The Social Patterns of a Biological Risk Factor for Disease: Race, Gender, Socioeconomic Position, and C-reactive Protein

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Running Head: Social Patterns of Biological Risk for Disease

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Abstract

Objective: Determine how fundamental causes of disease, especially race, get 'underneath the skin.' What is going on, at the biological level, which can help us understand persistent disparities in health?

Methods: Using the National Social Life, Health, and Aging Project (NSHAP) data, a study of older Americans aged 57-85, this study examines the link between race and C-reactive Protein (CRP).

Results:

Gender plays a critical role in explaining racial differences in CRP. While socioeconomic position better explains higher levels of CRP among Black men as compared to White men, healthy behaviors better explain higher levels of CRP among Black women as compared to White women. These findings are also striking, however, because of the strength of these racial differences and the relatively small explanatory power of generally powerful socioeconomic and healthy behavior mediators.

Discussion: Remaining race differences in CRP signal the need for research to examine mediators like discrimination. But this study makes clear that the pathways mediating race differences in CRP are different for men and women. Focusing on biological disease risk markers, such as CRP, provides a promising pathway to understand how social factors get 'under the skin', clarifying the links between social and biological phenomena.

Introduction

Race, class, and gender disparities in health have been persistent over historical time and over the individual life course leading social epidemiologists to argue that they are 'fundamental' causes of health (Link and Phelan 1995). Racial disparities in cardiovascular mortality, for example, have been rising over the latter half of the 20th century (Wyatt et al. 2005). Further, class and gender play critical roles in shaping race disparities. Race disparities in morbidity and mortality, in part, are a product of socioeconomic differences between Blacks and Whites. Race differences also vary by gender. For example, race differences are larger among women as compared to men for outcom4es like cardiovascular mortality and associated behavioral risk factors like obesity (Winkleby 1998).

But how do these fundamental causes, especially race disparities, get 'underneath the skin?' What is going on, at the biological level, which can help us understand persistent disparities in health? How do the social and biological connect? One promising pathway to understanding how racial disparities get underneath the skin is to focus on biological risk factors for disease. One such marker is C-reactive protein (CRP), a marker for inflammation. Inflammatory markers are an especially logical place to start given their association with cardiovascular disease, which is strongly patterned by race and is responsible for killing nearly 1 in 3 Americans (CDC 2010).

Using the National Social Life, Health, and Aging Project (NSHAP) data, a study of older Americans aged 57-85, this study examines the link between race and CRP. Because higher levels of CRP are correlated not only with existing problems, but also predictive of future disease, we are interested in exploring differences amongst those with similar existing health profile. Do Blacks have higher levels of CRP, controlling for existing health problems, as compared to Whites? If so, this indicates that Blacks are at a higher risk of developing new

health problems, especially cardiac problems. To what extent do socioeconomic and behavioral risk factors help explain race differences in CRP levels? And do race differences, and the importance of socioeconomic and behavioral risk factors in explaining race differences, vary by gender? We find race differences remain even after controlling for socioeconomic position (SEP) and a range of behavioral risk factors. But there are important gender differences. While SEP helps explain higher levels of CRP among Black men as compared to White men, healthy behaviors better explain higher levels of CRP among Black women as compared to White women. What is also striking about these findings is the strength of these racial differences and the relatively small explanatory power of generally powerful socioeconomic and healthy behavior mediators.

Fundamental Causes and Biological Pathways of Disease

Persistent health and mortality disparities by race have led social scientists to argue that race is a 'fundamental cause' of health (Link and Phelan 1995). Racial disparities in disease have been consistent and even growing over historical time. For example, while cardiovascular disease mortality has been rapidly declining since the 1950s, Whites have had disproportionate improvements as compared to Black Americans (Wyatt et al. 2005). Black Americans have simply not benefited as much as have White Americans from innovations to prevent and treat heart disease that emerged over the latter half of the twentieth century. Indeed, racial disparities in cardiovascular mortality were higher in the 1990s than they had been in the 1950s (Wyatt et al. 2005). This is also true for diabetes; the gaps are larger today than they were over 60 years ago (Wyatt et al. 2005). Racial differences also persist across the life course, even as different disease risks emerge across the life course. For example, Blacks have higher infant mortality, but

also a higher risk for later life diseases such as diabetes and hypertension (Williams and Collins 1995).

