Chronic Inflammation at the Intersection of Race and Gender: SES and Health Behaviors as Mechanisms

Aliza D. Richman
The Pennsylvania State University
Tetyana Pudrovska
The Pennsylvania State University

Abstract

Despite the knowledge that inflammatory risk differs on account of race and gender, little is known about the factors that contribute to these disparities. This paper explores obesity, socioeconomic disadvantage, educational attainment, smoking status, alcohol consumption, and physical activity as six potential mechanisms that underlie the joint and synergistic effects of race and gender on inflammation measured as C-reactive protein. Our study is based on a sample of 11,539 non-Hispanic white and non-Hispanic black adults from the National Health and Nutrition Examination Survey (NHANES). We use logistic regression and a decomposition into direct and indirect effects in a logistic mediation model. We find a strong interactive effect of gender and race, such that black women have significantly higher levels of chronic inflammation than other race × gender groups. Our preliminary findings indicate that obesity, socioeconomic disadvantage, educational attainment, alcohol consumption, and physical activity explain a higher level of inflammation among black women compared to other groups. The gap between black women and others diminishes when we adjust for each of these characteristics. In contrast, smoking status suppresses the gender × race effect such that the gap between black women and others becomes even greater when we include smoking.

Background

It is now well documented that chronic inflammation and dysregulation of the immune function are related to many chronic diseases of aging (heart disease, cancer, Alzheimer's) that are now major causes of death (Balkwill and Mantovani 2001; Erlinger 2004; Harris et al. 1999; Kuller et al. 1996; Pai et al. 2004; Rost et al. 2001). Moreover, these physiological processes are not randomly distributed in the population. A review of studies examining C-reactive protein (CRP), an acute-phase protein that increases in concentration in response to inflammation, found that non-Hispanic whites have the lowest CRP levels, while non-Hispanic blacks have the highest (Nazmi and Victora 2007). Among women, non-Hispanic blacks experience the highest allostatic load (a cumulative measure of wear and tear on the body's physiological systems) relative to other racial and ethnic groups (Chyu and Upchurch 2011). Whereas social epidemiological and demographic research is accumulating evidence linking gender and race to the biological mediators of mortality and morbidity, the mechanisms underlying this relationship have yet to be empirically identified.

This paper employs the intersectionality perspective (Choo and Ferree 2010) as well as the biosocial stress perspective (Krieger 2005) to analyze six potential mechanisms underlying how gender and race become embodied and influence inflammation levels. Our six pathways include obesity, socioeconomic disadvantage, educational attainment, smoking status, alcohol consumption, and physical activity. These mechanisms were selected based on studies consistently documenting that each of these characteristics is affected by gender and race while

also affecting inflammation (Deverts et al. 2012; Pampel, Krueger, and Denney 2010; Flegal et al. 2010; Visser and Bouter 1999; Petersen and Pedersen 2005; Gonçalves et al. 2011; Greenberg and Obin 2006; Imhof et al. 2001). Based on the "double jeopardy" and the accumulation of risks perspectives (Robert 1999; Ben-Shlomo and Kuh 2002), we hypothesize that the intersecting context of two disadvantaged social statuses (being a woman and being black) will be related to elevated inflammation compared to other race × gender groups. Moreover, we hypothesize that higher levels of chronic inflammation among black women will partly reflect their higher levels of obesity and socioeconomic disadvantage, and lower levels of education and inflammation-reducing health behaviors. The social location of being a black woman enhances exposure to chronic social stressors, including poverty and racial discrimination, that become embodied as inflammatory responses directly or through proinflammatory health behaviors that can be viewed as ways of coping with socially structured stressors.

Data

The present paper uses data from the National Health and Nutrition Examination Survey (NHANES), a nationally representative study of adults and children in the United States. Focusing on nutrition and health, NHANES samples approximately 5,000 individuals biannually and combines in-person interviews with physician-performed physical examinations and laboratory tests. Utilizing data from NHANES III (1988-1994) linked to 2010 mortality files, the analytic sample includes 11,539 non-Hispanic white and non-Hispanic black men and women ages 25 to 90 years (non-Hispanic white will hereafter be referred to as white and non-Hispanic black will hereafter be referred to as black).

Dependent Variable

The outcome measure is inflammation, measured by C-reactive protein (CRP). Clinically, CRP levels below 1 mg/L indicate low inflammation, levels between 1 mg/L and 3 mg/L specify average inflammatory risk, and levels above 3 mg/L demonstrate elevated inflammation (Ridker 2003). We dichotomize CRP levels into a binary measure, with a one denoting average or elevated CRP levels (>1 mg/L). Among the analytic sub-sample, the distribution of those with average or elevated inflammation is 9.71 percent, with black women overly represented in the category (14.24 percent; see Table 1).

