studies have shown that pregnancy and childbirth are associated with declines in physical activity (Brown, Heesch, & Miller, 2009; Pereira et al., 2007) but these levels of activity were comparable to or greater than those those typically reported in the general US adult population (Ainsworth et al., 2006; Carlson et al., 2010). We offer two possible explanations for the relatively high levels of physical activity in this sample.

First, the short form of the IPAQ used in this study asks participants to include physical activity accumulated during daily life including employment, household responsibilities and getting from place to place. Because all participants in this study had given birth to a child within the previous six months, the demands of caring for an infant may have contributed to weekly accumulation of physical activity. The IPAQ specifically mentions "carrying light loads" as an example of moderate activity, and most mothers of infants spend at least some time each day engaged in this type of activity since the average infant weighs about 16 lbs at 6 months of age. In addition to the daily workload of infant care, more than half of the sample had at least one other child in the home and 45% were working full or part time. Moreover, a majority of participants lived in urban areas where walking often serves as a means of getting from place to place.

Second, the high levels of physical activity in this sample may also be due to measurement error. Although the IPAQ has been widely used in research both in the U.S. and internationally, validation studies have shown weak to moderate correlations of the IPAQ with objective assessments of physical activity such as accelerometer data (Craig et al., 2003). While reporting bias is a problem with most if not all self-report measures of physical activity (Wilcox & Ainsworth, 2008), over-

reporting seems to be particularly problematic with the IPAQ and especially with its short form (Lee, Macfarlane, Lam, & Stewart, 2011).

Demographic Correlates of Physical Activity

Results of our analyses are consistent with previously reported differences in levels of physical activity by race/ethnicity in nationally representative samples (Carlson et al., 2010; Schiller, Lucas, Ward, & Peregoy, 2012), and we extend these findings by exploring these associations in multivariate models. African American women were less likely to be moderately or highly active than White or Latina participants, and these effects persisted even after controlling for potential confounders including household income, education, type of residential area, parity, relationship status, and age. In addition, Latina participants were less likely to be moderately or highly active than White participants, although the strength of this association varied slightly depending on covariates included in each analysis. These robust effects are consistent with prior studies reporting multivariate analyses and further add that race/ethnicity differences in physical activity are not entirely explained by confounding factors (Crespo, Smit, Andersen, Carter-Pokras, & Ainsworth, 2000; Mathieu et al., 2012).

Why were African American and Latina women in this sample less likely to be physically active during the postpartum year? First, women in these minority groups may be more likely than White women to live in unsafe neighborhoods that lack facilities such as parks or recreation centers, leading to fewer opportunities for exercise (Baruth, Sharpe, Parra-Medina, & Wilcox, 2014; Eyler et al., 1998; Fleury & Lee, 2006; Gordon-Larsen et al., 2006). Social norms around physical activity may also play a role in encouraging or discouraging activity among minority women. A recent review by Larsen and colleagues (2014) identified a number of culture-specific barriers to physical activity in Latina women. For example, the cultural importance of the caregiver role among Latinas emphasizes putting the needs of others before the needs of the self, and this expectation may discourage women

from taking the time to engage in self-care behaviors (Im et al., 2010; Juarbe, 1998; King et al., 2000). Cultural views on weight and body shape may also contribute to physical inactivity among African American and Latina women (Day, 2006; Mama et al., 2011; Viladrich, Yeh, Bruning, & Weiss, 2009). While a complete review of the large body of quantitative and qualitative work on cultural attitudes towards physical activity is beyond the scope of this discussion, it is important to remember that health behaviors occur within a larger social context that includes individual, interpersonal, organizational and neighborhood/community influences (Sorensen et al., 2003). Future studies should consider neighborhood-level predictors of physical activity and examine the importance of cultural norms around physical activity during the first postpartum year.

