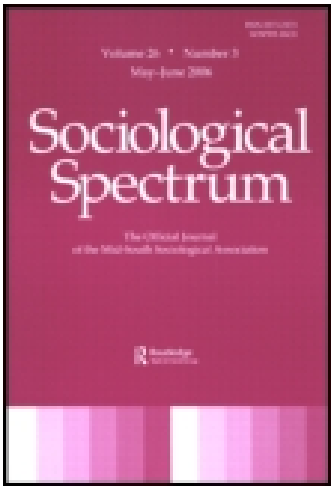

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Swell Foundations: Fundamental Social Causes and Chronic Inflammation

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Swell Foundations: Fundamental Social Causes and Chronic Inflammation

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We assess prior research identifying potential causal links between social disadvantage and chronic inflammation, using Fundamental Social Causes (see Link and Phelan 1995) and gender theory.

We use ordinary least-squares regression to investigate how social structure and relationship factors predict C-reactive protein serolevels among participants in the National Social Life, Health, and Aging Project (Waite et al. 2007).

Gender predicts chronic inflammation status more strongly and reliably than any other social factor; female participants show significantly higher CRP levels. Other reliable predictors include race, education, and marriage. Black race predicts significantly higher CRP; college education and marriage predict significantly lower CRP. Income predicts CRP inconsistently. Social support and communication variables do not significantly predict CRP in our models.

Gender inequalities and pressures may place women at heightened risk for chronic inflammation. Political advocacy and policy action approaches may thus prove more effective than medical interventions for inflammation prevention.

An emerging line of scholarship explores the ways social exposures may fundamentally cause (Link and Phelan 1995) the development of chronic inflammatory diseases (Crimmins and Seeman 2001). Utilizing datasets incorporating information on diagnosis, underlying pathology, personal attributes, socio-emotional experiences, and important biomarkers (like C-reactive protein and interleukin-6), for example, researchers have demonstrated some ways societal patterns of inequality facilitate and produce widespread health disparities within and between populations (Crimmins and Seeman 2001; Suzman 2009). In so doing, researchers have shown that biomarker data can help illuminate the social causes underlying chronic inflammation even in cases where people lack adequate medical access or care. While these studies have importantly

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revealed racial, socioeconomic, and gender disparities in the development of chronic inflammation (see McDade, Hawkley, and Cacioppo 2010; Kiecolt-Glaser, Gouin, and Hantsoo 2010), less is known about the social patterns that facilitate and produce these disparities.

In this study, we extend previous research into the social causes of chronic inflammation by demonstrating some ways disparities in chronic inflammation align with existing gender scholarship demonstrating the socially constructed and institutionalized nature of gender inequality (see, e.g., Connell 1987; Martin 2004; West and Zimmerman 1987). Specifically, our analysis suggests that ''doing gender'' (West and Zimmerman 1987), or navigating the life course in ways that signify and affirm feminine and masculine selves, may represent a fundamental social cause of disparities in chronic inflammatory conditions. Further, we show that gender disparities cannot fully be explained by social support, structural, or communication factors (suggestions prompted by the aforementioned studies). Rather, we argue that many of the activities typically ''coded'' (West and Zimmerman 1987) or ''institutionalized'' (Martin 2004) as feminine may better explain gender disparities in chronic inflammation. In so doing, we call for greater attention to the dynamics of gender and associated social relationships in the social etiology of chronic inflammation.

BACKGROUND

Extant sociological literature contains relatively few studies specifically addressing inflammatory biomarker levels as outcomes from social inequalities. However, sociologists have devoted copious attention to the social patterning of chronic health conditions. Many of these studies have included inflammatory conditions. Common inflammatory diseases addressed in these studies include different types of arthritis, asthma, bowel conditions, and cardiovascular disease. This body of research has developed strong and consistent evidence to suggest that different types of social disadvantage predispose people to developing a wide variety of chronic diseases (Marmot and Wilkinson 2005; Braveman, Egerter, and Williams 2011).

Such studies demonstrate that individuals tend to experience higher risk of almost every known chronic condition (Ben-Shlomo and Kuh 2002; Kuh and Ben-Shlomo 2004). Many of these chronic conditions also involve inflammation (McDade, Lindau, and Wroblewski 2010). Osteoarthritis and atherosclerosis constitute excellent examples of such diseases (Kuh and Hardy 2002). In fact, these conditions are so common among older adults that some researchers classify them as part of usual aging (Baumgartner 2000). Other chronic diseases are also far more common in older adults—each unit of time that passes represents an additional chance for a given person to develop disease. Conditions of a progressive nature also tend to worsen with time, and are likely to manifest as people reach older ages (Pinder 1992; Verbrugge and Jette 1994).