Key Independent and Control Variables

The primary independent variables are race, gender, and an interaction between the two (black females). Race includes white and black individuals, with the majority of the sample of white origin (60.61 percent). Females are 53.72 percent of the sample and black females account for 21.23 percent of respondents. Due to the likelihood of age, systolic blood pressure, diastolic blood pressure, cholesterol, triglycerides, and glycated hemoglobin influencing CRP levels, we control for these variables in our model.

Focal Mechanisms

Of principal interest in this study are six focal mechanisms that potentially explicate the relationship between race and gender and CRP: obesity, socioeconomic disadvantage, educational attainment, smoking status, alcohol consumption, and physical activity. Obesity is a binary measure based on respondent's body mass index, with a body mass index of 30 or above indicating obesity. NHANES calculates a poverty income ratio as a measure of socioeconomic

status. Using the appropriate poverty threshold given family size and composition, the ratio values reflect those living below the official poverty line (<1.00), those living at the official poverty line (1.00), and those living above the poverty level (>1.00). We characterize socioeconomic disadvantage as individuals with a poverty income ratio less than 2. Educational attainment divides the sample into three substantive groupings: those with less than a high school education (reference group), those with a high school education, and those with more than a high school education. Smoking status is categorized into those who have never smoked, former smokers, and current smokers, with the former serving as the reference category. Alcohol consumption divides respondents into one of three categories: light drinkers, heavy drinkers, or non-drinkers (reference group). Light drinkers include women who consume one alcoholic beverage per day and men who consume two alcoholic beverages per day. Heavy drinkers are those women and men who exceed one drink per day and two drinks per day, respectively. To capture physical activity, we create a binary variable which measures those respondents who have participated in jogging/running, swimming, bicycling, or aerobics in the past month.

Table 1: Summary Statistics for Study Variables by Race and Gender

	Full Sample	White Men	White Women	Black Men	Black Women
CRP >1 mg/L	9.71%	7.24%	9.98%	7.78%	14.24%
Mechanisms:					
Obesity	24.61%	18.58%	23.15%	20.95%	37.96%
Socioeconomic Disadvantage	16.41%	6.90%	10.22%	23.53%	32.37%
Educational Attainment					
Less than High School	35.41%	33.78%	30.27%	44.15%	37.96%
High School	33.19%	28.44%	35.85%	32.41%	36.08%
More than High School	31.40%	37.78%	33.88%	23.44%	25.96%
Smoking Status					
Current Smoker	25.87%	23.67%	20.57%	38.38%	26.20%
Former Smoker	26.63%	43.42%	22.94%	23.53%	12.69%
Never Smoked	47.50%	32.91%	56.50%	38.09%	61.10%
Alcohol Consumption					
Light Drinker	57.69%	58.92%	57.00%	59.90%	55.22%
Heavy Drinker	6.85%	7.86%	5.60%	10.36%	4.41%
Never Drinks	35.46%	33.22%	37.40%	29.74%	40.37%
Physical Activity	57.31%	57.44%	55.88%	64.68%	53.02%
Controls:					
Age	52.78	56.52	56.59	47.61	46.43
BP Systolic	127.52	130.10	126.86	128.76	124.06
BP Diastolic	74.73	76.08	72.15	78.16	73.95
Cholesterol	207.02	205.59	215.02	199.28	203.27
Triglycerides	135.61	158.06	143.45	119.52	107.65
Glycated Hemoglobin	5.55	5.51	5.45	5.66	5.68

Analytic Approach

Given that our dependent variable is dichotomous, we conduct logistic regression analyses to estimate the effects of race and gender on inflammation. To examine potential mechanisms that may underlie the observed association between being a black female and CRP,

we conduct decomposition analyses using the ldecomp command in Stata/SE 12.1. Using our full model which includes all control variables, we decompose the total effect into a direct effect (being black and female) and an indirect effect (obesity, socioeconomic disadvantage, educational attainment, smoking status, alcohol consumption, or physical activity). We run separate decomposition analyses for each focal mechanism. Finally, to understand which focal mechanisms most significantly affect CRP levels, we conduct a logistic regression analysis including obesity, socioeconomic disadvantage, educational attainment, smoking status, alcohol consumption, and physical activity.