Indeed, current evidence indicates persistent racial disparities across multiple disease categories. This is especially true for cardiovascular disease, hypertension, strokes and diabetes (Gorelick 1998). For example, Black Americans have twice the risk for cardiac arrest, and compared to Whites are less likely to survive the arrest (Becker et al. 1993). Further, stroke rates are twice as high among Black Americans as compared to Whites (Gorelick 1998). There is some evidence that part of this difference is due to differences in modifiable behaviors. For example, Blacks are at disproportionate risk for obesity, limited physical activity, and smoking (Wang and Beydoun 2007; Brownson et al. 2000; Winkleby et al. 1998). These behaviors, in turn, are especially predictive of cardiovascular diseases, strokes, hypertension and diabetes.

But, fundamental causes, especially race, class, and gender also interact with each to produce particular patterns of disease. Indeed, racism, especially as it operates to shape SEP, is at the heart of the explanation for why race operates as a fundamental cause for disease (Wyatt et al. 2005). Historical and current institutional racism, present in the labor market, educational system, and even the housing sector, have shaped persistent socioeconomic differences between Blacks and Whites (Wilson 1987). These socioeconomic differences, in turn, have shaped health and mortality differences, partially via behavioral risk factors like obesity and physical inactivity (Lantz et al. 1998; Herd 2006; Herd et al. 2007). Ultimately, SEP plays an important, often dominant role, in explaining race differences in health (Hayward et al. 2000).

But sometimes race disparities remain even after accounting for SEP and behavioral risk factors. For example, in many instances, adverse cardiovascular outcomes are observed in African-American women even after taking into account the effects of SEP, access to care, disease status, and behavioral risk factors (Jha et al. 2003). Among older people, race differences

in cardiovascular disease remain even after controlling for SEP (Rooks et al. 2002). One hypothesis for these unexplained race differences is racial discrimination. Indeed, there is an emerging literature documenting that discrimination may directly affect health by causing stress (Friedman et al. 2009).

Gender also plays an important role in understanding racial and socioeconomic disparities in disease. Some disparities appear to be particularly acute for African American women. For example, the Jackson Heart Study (JHS), found that age adjusted cardiovascular mortality rates for African American women were 75% higher than in European American women compared to 47% higher in African American men than in European American men. In addition, the Black surplus in prevalence of coronary heart disease over Whites is larger among women than among men (Taylor et al. 2005).

Behavioral risk factors also vary importantly across gender and race groups. For example, Gallant and Dorn (2001) find race differences in obesity rates among women, with higher rates for Black women, but not among men. In general, Black women have substantially higher behavioral risk factors, such as obesity and physical inactivity, for cardiovascular disease as compared to White women (Winkleby 1998).

But how do these fundamental causes, specifically racial disparities, get 'underneath the skin?' What is going on, at the biological level, which can help us understand persistent racial disparities in health? One promising pathway to understanding how racial disparities get underneath the skin is to focus on biological markers for disease. One such marker is C-Reactive Protein (CRP), an acute phase protein that is considered a marker for systemic inflammation. And if we want to understand how racial disparities get under the skin, inflammatory markers are a logical place to start. Inflammatory proteins are strongly associated with cardiovascular disease, which kills nearly 1 in 3 Americans (CDC 2010). And as already noted, cardiovascular

disease is strongly patterned by race. CRP predicts increased risk of later cardiovascular events in healthy individuals as well as increased risk of mortality in patients with a history of cardiovascular disease (Abraham et al. 2007; Bermudez et al. 2002; Tracy et al. 1997; Verma et al. 2005; Willerson and Ridker 2004). Indeed there is a sufficiently strong link between CRP and later cardiovascular disease, especially in women, that limited clinical screening for CRP levels is now recommended (Mitka 2003). Importantly, in many studies this marker independently predicts cardiovascular disease above and beyond traditional risk factors (Verma et al. 2005; Abraham et al. 2007; Ridker et al. 2000; Stork et al. 2006).