This is especially true for conditions involving inflammation, which can permanently damage many different bodily structures (Finch and Crimmins 2004). Indeed, researchers have begun to explore relationships between disadvantage and inflammatory conditions specifically (Braveman et al. 2011). Stress associated with disadvantage is suspected to harm health by increasing the body's physical burden, described by social scientists as ''allostatic load'' (Geronimus et al. 2006). These processes have been implicated in both the development of generalized inflammation (Cornwell and Waite 2009) and exacerbation of inflammation in diseases affecting

particular organ systems. Specific inflammatory diseases exacerbated by disadvantage-related stress include asthma (Chen et al. 2006), inflammatory bowel disease (Graff, Walker, and Bernstein 2009), Crohn's disease (Cámara et al. 2011), and rheumatoid arthritis (Symmons 2003). Social disadvantage may even predispose people to developing certain inflammatory conditions, such as asthma (Williams, Sternthal, and Wright 2009) and rheumatoid arthritis (Symmons 2002). However, it should be noted that exacerbation of an existing condition could mimic development of a new condition if these increased symptoms were responsible for a person actively seeking medical care and receiving a new diagnosis.

The biological consequences of aging may account for much of the variation in chronic disease risks observed across different age strata (Crimmins and Seeman 2001; Finch and Crimmins 2004). As cells lose their ability to divide, essential structures in the body may experience damage (Hayflick 1998; Hornsby 2006). However, sociologists have increasingly demonstrated that age-related discrimination may also increase psychological distress in late life (Calasanti and Slevin 2001). Specific sources of distress may include being terminated from paid employment, marginalized by family members (Treas 1977; Morycz 1985), ignored or patronized by healthcare providers, and viewed as irrelevant by society at large (Calasanti and Slevin 2001). Such distress can negatively impact physical health and increase the risk of developing many different chronic diseases (Tessler and Mechanic 1978; Watson and Pennebaker 1989).

While age usually increases people's risk of developing different chronic conditions, it may also reduce the severity of symptoms from existing diseases. This is especially true for inflammatory conditions, which often involve the immune system (Finch and Crimmins 2004; McDade et al. 2010). Since immune activity tends to subside somewhat in old age, people with inflammatory autoimmune diseases may find that their symptoms abate as they progress through late life (Finch and Crimmins 2004; McDade et al. 2010). Likewise, people who have lived with specific health conditions for long periods of time may gain valuable understanding of how to manage their diseases (Bury 1991; Charmaz 2000). Effective self-management practices can safeguard health by promoting overall wellness and slowing progression (Gately, Rogers, and Sanders 2007). Some older adults may thus experience comparatively milder symptoms than younger adults with the same conditions. If inflammatory disease status is captured using biological assays such as blood markers, older people may appear to have lower rates of severe disease compared to younger cohorts (Crimmins and Seeman 2001; McDade et al. 2010).

Sociologists have found that age-related increases in chronic disease risk occur on a more rapid basis for certain populations, especially black women. Indeed, ethno-racial background consistently exhibits a strong ability to fundamentally cause chronic disease (Williams and Sternthal 2010; Link and Phelan 1995; Williams 1997). In America, strong disparities exist between people whose heritage comes primarily from Europe and those who trace their ancestry to other parts of the world. Whites tend to experience the lowest rates of most chronic diseases.

Most non-white groups experience either comparable or markedly higher rates (Williams and Sternthal 2010; Williams 1997). Black and Native American groups experience highly elevated rates of nearly all chronic diseases (Williams and Sternthal 2010; Williams 1997). By contrast, Asian Americans generally show incidence and prevalence rates either slightly lower than or comparable to those of white Americans for most chronic conditions (Bratter and Eschbach 2005). Certain Hispanic groups (e.g., Mexican and Puerto Rican Americans) experience elevated

rates of multiple chronic conditions (Bratter and Eschbach 2005). Other Latino groups (e.g., Cuban Americans) show chronic disease rates comparable to those of white Americans (Bratter and Eschbach 2005). Further, these rates are higher among people whose families have remained in the United States for multiple generations.

In sum, being female, older, black, Native American, Mexican, Puerto Rican, divorced, and/or widowed is generally associated with higher risk of chronic disease. Having low educational attainment and/or household income can likewise predispose people to developing different chronic conditions. By contrast, being male, younger, white, Asian, Cuban, and/or married is generally associated with lower risk of chronic disease. Having high educational attainment and/or household income can likewise protect people from different chronic conditions. These patterns are consistent across a variety of different chronic diseases, including inflammatory conditions. Taken together, these findings suggest that both social structure and relationship factors can fundamentally cause chronic inflammatory disease.

In the preceding studies, chronic inflammatory disease is usually measured using diagnosis information. Most quantitative studies capture information about chronic conditions by asking questions like ''Has a doctor ever diagnosed you with any type of arthritis?'' (Bowling 2004). Such questions can indeed provide a wealth of useful information about physical health (Bowling 2004), and help researchers gain insight into the social experiences of people with chronic conditions. However, diagnosis data also have several inherent limitations (Mirowsky and Ross 1989). Specifically, the data they capture often reflect people's medical care history as much as their actual chronic disease status (Crimmins and Seeman 2001).