We also hypothesized that higher SES participants would report less physical activity than lower SES participants. Although bivariate analyses suggested that lower income and education were associated with a lower likelihood of adequate physical activity, this relationship was no longer significant in multivariate analyses. SES effects on physical activity have been reported in past studies (King et al., 2000; Trost, Owen, Bauman, Sallis, & Brown, 2002) and the discrepant findings in this sample may be due to the inclusion of non-leisure activities in our assessment of physical activity and an inability to untangle the two. By design, the CCHN sample over-represents individuals at the lower end of the SES spectrum, which may have curtailed the effects of SES on physical activity in this study.

Contrary to hypotheses, women who were working full- or part-time were more likely to be highly active than women who were unemployed or not working by choice. This higher level of activity among employed women may be due to additional movement accumulated while traveling to and from work, or may reflect the type of jobs held by women in this sample of predominantly low-income individuals. Classifying the types of jobs held by CCHN participants and linking occupational

categories to activity levels may be helpful in better understanding the higher levels of physical activity reported by employed women in this sample during the first postpartum year.

This study also confirms that type of residential area is important in shaping physical activity. The highest rates of physical inactivity were observed in the rural North Carolina site, and participants in this rural area had 50% lower likelihood of being moderately or highly active than urban residents in analyses adjusted for race/ethnicity, SES, parity, relationship status, employment status, and age. This finding is consistent with results from national cross-sectional studies showing lower physical activity among residents of less densely populated areas (Brownson, Baker, Housemann, Brennan, & Bacak, 2001; Parks, Housemann, & Brownson, 2003; Wilcox, Castro, King, Housemann, & Brownson, 2000).

Stress and Physical Activity

Greater chronic stress was not associated with lower levels of physical activity as hypothesized. Of the nine stress variables considered in this study, only Financial Stress differed by physical activity level but the direction of the effect was such that greater stress was associated with a greater likelihood of being highly active. This finding is puzzling given the inconsistency with studies showing an inverse association between physical activity and perceptions of stress (Hamer, Endrighi, & Poole, 2012; Ng & Jeffery, 2003; Penedo & Dahn, 2005). The unexpected pattern may be due to the fact that participants were asked to report on "activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport." Though analyses controlled for employment status, financially stressed participants may work a greater number of hours at more physically demanding jobs out of necessity. Transportation options may be more limited among women experiencing financial stress, resulting in reliance on public transportation and walking as a means of getting from place to place. Assistance with household chores and childcare duties may also be more limited, requiring additional energy expenditure to complete the activities of daily life. In sum, women with fewer economic resources may be able to afford fewer necessities and

conveniences than women who have greater resources, and may experience more physical demands during their daily tasks as a result.

CRP in CCHN mothers

The present study examined associations of chronic stressors with CRP, an acute phase protein that serves as a useful marker of low-grade systemic inflammation. 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 this sample of women from five sites across the U.S., a striking number of participants had clinically elevated levels of CRP. Forty-eight percent of participants showed evidence of elevated CRP at 6 months postpartum, and 46% had elevated CRP at 12 months postpartum. Also notable is the high frequency of repeated CRP values exceeding the clinical cutpoint of 3 mg/L, which were present in 35% of the sample.

The elevated levels of CRP in this sample are not entirely unexpected given the high rates of obesity and abdominal adiposity. Consistent with evidence that visceral adipose tissue serves as a source of pro-inflammatory cytokines, both BMI and waist circumference were associated with elevated CRP in this sample. Systemic inflammation may also be related to 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 (Cho et al., 2011; Groer et al., 2005; Watanabe et al., 1997). Limited data have been published on the normal ranges of CRP in women during the first postpartum year, though a recent paper found that CRP was higher in parous women as compared to nulliparous women in a sample of 822 young women in the Philippines (Kuzawa et al., 2013). Because little is known about the normal range of CRP during the first postpartum year, it is unclear whether the high rates of elevated CRP in this sample are fairly typical for women in the year after the birth of a child or reflective of a particularly at-risk sample.