To date, however, there is limited research exploring how risk factors (especially gender, class, and behavioral factors) vary and interact to mediate the relationship between race and CRP levels. The research, has, however, demonstrated that race, gender, and SEP separately predict CRP levels (Alley et al. 2005; Friedman and Herd 2010; Lubbock et al. 2005; McDade et al. 2010). For example, McDade and colleagues (2010) employed NSHAP data to explore whether race and class independently predict CRP levels, but they did not explore how these patterns varied by gender. Further, these analyses only controlled for health measures (such as chronic conditions) in the final model. Thus, for example, when they examined how the introduction of socioeconomic variables affected racial variation, they did not account for variation across race groups in current chronic conditions and existing health problems.

Consequently, this study focuses on three key research questions. First, baseline analyses explore whether Blacks have higher levels of CRP, controlling for existing health problems, as compared to Whites. If so, this indicates that Blacks are at a higher risk of developing new health problems, especially cardiac problems. Next, to what extent does SEP explain race differences in CRP levels amongst those with similar health profiles? And to what extent do behavioral risk factors explain race differences in CRP levels, after controlling for SEP, amongst

those with similar health profiles? Finally, do race differences, and the importance of socioeconomic and behavioral risk factors in explaining race differences, vary by gender?

Methods

Data

Collected from summer 2005 to spring 2006, the National Social Life, Health, and Aging Project (NSHAP) is a nationally representative study of non-institutionalized older adults aged 57 to 85. The NSHAP data contains information on the demographic characteristics; romantic, sexual, and social relationships; and physical and mental health—including biomarkers—of 3,005 American men and women aged 57 to 85. The final response rate was 74.8% (O'Muircheartaigh, Eckman, and Smith 2009). Most data were collected in an in-home interview. Data were also collected via take-home questionnaire that respondents completed and mailed back to researchers.

The NSHAP sample was chosen from a multistage area probability design selected by the Institute for Social Research (ISR) for the Health and Retirement Study (HRS) (O'Muircheartaigh et al. 2009). NSHAP selected 4,400 possible respondents from the HRS sample surplus (O'Muircheartaigh et al. 2009). NSHAP oversampled Blacks and Latinos and balanced age and gender subgroups (O'Muircheartaigh et al. 2009). Appropriate weights are used in analyses to account for oversampling and non-response.

Measures

Outcome

C-reactive protein (CRP) was obtained in NSHAP via blood spot samples, which were collected on filter paper (Williams & McDade, 2009). The samples were processed in the Laboratory for Human Biology Research at Northwestern University. This method of CRP data

collection has been shown to be precise and reliable (McDade et al. 2004). Among the 2032 respondents who provided samples, 95% had usable samples.

Covariates

Demographics: Race is based on self reported data. We classified respondents as Black/non-Hispanic, White non-Hispanic, Hispanic, and other. The "other" category was comprised of individuals from multiple groups whereby there was not a large enough sample to do separate analyses. We employ both age and age squared terms to explore the non-linear relationship between age, race, and CRP, however, final analyses employ only an age centered (at age 68) term. Sex is male or female.

SEP: We employed three measures of SEP. The first measure is educational attainment. Respondents were asked their highest completed degree. Based on this information we created 5 categories: no high school degree, high school degree, some college, college, and graduate degree. We also included a measure of income. Income includes pre-tax household income from wages, pensions, social security, and government assistance. Income was treated as a continuous variable in analyses examining linear associations between income and CRP. However, results from earlier studies have shown that low income groups, but not moderate income groups, had significantly higher levels of inflammatory markers compared to high income groups. To allow for such non-linear associations we also stratified the sample into income quintiles. The income ranges that corresponded to each quintile were: Q1: $17,838 or less; Q2: $17,839 – $35,037; Q3: $35,038 – $50,161; Q4: $50,162 – 76,809; Q5: $76,810 or more. Each quintile was dummy-coded and included in statistical analyses with the bottom quintile serving as the referent. Because there was significant numbers of missing data on income, we employed multiple imputation to impute 359 of 1541 cases income values. Multiple

imputation is a better strategy for missing data issues than stepwise deletion because it reduces concerns about selection that could bias the findings (Rubin 2004).