In recent years, new ways of measuring inflammation via biological assays have become available and widely accessible (Crimmins and Seeman 2001). These biomarkers offer insight into the underlying physical processes that affect people regardless of their ability or desire to access medical care (Crimmins and Seeman 2001; Shih, Fernandes, and Bird 2010). C-reactive protein and interleukin-6 are two of the most common inflammatory biomarkers collected (Crimmins and Seeman 2001). Unlike diagnosis information, collecting these measurements does not require people to visit medical care settings—blood samples to test for inflammatory biomarkers can be collected in homes and communities (Crimmins and Seeman 2001; Shih et al. 2010). Indeed, sociologists have increasingly used these biomarkers to look at the relationships between chronic inflammation and social life (Shih et al. 2010).

For example, McDade and associates (2010) conducted an in-depth analysis of different social structural factors' possible contributions to inflammatory pathology in late life. Consistent with other studies of fundamental causation, the authors found that several different elements of social structure can influence inflammation status as people age. Specifically, the authors found that being female strongly predicts high levels of inflammatory biomarkers. By contrast, they found that having higher levels of formal education strongly predicts milder inflammatory pathology. The authors encourage other researchers to explore different types of relationships between inflammation, health, social interaction, and quality of life.

Kiecolt-Glaser and colleagues (2010) conducted a review of different social relationship factors' ability to fundamentally cause chronic inflammation. Their findings likewise mirror prior evidence suggesting that intimate relationship participation and dynamics can powerfully shape health outcomes. Specifically, the authors found that participating in long-term intimate relationship—especially marriage—predicts significantly lower levels of inflammatory biomarkers.

Likewise, they found that receiving ample social support predicts lower overall levels of chronic inflammation. Their review suggests that biomarkers of inflammation hold significant promise for medical sociologists interested in a variety of different social factors that contribute to inflammatory pathology.

Both the study by McDade and colleagues (2010) and several of those reviewed by Kiecolt-Glaser and colleagues (2010) used C-reactive protein to assess inflammation status. Recent work in the field thus suggests that C-reactive protein holds substantial promise for further sociological research on inflammation. Indeed, Crimmins and Seeman (2001) cite C-reactive protein as a useful outcome measure for studies of inflammatory diseases. Pepys and Hirschfield (2003) summarize a robust evidence basis for using this biomarker to assess chronic inflammation specifically.

We thus crafted our own study synthesizing and contextualizing a variety of social determinants of chronic inflammation. In so doing, we follow up on unexplored findings from previous research by McDade and colleagues (2010). Because structural and relational disadvantage can occur simultaneously, and even influence one another, we found value in aggregating both types of predictors into more inclusive models of chronic inflammation using C-reactive protein as the outcome. From these efforts, we illuminate key areas in need of further exploration, and show how gender dynamics may predispose people to chronic inflammation.

METHODS

Data and Subject Selection

We used data from Wave I of the NSHAP. Developed between 2005 and 2006, this dataset provides information on physical, mental, and social health among United States residents aged 57 to 85. The NSHAP includes data on 3,005 individuals living in and around Chicago, Illinois. Oversampling of specific demographic groups (African Americans, Latinos, males, and persons 75 to 85 years of age) allows the dataset to be nationally representative (Waite et al. 2007). Data for the NSHAP are collected via a combination of in-home interviews, supplemental questionnaires, blood spot blotters, and saliva cultures.

Life course sociology literature increasingly suggests that disadvantage in psychosocial outcomes accumulates over time from adverse social experience (see also Ferraro and Shippee 2009; Mayer 2009). While the NSHAP data remain largely cross-sectional at present, the inclusion criteria guarantee at least 57 years elapsed between birth and data collection for a given subject. Analyzing an older population cross-sectionally allows researchers to capture long-term impacts from biosocial exposures that began in early life (Geronimus 1992). Consequently, the NSHAP can illuminate potential accumulation of social disadvantage that translates to poor health outcomes.

We used C-reactive protein as a proxy for chronic inflammation outcomes in all analyses. This biomarker has a strong evidence basis supporting its use in biosocial research on inflammation (see Pepys and Hirschfield 2003). C-reactive protein also provides the best evidence of chronic inflammation available in the NSHAP. Using correlational tables, we checked all variables for differential attrition owing to the lack of successful C-reactive protein measurements for about 35% of participants. We also noted challenges with inference related to the original measurement strategies.

The dataset captures information on sex only as a binary construct. It also does not (despite calling its sex variable ''gender'') collect any information on gender identity. However, most NSHAP participants (age minimum 57 years at Wave I) were probably socialized in environments that conflated sex and gender. ''Females'' and ''males'' in the sample are thus likely to experience social norms and pressures associated with being ''women'' or ''men.'' As a result, we note the lack of an explicit measure of gender in the dataset while using the existing measure of sex (e.g., ''females'' and ''males'') as a proxy for gender experience and identification (e.g., respondents were forced to ''do gender'' by selecting from binary options on the survey instrument). Assessing possible effects from ethno-racial background presented similar challenges. NSHAP data do not provide detailed information on ethnic identity or cultural socialization. We were thus not able to separate these elements from the broader biosocial idea of race. Table 1 describes the data available in NSHAP for all variables used in our analyses.