Chronic Stress and Inflammation

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 individual chronic stressors were examined, only Financial Stress emerged as a significant predictor of log transformed T2 CRP and T3 CRP. 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, results were attenuated to non-significance when BMI was included in the regression model. 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. Additional analyses using structural equation modeling revealed that the association between Financial Stress and CRP was indeed mediated through 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 (Gallo, Jiménez, Shivpuri, de los Monteros, & Mills, 2011; Georgiades, Janszky, Blom, László, & Ahnve, 2009; Steptoe, Brydon, & Kunz-Ebrecht, 2005). 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 (Block, He, Zaslavsky, Ding, & Ayanian, 2009; Siahpush et al., 2014).

Several pathways may underlie this association between Financial Stress and increased adiposity. First, energy-dense foods that include refined grains, added sugars, and added saturated/trans fats are less expensive than fresh fruits and vegetables (Drewnowski & Darmon, 2005; Drewnowski & Specter, 2004; Monsivais & Drewnowski, 2007). Inadequate financial resources and limited food budgets may lead individuals to choose cheaper, less nutritious foods that promote weight gain.

Second, many individuals and especially women increase calorie intake in response to stressful circumstances or choose "comfort foods" with increased fat and/or sugar content to cope with emotional distress (Finch & Tomiyama, 2014). Finally, stress-related dysregulation of metabolic processes may play a role in the association between obesity and chronic psychological stressors such as financial strain (Bjorntorp, 2001; Black, 2006).

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 (Brummett et al., 2013; Gruenewald et al., 2009; Pollitt et al., 2008). 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. However, another possibility is that the measure used in this study reflects individual appraisals of financial stress, which are typically conceptualized as more proximal to physiological stress responses than stress exposures. Financial Stress was negatively correlated with income in this sample, but this correlation was not perfect and there was a good deal of variability at different income levels. The unique variance may be due to differences in stress appraisals that arise due to individual differences in resources for coping with stress. For example, even among the 1,028 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 partially attributable to the high levels of resilience resources reported by participants in this sample, as most participants reported high levels of optimism, mastery, self-esteem, and perceived support. Regardless, they indicate that low income must be appraised through the lens of one's other coping resources in order to determine effects on physiology and health outcomes (Cohen et al., 1983; Lazarus & Folkman, 1984).

It is interesting that associations between CRP and Financial Stress were stronger than those found for other indicators of difficult life circumstances. Financial Stress was the only one of the nine stress variables associated with CRP levels. But 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.

Another possible explanation for the unique effects of Financial Stress relates to differences in how domains of chronic stress were measured in this study. Rather than using items to assess participants' subjective appraisals of stress, Neighborhood, Family Relationship, Partner Relationship, and Co-Parenting Stress were assessed using the CCHN LSI, which uses more objective interviewer ratings of difficult life circumstances. If the degree of dysregulation associated with chronic stress depends on subjective appraisals of distress, then the advantages of the LSI as an objective stress assessment may obscure associations with chronic stressors in these domains. However, there was no association between CRP and perceived stress in this study either, and other studies have reported null findings or even inverse relationships for perceived stress (Kiecolt Glaser et al 2003; Christian, 2009; Doyle & Molix, 2014). 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.

Health Behaviors and Inflammation

This study tested associations between CRP and health behaviors including physical activity, sleep, smoking, breastfeeding, and alcohol use. A number of unexpected findings emerged. First, physical activity was not associated with lower levels of CRP as hypothesized. While this finding conflicts with results from several observational studies with large samples of adult men and women (Abramson & Vaccarino, 2002; Borodulin, Laatikainen, Salomaa, & Jousilahti, 2006; Ford, 2002; Pischon et al., 2003; Pitsavos et al., 2003; Rueben, Judd-Hamilton, Harris, & Seeman, 2003), the evidence for anti-inflammatory effects of physical activity is not entirely consistent in the general population and has not been explored in the context of the postpartum period. A number of randomized controlled trials of exercise aimed at reducing CRP have reported null findings (Campbell et al, 2008; Hammett et al., 2006; Huffman et al., 2006; Marcell et al., 2005; Murphy et al., 2006) and others have reported findings suggesting that reductions in CRP are contingent on whether the lifestyle intervention affects body weight or not (Esposito et al., 2003; Villareal et al., 2006).