Physical Health: Physical health status was assessed using self-reported measures. There are a number of diseases particularly linked to inflammation. Thus, we included a range of the most relevant health measures. Participants were classified as having high blood pressure or hypertension, diabetes or high blood sugar if they reported that a medical doctor had ever told them they had these conditions. Individuals are classified with heart disease if they report ever having a heart attack, ever being treated for heart failure, or having had an operation to unclog or bypass the arteries in your legs. Further, we include a control for general self assessed health. Respondents answered the following question "How would you rate your physical health: excellent, very good, good, fair, or poor?" Self reported general health appears to capture much of the health related variation in CRP. Sensitivity analyses included a broader range of health conditions, including a Center for Epidemiological Studies 11-item depression scale , but the findings did not vary from the findings presented here and the inclusion of all health measures started to introduce issues with multicollinearity.

Prescription Drugs: Many drugs, both prescription and over-the-counter, including antihypertensive, cholesterol-lowering, and anti-depressant, have been shown to have anti-inflammatory properties (Jaim and Ridker 2005; Kenis and Maes 2002; Pradhan et al. 2002; Ridker et al. 1999). Further, steroid medications, particularly as part of a hormone replacement regimen, have been shown to increase CRP levels. Dummy coded variables indicating current use of these medications were included in all analyses. Specifically, we included measures for cholesterol absorption inhibitor medication, combination antihyperlipidemic medication, estrogens, progestin, nonsteroidal anti-inflammatories, steroids, and antidepressants. These

drugs were categorized using the Multum Drug Database: Lexicon Plus version (Qato et al., 2008).

Healthy Behaviors: Height and weight were used to calculate body mass index (BMI; weight in kilograms divided by the square of height in meters). We also included smoking measures, which capture whether the respondent is a current smoker, former smoker, or never smoked. Finally, we include measures of physical activity. These include little exercise (fewer than four times per month) moderate (1-2 times per week) heavy (3 or more times per week).

Table 1 presents descriptive statistics on the dependent variable and covariates.

[Insert Table 1 Here]

Analytic Techniques

The relationship between C-reactive protein and socioeconomic status, race/ethnicity, and gender was examined using ordinary least-squares regression, which included weights and statistical adjustments for the NSHAP sample frame (O'Muircheartaigh, Eckman, and Smith 2009). Because C-reactive protein levels were highly skewed, values were log-transformed. Prior research (Pearson et al. 2003) suggests that plasma C-reactive protein levels greater than 10 mg/L should be excluded. NSHAP collected C-reactive protein levels from dried blood spots. Different cut-off criteria for maximum C-reactive protein levels have been established for plasma versus blood spot samples (McDade, Burhop, and Dohnal 2004). A plasma level of 10 mg/L roughly corresponds to a blood spot level of 8.6 mg/L, so C-reactive protein levels greater than 8.6 mg/L are excluded from the analytic sample (see McDade, Lindau, and Wroblewski 2010).

The first set of analyses is conducted on the full sample to address two research questions. First, are there race differences in CRP holding current health status constant? Thus, model 1 includes gender, race/ethnicity, age centered at age 68, self-rated physical health, medications, and health conditions. Second, what explains race differences in CRP levels?

Model 2 tests for the role of SEP by including income and education. Model 2 also includes tests for race and gender and race and age interactions. Model 3 tests for the role of healthy behaviors by including body mass index (bmi), smoking behavior, and exercise frequency.

The second set of analyses breaks the sample down by gender and age (ages 57-74 and 75-85). Both prior research on CRP (Mitka 2003; McDade et al. 2010) and our own sensitivity analyses testing interaction terms, indicate that sub-analyses by gender and age are appropriate. Our own sensitivity analyses confirmed this given important gender and age variation in CRP levels. Further, prior work using NSHAP, in addition to our own sensitivity analyses, indicate significant interactions between race, age and CRP levels (McDade et al. 2010). At older ages, between 75 and 80, there is significant convergence in CRP levels between Blacks and Whites. This convergence is nearly universally consistent when examining race and morbidity and mortality trends across the life course (Johnson 2000). It is generally assumed to be a function of mortality selection. Blacks who survive to the oldest ages are relatively select compared to White survivors, hence the convergence in morbidity and mortality profiles (Johnson 2000).