As suggested above, some variables in the NSHAP may have problematic values due to measurement error. This concern is most pressing for the C-reactive protein variable. Although most values are clustered within a range of 10 mg/L, some values are closer to 50 and one

TABLE 1
Descriptions and Summary Statistics for Included Variables

Construct	Variable	Units	Cases	Mean	St. Dev.	Min.	Max.
Chronic inflammation	C-reactive protein	mg/L	1,939	3.20	6.03	0	100
Sex	Female	Yes/No	3,005	0.52	0.50	0	1
	Male			0.48	0.50		
Age	Years of age	Years	3,005	69.3	7.85	57	85
	57 to 64	Yes/No	3,005	0.34	0.47	0	1
	65 to 74			0.36	0.48		
	75 to 85			0.30	0.46		
Education	Years of education	Years	2,971	12.48	4.10	0	32
	Less than high school	Yes/No	3,005	0.23	0.42	0	1
	High school or equivalent			0.26	0.44		
	Some college			0.28	0.45		
	Finished college			0.22	0.41		
Race	White	Yes/No	2,993	0.70	0.46	0	1
	Any minority			0.30	0.46		
	Black			0.17	0.38		
	Hispanic			0.10	0.30		
	Other race			0.02	0.15		
Income	Household earnings	$/year	2,124	51,264	76,559	0	1,800,000
Social support and communication	Open with partner	Points	2,012	2.71	0.54	1	3
	Open with friends		2,704	2.02	0.73		
	Relying on family	Points	2,793	2.57	0.65	1	3
	Relying on friends		2,680	2.30	0.71		
Relationship status	Married	Yes/No	3,005	0.60	0.49	0	1
	Cohabitating			0.02	0.14		
	Separated			0.02	0.13		
	Divorced			0.11	0.31		
	Widowed			0.22	0.41		
	Never married			0.04	0.19		

person appears to have a value of 100. Since C-reactive protein levels usually remain in the single digits even for people with severe inflammation, these very high values are more likely attributable to measurement issues. Indeed, data documentation for the NSHAP suggests that most extreme measurements on C-reactive protein owe to problems with data collection and processing. Previous researchers (McDade et al. 2010) have addressed this issue by cutting C-reactive protein values at 8.6 mg/L, above which threshold recorded values from blood spots are likely to reflect either measurement error or severe acute inflammation. We used the same approach.

The income indicator has distributional issues similar to those outlined above for the C-reactive protein variable. After conducting extensive sensitivity analysis, however, we did not elect to exclude any NSHAP participants from the final models based on their annual household earnings.

Techniques for Analysis

We began this study with the basic desire to explore extant findings concerning fundamental social causes of chronic inflammation in greater detail, and with a higher degree of synthesis between different types of social forces. We considered three key questions:

1. Do social structure factors fundamentally predict chronic inflammation status?
2. Do social relationship factors fundamentally predict chronic inflammation status?
3. When these factors are considered together, which ones appear to predict inflammation status most reliably?

We explored possible fundamental causation processes by creating a series of OLS regression models, using C-reactive protein as the outcome and each social structure or relationship variable as an independent predictor. An OLS approach was indicated by the continuous nature of the C-reactive protein measure, as well as the fine gradations observed between different values for this item in the NSHAP. Nearly all NSHAP participants' blood levels of C-reactive protein fall between 0 and 8.6 milligrams per liter. Because of right skew in the distribution of NSHAP participants' C-reactive protein levels (median 1.5 mg/L, mean 3.5 mg/L) we log-transformed our outcome variable. This approach was also used by McDade and colleagues (2010). It allowed us to correct for right skew in our data and also heteroskedasticity of residuals. To address the latter, we also re-estimated all regressions with Huber-White standard errors.

We computed one bivariate model (see Table 2) for each covariate of interest to determine the potential net effects of each social factor on inflammation status for people in the sample. We then analyzed the contributions of each group of social influences (structure and relationship) when modeled together as a block. Finally, we computed an additional multivariate model incorporating both blocks of social variables simultaneously (see Table 3). This enabled us to assess which particular social factors might exert stronger influences on chronic inflammation status, net of other key determinants of health inequality.

We began with a multi-predictor approach because we were interested in the independent and synergistic predictive value of a variety of social predictors analyzed by McDade and colleagues (2010). Our analyses stemmed from curiosity about why this previous study did not delve into its relatively strong findings concerning the possible influence of gender. However, in order to explore those findings we first needed to reproduce them rather than

TABLE 2
Ordinary-Least Squares Regressions of Logged C-Reactive Protein on Individual Social Factors ($n = 803$)