This study also examined whether chronic stress had differential associations with CRP in physically active persons as compared to inactive participants. This hypothesis was based on prior studies showing that the health benefits of physical activity are more pronounced among individuals experiencing high as compared to low levels of psychological stress (e.g., Carmack, de Moor, Boudreaux, Amaral-Melendez, & Brantley, 1999; Gerber, Kellmann, Hartmann, & Pühse, 2010; Puterman et al., 2010; Puterman, Adler, Matthews, & Epel, 2012; Rueggeberg, Wrosch, & Miller, 2012). The moderating role of physical activity on health outcomes in the context of life stress has been reported in the literature, though not previously examined in the context of the postpartum period. The effects of chronic stress did not vary by level of physical activity in this sample. Resilience resources were also tested as a moderator of the effects of chronic stress on inflammation, and findings

did not provide any evidence that resilience resources weakened associations between chronic stress and CRP.

There are several possible explanations for why the expected moderation effects were not observed. Most notably, physical activity as assessed in this study is not synonymous with exercise which refers to a regular, structured, leisure-time type of physical activity. There is a degree of control inherent in exercise, as an individual can choose when, where, how, and how often to engage in this type of activity. On the other hand, physical activity related to occupational and domestic tasks is driven by necessity rather than by choice. Previous studies have shown that parents report low perceived behavioral control over physical activity, possibly due to increased time demands and workload associated with caring for children (McIntyre & Rhodes, 2009; Rhodes et al., in press). Physical activity that arises as part of job, household, and childcare responsibilities may also lack the cognitive benefits of leisure-time physical activity, as evidenced by the lack of association with resilience resources such as mastery, self-esteem and optimism. Unfortunately, it is not possible to disaggregate types of physical activity in this study but evidence from other studies shows that mothers spend more time in household and lifestyle activity and less time in leisure-time activity than childless women (Grace, Williams, Stewart, & Franche, 2006; Scharff, Homan, Kreuter, & Brennan, 1999; Sternfeld, Ainsworth, & Quesenberry, 1999).

While there were no significant associations between physical activity and CRP, another health behavior showed a powerful protective effect in this sample of women during the first year postpartum. Breastfeeding was associated with lower CRP, such that women who were still breastfeeding their six month old infants had lower CRP both concurrently and again six months later. Women who breastfed for six months or more were also significantly less likely to have chronically elevated CRP, even after controlling for BMI. These results add to a number of documented health benefits of breastfeeding for both mothers and their babies, including protective effects on immune function (Du et al., 2012; Chien

& Howie, 2001; McDade et al., 2010; Ip, Chung, Raman, Trikalinos, & Lau, 2007) and lowered risk of obesity (Chivers et al., 2010; Dewey, Heinig, & Nommsen, 1993; Oddy, 2012). Because of its links to lowered risk of postpartum weight retention and other metabolic benefits, breastfeeding may be the most important powerful health behavior that a woman can engage in during the first postpartum year.