The second set of analyses aims to establish how the pathways mediating the relationship between race and CRP levels vary by gender and age. Model 1 includes race, age, race and age interacted, self-rated physical health, medications, and health conditions to establish baseline race differences for women as compared to men. Model 2 adds socioeconomic variables (education and income) to establish the extent to which SEP mediates the relationship between race and CRP for women as compared to men. Model 3 adds body mass index (bmi), smoking behavior, and exercise frequency to test the extent to which these variables mediate the relationship between race and CRP levels differentially for men as compared to women.

[Insert Table 2 Here]

Results

Amongst those with similar health profiles, including controls for prescription drug use, CRP levels are significantly higher amongst Blacks as compared to Whites in this sample of those aged 57 to 85 (Table 2, Model 1). Black respondents had CRP levels 26 percent higher than comparable Whites. But that variation is age graded (Table 2, Model 2). At older ages (sensitivity analyses indicate above age 75-80), those differences become insignificant, both statistically and in terms of coefficient size (results shown in greater detail in Table 3). In short, there is racial crossover in CRP levels amongst the oldest sample respondents. This finding is robust given that NSHAP oversampled Blacks over age 80—though models are weighted to adjust for oversampling by race, among other characteristics.

But what explains these race differences among those with similar health profiles? SEP and gender and race interactions play an important role (Table 2, Model 3). Compared to those without a high school degree, those with college degrees had CRP levels 19 percent lower and those with professional degrees had levels that were 31 percent lower. Further, there is an interaction between race and gender indicating that Black women had the highest CRP levels relative to other groups. The inclusion of both of these terms actually eliminates the relationship between race and CRP.

Risky health behaviors further impact racial variation in CRP (Table 2, Model 4). The inclusion of BMI, physical activity, smoking patterns and alcohol consumption eliminate the gender and race interaction, but the basic race effect remerges to marginal statistical significance. Compared to Whites, Blacks have CRP levels that are 25 percent higher than Whites, although this difference is marginally significant.

Given the interactions between race and gender and age in Table 2, as well as prior research focused on CRP that indicates important race and gender variation in CRP levels (Mitka

2003), Table 3 breaks the analyses down by age (those over and under age 75) and by gender to get a clearer sense of how the relationship between race and CRP varies among these subgroups.

[Insert Table 3 Here]

Table 3 demonstrates that race differences shrink significantly after age 75, but that these differences shrink more rapidly for men as compared to women. Basic race differences in CRP are nearly identical in magnitude for women and men under age 75 (Model 1a and Model 1b), while the race differences are not significant for either women or men age 75-85 (Model 1c and Model 1d). The difference in race coefficients over age 75 between men and women, however, is statistically significant (results not presented here). In short, race differences shrink much more dramatically for men at older ages compared to women.

Models 2a-2d demonstrate that SEP is more important in explaining racial variation among men as compared to women. Among women, the race difference actually increases slightly, once controlling for SEP. SEP is not a significant predictor of CRP levels among women under age 75 (model 2a), though over age 75 the relationship between education and CRP is statistically significant (model 2c). Higher educational attainment is correlated with lower CRP levels. SEP, however, does not help explain why Black women have higher CRP levels as compared to White women. Among men under age 75, the race coefficient shrinks by 17 percent and becomes marginally significant ($p<.10$) (model 2b). Further, the education coefficients are large and statistically significant for men among both those under and over age 75, though the relationship is stronger for those under age 75. Higher educational attainment is correlated with lower CRP levels.

Models 3a-3d demonstrate that healthy behaviors are more important in explaining racial variation among women as compared to men under age 75. Among women under age 75, behaviors (BMI and smoking) explain 16 percent of the higher levels of CRP among Black

women as compare to White women (model 3a). But the race coefficient remains relatively large and significant. Contrastingly, among men under age 75, while healthy behaviors are both significant predictors of CRP levels and help explain educational variation in CRP levels, they do not help explain the racial differences (model 3b). Indeed, the race coefficient increases slightly in both size and significance once behaviors (BMI, exercise, and smoking) are included in the models. Among women and men aged 75-85 (models 3c and 3d), race differences in CRP are not significant, though BMI is independently associated with CRP.