Outcome	Construct	Predictor	Coefficient
C-reactive protein serolevel (log-transformed)	Sex	Female	$+0.346^{***}$
		Male	-0.346^{***}
	Age	Years of age	-0.003
	Race	Racial minority	$+0.232^{*}$
		White	-0.232^{*}
		Black	$+0.522^{***}$
		Hispanic	$+0.065$
		Other race	-0.386
	Education	Years of education	-0.042^{***}
		Less than high school	$+0.289^{**}$
		High school or equivalency	$+0.096$
		Some college or vocational certificate	$+0.152^{\dagger}$
		College graduate	-0.423^{***}
	Income	Annual household earnings ($1 K/yr)	$-9.41 \times 10^{-10*}$
	Social support and communication	Can be open with partner	-0.087
		Can be open with friends	-0.032
		Can rely on family	-0.025
		Can rely on friends	-0.044
	Relationship status	Married	-0.286^{*}
		Cohabitating	$+0.411$
		Separated	$+1.829^{\dagger}$
		Divorced	$+0.308$
		Widowed	$+0.137$
		Never married	-0.157

$^{\dagger}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

TABLE 3
Logged C-Reactive Protein Serolevels Regressed on All Social Factors

Outcome	Variable	Model 1	Model 2	Model 3
C-reactive protein serolevel (log-transformed)	Female	$+0.299^{***}$	–	$+0.318^{***}$
	Age	-0.004	–	-0.005
	Less than HS education	$+0.183$	–	$+0.193$
	Some college education	$+0.036$	–	$+0.040$
	Full college education	-0.324^{**}	–	-0.329^{**}
	Black	$+0.388^{**}$	–	$+0.345^{*}$
	Hispanic	-0.100	–	-0.076
	Other racial minority	-0.248	–	-0.246
	Household income	-4.86×10^{-10}	–	-4.72×10^{-10}
	Married	–	-0.288^{*}	-0.311^{*}
	Openness with partner	–	-0.075	-0.023
	Openness with friends	–	-0.029	-0.073
	Can rely on friends	–	$+0.012$	$+0.048$
	Can rely on family	–	-0.024	-0.037
Sample size		*803*	*803*	*803*
Prob>F		*0.0000*	*0.2242*	*0.0000*
R-squared value		*0.0716*	*0.0087*	*0.0811*

$^{\dagger}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

immediately conceptualizing gender (utilizing sex identification as male or female as a proxy) as the sole predictor and other social variables as ''controls.''

Preliminary analyses also included distributional assessment of each variable to determine modeling approaches. These explorations suggested that we might experience significant issues with heteroskedasticity in modeling the C-reactive protein outcome. After computing our initial models, we performed a series of supplemental diagnostics that corroborated this concern. We first performed collinearity diagnostics on all predictors to ensure that the core assumptions of our OLS models were not substantially violated. We then ran Breusch-Pagan procedures to check for violation of regression assumptions.

Finally, we conducted sensitivity analyses incorporating other suspected behavioral and biological determinants of chronic inflammation. While the original McDade and colleagues (2010) study did not incorporate many covariates beyond the studied range of social determinants, a peer reviewer suggested that we compute these additional models to contextualize the predictive value of our original regression models. We thus modeled physical, mental, and behavioral

TABLE 4
Sensitivity Analyses with Other Health-Related Variables

Outcome	Variable	Old Model	New Model
C-reactive protein serolevel (log-transformed)	Female	$+0.430^{***}$	$+0.470^{***}$
	Age	-0.007	-2.70×10^{-10}
	Less than HS education	$+0.051$	-0.049
	Some college education	-0.085	-0.072
	Full college education	-0.467^{**}	-0.388^{**}
	Black	$+0.396^{\dagger}$	$+0.293$
	Hispanic	$+0.122$	$+0.085$
	Other racial minority	$+0.022$	-0.078
	Household income	-6.130×10^{-10}	-2.70×10^{-10}
	Married	-0.214	-0.279
	Openness with partner	$+0.021$	$+0.005$
	Openness with friends	-0.101	-0.050
	Can rely on friends	-0.036	-0.030
	Can rely on family	-0.064	-0.112
	Body mass index	–	$+0.032^{***}$
	Self-rated physical health	–	$-0.118^{\dagger}$
	Health relative to age peers	–	$-0.120^{\dagger}$
	Self-rated mental health	–	$+0.052$
	Stress level	–	-0.090
	Health insurance coverage	–	-0.186
	Have a medical home	–	-0.132
	At least one chronic condition	–	-0.130
	At least one STD	–	-0.186
	History of smoking	–	$+0.031$
	Currently a caregiver	–	-0.247^{*}
Sample size		*377*	*377*
Prob>F		*0.0000*	*0.0000*
R-squared value		*0.1104*	*0.2201*

$^{\dagger}p < 0.10$, $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$.

health attributes (shown in Table 4) available in NSHAP that are indicated by the literature as being relevant for physical health outcomes (see Johnson and Krueger 2005). In several cases we were able to incorporate constructs demonstrated to predict chronic inflammation specifically (see McDade et al. 2006). These included persistent stress, higher body mass relative to height, and past or present smoking.

Because the C-reactive protein variable did indeed display significant heteroskedasticity in some modeling frameworks, we recomputed all models using log transformation (see also McDade et al. 2010). Mathematically, results from these aggregated models closely parallel those obtained by McDade and associates (see also Kiecolt-Glaser et al. 2010). Where possible, we compared our results to previous research, then drew on these comparisons to illuminate key gaps for exploration in future studies.