Puzzlingly, smoking was associated with lower CRP at both six months and one year postpartum. This finding contrasts with a number of studies showing that smokers tend to have higher levels of markers of systemic inflammation including IL-6 and CRP (Bermudez et al., 2002; Haddy et al., 2005; McDade et al., 2006; McDade et al., 2010; Nanri et al., 2007; Wannamethee et al., 2007). However, some studies have reported higher CRP values in male but not female smokers, suggesting that there may be sex differences in the association between smoking and CRP (Bo et al., 2005; Brummett et al., 2013; Nazmi et al., 2008). Although smokers in the present sample were less likely to be overweight or obese than non-smokers, the unexpected direction of the relationship between smoking and CRP is unlikely to be due to adiposity given that this was controlled in analyses. One possible explanation for the unexpected finding is that studies have shown a graded relationship between amount smoked over a lifetime and increases in inflammatory markers (Bazzano et al., 2002; Bermudez et al., 2002; Mendall et al., 2000) The women who reported smoking in this sample were young and not heavy smokers. More than 25% of smokers reported smoking occasionally rather than daily, and 90% of daily smokers reported smoking fewer than 10 cigarettes per day. These unexpected results suggest that behavioral influences on inflammation during the postpartum period are quite complex.

Strengths and Limitations

This study had several methodological strengths. The longitudinal design allowed for multiple assessments over the course of the first year postpartum. Both continuous and dichotomous outcome variables were examined, and structural equation modeling allowed us to estimate associations of

financial stress and adiposity with an error-free latent construct representing CRP levels between 6 and 12 months postpartum. The use of community-based participatory research methods led to the inclusion of a multiple measures assessing stress in domains most relevant to the study population. In addition, the use of the CCHN LSI provided a more objective measure of chronic stress in the domains of neighborhood, family relationships, partner relationship, and co-parenting, and financial stress was assessed directly rather than inferred from conditions such as poverty. In addition, resilience resources were examined whereas many studies neglect to consider the many resources that individuals bring to bear on stressful situations. This study also included a diverse sample of participants from underserved populations that are often underrepresented in research on stress and health, including a large proportion of very low SES individuals.

Several limitations deserve consideration. As noted above, assessment of physical activity by self-report can be inaccurate, and the instrument used here was problematic as well for understanding differences between leisure and required activities. Future studies with objective assessments of physical activity using accelerometers along with daily diaries could address methodological problems with self-reported physical activity and better characterize the purpose with which physical activity is undertaken in this population. This study examined only individual-level predictors of physical activity, but physical activity is likely shaped by factors at multiple levels including family, neighborhood, and region. Future work could utilize census tract data collected by the CCHN to link individual physical activity levels to community-level factors such as neighborhood SES.

Because of the observational nature of the data, causal relationships between study variables cannot be inferred. For example, it is possible that high levels of inflammation related to chronic illness contribute to Financial Stress by limiting employment opportunities or generating additional healthcare expenses. Ideally, future studies should assess Financial Stress at multiple time points to explore the directionality of effects.

Additional issues may have reduced ability to detect a significant association between chronic stress and inflammation. Participants with current infections or injuries were not excluded from blood sampling. Although, we attempted to control for recent illness as a covariate, the extent of data collection on infectious illness was limited and by quick self-report. In addition, CRP was sampled only once at each time point and ideally, the mean of two independent measures of CRP two weeks apart is recommended (Pearson et al., 2003).

Implications and General Conclusions

Findings from these analyses have potential implications for maternal health. Both physical inactivity and low-grade systemic inflammation have been tied to adverse cardiovascular outcomes, and women with evidence of these risk factors during the first year postpartum may be more likely to develop health problems later in life. Promotion of adequate physical activity in mothers is particularly important because parents play a role in modeling physical activity for their children. Although the current study did not assess specific barriers that prevent women from achieving adequate levels of physical activity during the first postpartum year, the findings suggest that African American, Latina, rural-dwelling women groups may benefit most from physical activity promotion efforts.

Study findings suggest that financial stress stemming from socioeconomic disadvantage may be a particular deleterious form of stress. Inadequate financial resources during the year after the birth of a child may have consequences for a mother's disease risk later in life and possibly future pregnancy outcomes. It is difficult to propose practical intervention strategies in light of the fact that financial strain among families living in poverty stems from larger issues such as income inequality and inadequate allocation of resources to economic assistance programs. 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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