Discussion

This study lends new insights into how racial disparities in health get "underneath the skin." The findings emphasize the importance of gender and age in understanding race differences in a biological risk factor for disease. In short, gender and age play an important role in explaining higher CRP levels amongst Black compared to White Americans aged 57 and older with similar health profiles. In particular, while SEP better explains higher levels of CRP among Black men as compared to White men, healthy behaviors better explain higher levels of CRP among Black women as compared to White women. But what is also striking about these findings is the strength of these racial differences and the relatively small explanatory power of generally powerful socioeconomic and healthy behavior mediators.

Similar to other studies focused on general age trends in racial health differences, we find diminishment in racial differences for this biological risk factor for disease at the oldest ages, but the patterning is different for men and women. In short, there is significant convergence after age 75, especially for men. For men, race differences in CRP levels not only converge over age 75, but even reverse themselves, though the difference is not statistically significant. Contrastingly for women, race differences diminish over age 75, but women still have slightly higher levels, though the difference is not statistically significant. It is unlikely that this

convergence is a cohort effect resulting from cross sectional data. Indeed, racial differences should be larger in older cohorts given general improvements in economic well-being and declining discrimination for younger cohorts. Healthy survivor selection likely plays a role in these declining differences at the oldest ages. However, it is important to point out that the age at which black and white mortality rates converge does not vary between men and women. Whereas in our sample there is a large and significant gender difference in the age patterning of racial convergence in CRP levels.

We find that SEP, particularly education, and healthy behaviors help explain racial differences in the biological risk for disease. But the explanatory potential of SEP and healthy behaviors varies for men and women. SEP differences only help explain the race differences for men, while BMI only helps explain race differences for women. And neither explains more than one-fifth of the relationship. SEP explains 20 percent of the race difference for men ages 57-75, while BMI explains 10-15 percent of the relationship for women. Moreover, while education was predictive of CRP, income was not. Sensitivity analyses that included both linear and varying nonlinear functions of income all produced the same result.

What does this mean? A few studies have found smaller effects of educational attainment for women as compared to men on general health outcomes, but this finding is by no means universal. In this sample, the lacking explanatory power of educational attainment in explaining racial variation among women appears to be a product of the generally weak relationship with education and CRP among women. The difference in BMI as a mediator for race differences for women as compared to men is rooted in the fact that there are no meaningful race differences in BMI among men, but relatively large race differences among women. Finally, remaining racial differences in CRP levels, even after controlling for SEP and healthy behaviors does parallel some findings regarding race differences in cardiac disease and cardiac

disease mortality risk. Some studies find that these risks remain, to some extent, even after controlling for SEP and behavioral factors (Jha et al. 2003).

What explains the remaining race differences in CRP levels? One hypothesis that should be pursued in future work is the potential effects of stress, particularly discrimination. There is growing evidence that stress generally is linked to higher levels of inflammation. A recent meta-analysis of laboratory studies concluded that acute psychological stressors produce increases in circulating levels of inflammatory proteins, including CRP (the effects were marginally significant at P<.09) (Steptoe et al. 2007). Greater perceived stress in a community-based sample and more frequent interpersonal stressors in a sample of adolescents were associated with higher circulating levels of CRP (McDade et al. 2006; Fuligni et al. 2009). Exposure to low SEP and harsh family environments in childhood increases the likelihood of higher levels of CRP in adulthood (Taylor et al. 2006). In addition, reduced stress associated with practicing yoga and being married (in men) predicted lower levels of CRP (Kiecolt-Glaser et al. 2010).

Racial discrimination is thought to represent a particularly noxious type of chronic stress that is linked to a range of adverse health outcomes in Blacks, and there is recent evidence that chronic discrimination is associated with higher levels of CRP in Black adults (Williams 1997; Williams et al. 2008; Lewis et al. 2010). For example, discrimination, of multiple types, is linked to heart disease-coronary calcification in Black American women (Lewis et al. 2010). Discrimination is also linked to health behaviors, including substance abuse among Blacks (Borrell et al. 2007). Future research must consider chronic stress generally, and experiences of discrimination specifically, as potential contributors to racial differences in systemic inflammation.

While this study does not fully explain what mediates race difference in CRP, it does make clear that the pathways mediating race differences in CRP may be different for men and

women. Future work will likely need to consider the role that gender plays when examining hypothesized mediators. Ultimately, focusing on biological disease risk markers, such as CRP, however, provides a promising pathway to understand how social factors get 'under the skin.'