RESULTS

Bivariate Models

Using OLS regression techniques, we computed a variety of models to predict logged C-reactive protein serolevels among participants. We first used each social structure or process factor as the predictor in its own independent model to predict inflammatory biomarker levels. In cases where we documented a significant effect from an aggregated social predictor, we also decomposed that variable and assessed the independent effects of its component constructs. For example, when we found significant associations with ethno-racial minority status, we investigated the specific impact of being white, black, Hispanic, or another race. Results from these analyses are shown in Table 2.

Gender predicted chronic inflammation status when modeled independently. Identifying as female is associated with higher levels of logged C-reactive protein ($\beta = 0.346$, $p < 0.001$). Identifying as male predicted significantly lower logged C-reactive protein serolevels ($\beta = -0.346$, $p < 0.001$). Racial background likewise predicted participants' average levels of logged C-reactive protein. We found significant positive associations for our aggregate measure of ethno-racial minority group membership ($\beta = 0.232$, $p < 0.05$). This relationship appears to be driven mostly by results for black participants. Identifying as black predicts higher logged C-reactive protein serolevels ($\beta = 0.522$, $p < 0.001$). Other racial identities, such as Hispanic and ''other,'' did not significantly predict participants' average logged C-reactive protein levels.

Overall, education significantly predicted logged C-reactive protein values. Additional years of education were associated with slightly lower logged C-reactive protein levels ($\beta = -0.042$, $p < 0.001$). We also found significant associations for two levels of education: having less than a high school education ($\beta = 0.289$, $p < 0.01$) and having graduated from college ($\beta = -0.423$, $p < 0.001$). Completing some college coursework marginally predicted logged C-reactive protein levels ($\beta = 0.152$, $p < 0.10$). High school education itself did not significantly predict logged C-reactive protein serolevels. Each additional $1,000 of annual income was also associated with a very small (9.41×10^{-10} milligrams per liter) decrease in average C-reactive protein levels (p-value less than 0.05, greater than 0.01).

We began our exploration of relationship dynamics' ability to act as fundamental causes of chronic inflammation by independently modeling the influence of communication and

social support constructs. In bivariate models, none of these constructs significantly predicted C-reactive protein serolevels. These findings contrast sharply with those of Kiecolt-Glaser et al. (2010). Of the relationship statuses that we modeled, being married appeared to exert the most substantial influence on chronic inflammatory biomarkers. Being married predicted lower logged C-reactive protein serolevels ($\beta = -0.286$, $p < 0.05$) compared to average values for unmarried persons. In addition, being separated marginally predicted higher levels of chronic inflammation ($\beta = 1.829$, $p < 0.10$). Other marital statuses did not significantly predict C-reactive protein serolevels.

Multivariate Models

We also assessed the collective fundamental causation effects of all included social variables. First, we computed two integrated regressions incorporating the social structure and intimate relationship variables respectively. Next, we generated a more complex model including both groups of variables. Results from these analyses are shown below in Table 3.

Modeling structural predictors simultaneously returned significant results for participants that identified as female, college education, and participants that identified as black. Net of other social structure variables, identifying as female was associated with higher logged C-reactive protein levels ($\beta = 0.299$, $p < 0.001$) than those for males. Being a college graduate was associated with lower average logged serolevels of C-reactive protein ($\beta = -0.183$, $p < 0.01$). Having black heritage was associated with higher logged C-reactive protein levels ($\beta = 0.388$, $P < 0.01$). Neither age nor household income significantly predicted logged C-reactive protein when modeled with other social structure covariates.

Modeling relationship predictors simultaneously returned significant results for marriage. Net of other variables related to dynamics of social relationships, marriage was associated with lower logged C-reactive protein serolevels ($\beta = -0.288$, $p < 0.05$). Variables capturing openness with intimate partners, openness with friends, ability to rely on friends, and ability to rely on family did not significantly predict logged C-reactive protein levels when modeled collectively with the marriage predictor.

Aggregated multivariate modeling combining both sets of social predictors showed significant results for the same four social factors: female identification, college education, black identification, and marriage. Identifying as female predicted higher average logged C-reactive protein serolevels ($\beta = 0.388$, $p < 0.001$) with very high significance. Having a college education predicted lower logged C-reactive protein values ($\beta = -0.329$, $p < 0.01$) with high significance. Identifying as black predicted higher logged C-reactive protein values ($\beta = 0.345$, $p < 0.01$) with high significance. Marriage significantly predicted lower logged C-reactive protein levels ($\beta = -0.311$, $p < 0.05$).

Taken together, these results support many previous findings concerning the ability of both social status and intimate relationship constructs to fundamentally influence chronic inflammation. However, they also indicate that the included predictors can only explain a relatively small amount of the total variance observed in logged C-reactive protein levels among NSHAP participants. The overall model R-squared value for the final regression is 0.0811, suggesting that this model accounts for just over 8% of total variance in inflammatory biomarker levels. Considering that serum markers of inflammation are a biological construct, the ability of social factors to predict over 8% of variance in people's

levels of C-reactive protein may be very important to consider in conceptualizing why people experience chronic inflammation.