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Table 1. Descriptive Statistics. Weighted Means and Proportions (N=1541)

	Mean/Proportion	SE	Range
CRP (mg/L)	2.12	0.06	.102-8.572
Log CRP	0.28	0.03	-2.28-2.15
Sociodemographic factors			
Age (years)	70.59	0.23	57-85
Gender (female)	0.53	0.01	
Race/ethnicity			
White, non-Hispanic	0.62	0.02	
Black, non-Hispanic	0.20	0.01	
Hispanic (any race)	0.15	0.01	
Other	0.02	0.00	
Education			
Less than high school	0.29	0.01	
High school/GED	0.36	0.01	
Some college/associates/vocational certificate	0.16	0.01	
Undergraduate degree	0.11	0.01	
Post-undergraduate degree	0.09	0.01	
Household income			
1st quintile			17,838 or less
2nd quintile			$17,839 – $35,037
3rd quintile			$35,038 – $50,161
4th quintile			$50,162 – 76,809
5th quintile			$76,810 or more
Physical health factors			
Comorbidities			
Heart failure	0.10	0.01	
Heart attack	0.12	0.01	
Angioplasty, cardiac catheterization, or angiogram	0.27	0.01	
Hypertension	0.57	0.01	
Diabetes	0.23	0.01	
Arthritis	0.52	0.01	
COPD/emphysema	0.11	0.01	
Asthma	0.10	0.01	
Stroke	0.08	0.01	
Dementia	0.01	0.00	
Self-rated physical health	3.19	0.03	1= poor, 5= excellent
Medication use			
Cholesterol absorption inhibitors	0.05	0.01	
Antihyperlipidemic combinations	0.02	0.00	
Estrogens	0.05	0.01	
Progestins	0.01	0.00	
Nonsteroidal anti-inflammatory agents	0.11	0.01	
Steroids*	0.05	0.01	
Antidepressants **	0.11	0.01	
Health Behaviors			
Body mass index	29.05	0.18	15.55-67.30
Smoking			
never	0.44	0.01	
former	0.41	0.01	
current	0.15	0.01	
Exercise			
1-3 times per month or less	0.21	0.01	
1-2 times per week	0.15	0.01	
3 or more times per week	0.64	0.01	

Notes: CRP = C-reactive protein; GED= general education diploma

* includes androgens and anabolic steroids, nasal steroids, and inhaled corticosteroids

** includes ssri, tricyclic, phenylpiperazine, tetracyclic, ssnri, and other miscellaneous antidepressants

Table 2. Ordinary Least Squares Coefficients for C-Reactive Protein Among Older Adults All Ages (N=1541)

	Model 1	Model 2	Model 3	Model 4
Race/Ethnicity				
White, non-Hispanic-omitted	-	-	-	-
Black, non-Hispanic	0.263 **	0.322 ***	0.114	0.257 †
Hispanic	0.026	0.026	-0.075	-0.045
Other	-0.153	-0.139	-0.256	-0.121
Age (centered at 68)	-0.007 †	-0.004	-0.005	0.003
Female	0.208 **	0.207 **	0.122 †	0.191 †
Race/EthnicityxAge (centered at 68)				
White, non-Hispanic-omitted		-	-	-
Black, non-Hispanic		-0.023 †	-0.024 *	-0.027 *
Hispanic		0.004	0.005	0.004
Other		-0.007	-0.011	-0.020
Race/EthnicityxFemale				
White, non-Hispanic-omitted			-	-
Black, non-Hispanic			0.316 †	0.081
Hispanic			0.065	-0.019
Other			0.319	0.284
Education				
Less than high school-omitted			-	-
High school/GED			-0.130	-0.115
Some college/associate's degree			-0.122	-0.082
College degree			-0.191 †	-0.133
Professional degree			-0.311 *	-0.224 †
Income quintile				
1-omitted			-	-
2			0.067	0.004
3			0.090	0.025
4			0.072	0.071
5			0.034	0.023
Bmi				0.051 ***
Smoking				
Never (reference)				
Former				0.128 *
Current				0.249 **
Exercise				
Once a week or less (reference)				
1-2 times per week				0.102
3+ times per week				-0.032
Constant	0.453 **	0.444 **	0.548 **	-1.008 ***
F-statistic	5.300 ***	5.04 ***	3.75 ***	6.57 ***
df	(23, 1515)	(26, 1512)	(37, 1498.1)	(42, 1493.7)

Sources: Data are from National Social Life, Health, and Aging Project (NSHAP).
Notes: : Significance levels for two-tailed tests of coefficients: †p<0.1* p<0.05; ** p<0.01; *** p<0.001. All models control for physical health and medication measures described in the methods section.

Table 3. Ordinary Least Squares Coefficients for C-Reactive Protein Among Older Adults by Age 57-74 and Age 75-85 (N=1541)

	Model 1a Women Ages 57-74	Model 1b Men Ages 57-74	Model 1c Women Ages 75-85	Model 1d Men Ages 75-85	Model 2a Women Ages 57-74	Model 2b Men Ages 57-74	Model 2c Women Ages 75-85	Model 2d Men Ages 75-85	Model 3a Women Ages 57-74	Model 3b Men Ages 57-74	Model 3c Women Ages 75-85	Model 3d Men Ages 75-85
Race/Ethnicity												
White, non-Hispanic (reference)	-	-	-	-	-	-	-	-	-	-	-	-
Black, non-Hispanic	0.400 **	0.358 *	0.287	-0.423	0.428 **	0.285 †	0.283	-0.478	0.356 **	0.321 †	0.149	-0.303
Hispanic	0.038	0.049	0.043	-0.094	0.029	-0.161	-0.096	-0.244	0.020	-0.090	-0.205	-0.073
Other	0.056	-0.165	-0.277	-0.382	0.085	-0.125	-0.101	-0.534	0.305	-0.013	-0.507	-0.393
Age	-0.008	-0.007	-0.015	0.013	-0.007	-0.008	-0.017	0.010	-0.002	0.003	-0.013	0.028
Education												
Less than high school (reference)					-	-	-	-	-	-	-	-
High school/GED					-0.107	-0.313 *	-0.263	0.165	-0.064	-0.282 *	-0.241	0.180
Some college/associate's degree					-0.008	-0.213	-0.560 *	-0.052	0.079	-0.188	-0.454 †	-0.069
College degree					-0.095	-0.398 *	-0.298	-0.026	-0.100	-0.295	-0.266	0.047
Professional degree					0.078	-0.527 **	-0.970 *	-0.582 †	0.120	-0.377 †	-0.835 *	-0.435
Income quintile												
1 (reference)					-	-	-	-	-	-	-	-
2					-0.063	0.233	0.346 †	-0.427	-0.178	0.137	0.233	-0.335
3					0.118	0.099	0.319	-0.368	0.025	0.043	0.245	-0.420
4					0.166	0.194	-0.015	-0.536	0.137	0.173	0.007	-0.447
5					0.154	0.029	0.152	-0.435	0.176	-0.014	0.118	-0.334
Bmi									0.051 ***	0.033 **	0.065 ***	0.072 ***
Smoking												
Never (reference)									-	-	-	-
Former									0.152 †	0.202 †	-0.153	0.255
Current									0.115	0.563 ***	-0.156	0.112
Exercise												
Once a week or less (reference)									-	-	-	-
1-2 times per week									-0.056	0.308 †	-0.214	0.281
3+ times per week									-0.085	-0.035	-0.008	0.090
Constant	1.129 †	0.734	1.734	-0.179	1.023	0.923	1.964	0.460	-0.785	-0.978	0.126	-3.440
F-statistic	4.59 ***	14.21 ***	90.82 ***	1.97 *	3.48 ***	3.53 ***	22.26 ***	1.54 †	4.65 ***	4.58 ***	6.78 ***	2.62 ***
df	(22, 518.0)	(20, 526.0)	(22, 229.0)	(20, 172.0)	(30, 508.5)	(28, 516.9)	(30, 219.3)	(28, 163.2)	(35, 503.9)	(33, 512.1)	(35, 215.1)	(33, 158.4)
N	543	549	254	195	543	549	254	195	543	549	254	195

Source : Data are from the National Social Life, Health and Aging Project (NSHAP)

Notes : Significance levels for two-tailed tests of coefficients: †p<0.1* p<0.05; ** p<0.01; *** p<0.001. All models control for physical health and medication measures outlined in the data and methods section.