Computing a final set of models incorporating physical, mental, and behavioral health covariates not only produced an expanded model with substantially greater predictive capability of our original full model (see Table 3), but also strengthened the patterns we observed in our original regressions. By generating a reduced model incorporating the same independent variables from our original full model using only those NSHAP cases with valid data on all of the new covariates, we were able to compare results directly across model specifications. Using only the 377 NSHAP cases with real data for all included variables in the expanded model, we produced a reduced model with an *R*-squared value of 0.1104. By comparison, our expanded model yielded an *R*-squared value of 0.2201—nearly a 100% increase from the original value.

In this new model, our sex coefficient remained extremely significant with a p-value reported as 0.000. In addition, the parameter estimate for this coefficient was 0.470 in the expanded model compared to 0.430 in the reduced model. We note that in the expanded model, sex was the only social predictor that remained highly significant. The magnitude of the parameter estimates in both the full and reduced models is noteworthy as well, especially given that the outcome was log-transformed rather than unadjusted. Our expanded model suggests that net of a variety of other social, physical, mental, and behavioral factors, female NSHAP participants have an average logged C-reactive protein level 0.47 units higher than that of their male peers.

DISCUSSION

Overall, we found that gender (using sex identification as a proxy) predicts chronic inflammation status more strongly and reliably than any other social factor. Net of other forms of social disadvantage, participants that identify as female appear to have significantly higher logged levels of C-reactive protein. Other social factors reliably associated with inflammation status include race, education, and marriage. Identifying as black predicts significantly higher logged levels of C-reactive protein, but college education and marriage both predict significantly lower logged C-reactive protein levels. Income is inconsistently associated with inflammation status across different model specifications, but appears to predict logged C-reactive protein levels more reliably than asset-based measures of SES. We did not find significant associations between logged C-reactive protein and any of the four social support and communication variables in either bivariate or multivariate models.

These findings provide new insight into research concerning gender differences in the incidence (onset of new cases) and prevalence (total observed cases) of chronic inflammation. While previous literature suggests participants identifying as female are predisposed to increased risk of chronic inflammation (see McDade et al. 2010), it also presents less consistent results, and more modest parameter estimates for relationships between gender and C-reactive protein. In contrast, we found that gender consistently and strongly predicted C-reactive protein levels across models with markedly different parameter estimates. While this difference may reflect methodological distinctions rather than deficiencies, such difference calls for further exploration.

These findings also highlight the central role of gender in the experience of chronic inflammation. Whereas previous research reveals that women tend to seek medical care earlier and

more often than men and explains these patterns via gender identity (Courtenay 2000) or usual household roles (Umberson 1992), our analysis reveals that participants identifying as female are also more likely to experience chronic inflammation itself regardless of their use of medical (and thus diagnostic) care. Rather than simply a patterned health outcome, these findings suggest women's introduction to and experience of chronic inflammation may be especially tied to existing patterns of gender inequality, such as the second (Hochschild 1989) and third (Kleinman 2007) shifts that lead women to do more emotional, household, and interpersonal labor than men throughout the life course.

These findings also suggest there may be much to learn from examining relationships between behaviors typically associated with femininities and chronic inflammation. In their analysis, for example, McDade and associates (2010) incorporated behaviors and attributes like waist circumference, physical activity, smoking, sleep quality, household cleanliness, alcohol use, medication use, heart failure, and arthritis. Further, Kiecolt-Glaser and associates (2010) noted increased inflammation levels associated with increased stress levels, familial and social obligations, and perceptions of weight. Considering that many of the behaviors and attributes related to chronic inflammation – such as waist circumference or weight perception, familial and social obligations, household cleanliness, and hormonal medications – are typically associated with the performance of femininity (see Connell 1987), the combination of our analyses and previous findings suggest women's efforts to properly ''do gender'' (West and Zimmerman 1987) may represent a fundamental cause of chronic inflammation.

Our findings concerning racial minorities also mirror a large body of research demonstrating strong associations between non-white racial identities and adverse health outcomes. Considering that experiencing racism—implicitly and explicitly—can impact a wide variety of health outcomes for adults and children (Geronimus 1992; Hummer 1996), our findings suggest black racial identities may represent another fundamental cause of chronic inflammation. Further, our findings echo McDade and associates (2010) suggestion for greater attention to the ways racial minorities may experience adverse biological consequences due to their location within racially stratified societies.

While our findings concerning education mirror existing arguments concerning the health benefits of ''learned effectiveness'' (Mirowsky and Ross 2003), they also demonstrate nuances between different levels of educational attainment. Similarly, our mixed results concerning income lend weight to previous research suggesting complicated relationships between economic resources and health (Ross and Mirowsky 2000) and the potential for assets to better determine health predictions. Further, our results demonstrate that age was the only variable to elicit no meaningful ability to predict chronic inflammation, but this may be due to the relatively narrow age range of NSHAP respondents.

Age-related disadvantage remains a pressing concern in scholarship on late life populations, especially in a political climate where social welfare programs for older adults are being curtailed (Foner 2000). The fact that our income variable predicted NSHAP participants' inflammation status more reliably than the wealth measure used by McDade and colleagues (2010) may indicate that annual income remains a more salient predictor of health in late life because more U.S. residents depend on employment income for subsistence at older ages. The systemic transition from defined benefit to defined contribution retirement plans (Kruse 1995) may sharpen disparities already present in earlier portions of the life course. However, it may also indicate one or more positive trends. People may remain employed for longer periods of time because it makes them happy (Hao 2008). If older adults are succeeding in finding and sustaining paid

employment, this may reflect declining levels of the stark discrimination historically levied against older workers (see Bendick, Brown, and Wall 1999).

Taken together, these findings echo previous research into chronic inflammation while suggesting some potential nuances worthy of further consideration. Our findings for marriage, however, both supported and complicated existing literature. While our findings echo many studies demonstrating marital health benefits (Umberson 1992), we did not find other relationship forms to exhibit negative influences. Rather, we found no significant relationships between divorce, widowhood, never married, or cohabitation and C-reactive protein levels, but did identify significant associations linked to separation. Considering that marriage decreases the likelihood of chronic inflammation while other relationship structures neither decrease or increase a person's odds, it is possible that the health benefits associated with marriage do not come from the relationship itself, but rather, may be related to the privileged positon marriage occupies in our current social structure (see also Hochschild 1989). If marriage itself—as a form of relationship—provided health benefits, then one would expect other forms of relationships to suffer in comparison. Our findings, however, do not support this line of argument. Instead, we found that marriage conveys some benefits while other types of relationships exert little-to-no effect in any direction. This finding suggests health researchers should critically investigate the exact ways marriage may produce health benefits.

While the predictive strength of C-reactive protein levels, a vast array of social variables, and a nationally representative sample of older adults made the NSHAP ideal for our study, there are important limitations to our efforts. While we were able to assess each social factor's ability to influence overall associations, for example, this approach did not allow us to assess the synergistic influence of other constructs (like health behaviors and attributes) and may have introduced Type II errors due to the large models. Further, the use of C-reactive protein itself introduced limitations. Specifically, our understanding of C-reactive protein's role in the body continues to evolve (Pepys and Hirschfield 2003), and it may not capture every type of chronic inflammation with equal precision. As such, future research needs to continue developing the usefulness, precision, and nuances concerning C-reactive protein levels and relationships between large sets of variables.

There are also social structural limitations due to study design issues in the NSHAP. First, prior research indicated the usefulness of incorporating both racial and ethnic identities, but the NSHAP's measurements do not allow this. Second, prior research suggests examining gender and resultant social expectations, but the NSHAP only captures sex at birth, and thus leaves any nuanced examination of gender out of reach. Considering that not all females ''do gender'' as women and not all males ''do gender'' as men (West and Zimmerman 1987), our use of sex identification as a proxy for gender may miss out nuances related to gender display, identity, and socialization, and leads us to echo gender scholars calling for the incorporation of systematic gender measurements into large scale survey designs. Third, prior research suggests the importance of sexualities as moderating influences, but NSHAP only provides sexual behavior history and does not offer information on sexual preference. Finally, due to our wide assortment of variables, we were unable to assess lifestyle factors (but see McDade et al. 2010 for analyses of such factors), which may have influenced racial, gendered, and / or sexual influences upon chronic inflammation. Because of these limitations, we could not fully model social structural nuances in relation to chronic inflammation, and thus suggest future research take up this task to more fully articulate social nuances within and causes of chronic inflammation.

Finally, our project relied upon a cross-sectional design, which limits causal inference. We thus cannot claim to establish confirmatory evidence of causal influences within and between models. While we did succeed in developing preliminary suggestions of potential causal influences, we encourage readers to view this evidence prescriptive for future research. Further, we could not address the potential impact of current status bias because we only used one wave of data. We thus caution readers against extrapolating our findings to time points not actually captured within our specific study even though using C-reactive protein measurements (which typically remain relatively stable across time) helped limit potential problems.

Despite these limitations, our analyses direct attention to the centrality of gender in the experience of chronic inflammation. Echoing prior elaborations of fundamental social causes of health disparities due to socioeconomic inequalities (Link and Phelan 1995), our findings suggest that existing patterns of gender inequality and definitions of femininities place women at greater risk for chronic inflammation, and in so doing, curtail their overall health and quality of life. We would thus suggest that in many cases ''doing gender'' (West and Zimmerman 1987) might provide the foundation for adverse health effects regardless of the medical interventions we are able to provide. While fully understanding the implications of these findings will require critically evaluating and comparing the multitude of ways people develop and experience chronic inflammation, our analysis suggests that women's biological health may rely more heavily upon gender-based interventions than those of the medical variety.

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