Preliminary Results

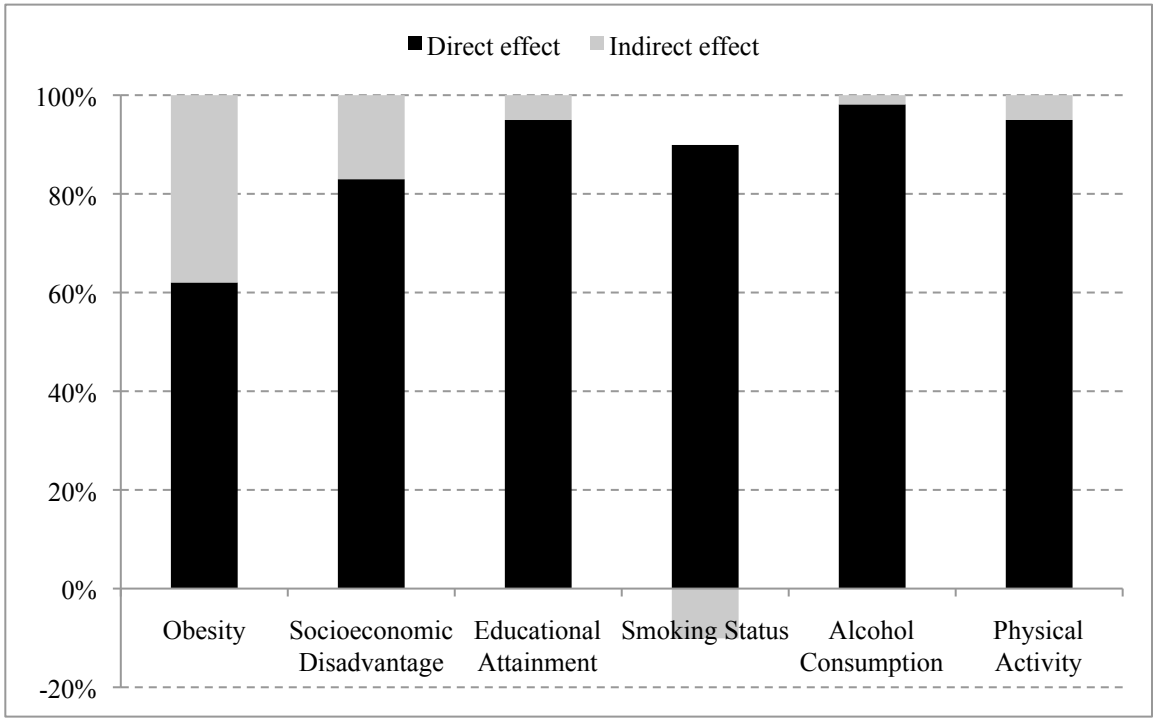
Model 1 in Table 2 estimates the relationship between race and gender and CRP controlling for age, systolic and diastolic blood pressure, cholesterol, triglycerides, and glycated hemoglobin. We find that being a black woman increases the odds of having average or elevated CRP by 37.7 percent. In Models 2 through 7, we separately introduce six plausible mechanisms to explain the increased inflammation experienced among black women. Figure 1 uses these estimates to decompose the main effect into the direct effect of being black and female and the indirect effect of the mechanism under analysis. Interpreting Tables 2 and Figure 1 together indicates that being obese accounts for 38 percent of the increased CRP risk associated with black women. Likewise, socioeconomic disadvantage, educational attainment, alcohol consumption, and physical activity also significantly mediate the effect of being black and female on CRP levels, though to a lesser extent than obesity (See Figure 1). Smoking status, however, suppresses the relationship of being a black woman and increased risk for average or elevated CRP. We find that smoking status reduces the effect of being a black female on CRP by 12.6 percent. This finding is largely attributable to lower smoking rates among black females (See Table 1). Model 8 controls for all six mechanisms and our focal association between black females and CRP disappears. This model indicates that the causes of average or elevated CRP levels among black women are primarily due to obesity, smoking status, physical activity, and socioeconomic disadvantage.

Table 2: Logistic Regression Odds Ratios for the Interaction of Race*Gender on CRP, n=11539

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
Female	1.540***	1.438***	1.501***	1.541***	1.685***	1.533***	1.523***	1.525***
	(0.138)	(0.130)	(0.135)	(0.139)	(0.155)	(0.138)	(0.137)	(0.143)
Black	1.152	1.149	1.055	1.096	1.151	1.157	1.162	1.079
	(0.127)	(0.128)	(0.119)	(0.122)	(0.128)	(0.128)	(0.128)	(0.124)
Female*Black	1.377*	1.237	1.387*	1.391*	1.395*	1.362*	1.321*	1.216
	(0.186)	(0.168)	(0.187)	(0.188)	(0.189)	(0.184)	(0.179)	(0.167)
Mechanisms:								
Obesity		2.353***						2.377***
		(0.162)						(0.166)
Socioeconomic Disadvantage			1.280***					1.173*
			(0.088)					(0.087)
Educational Attainment								
High School				0.859*				0.929
				(0.066)				(0.075)
More than High School				0.745***				0.904
				(0.063)				(0.083)
Smoking Status								
Current Smoker					1.596***			1.670***
					(0.128)			(0.138)
Former Smoker					1.385***			1.356***
					(0.113)			(0.114)
Alcohol Consumption								
Light Drinker						0.840**		0.900
						(0.056)		(0.062)
Heavy Drinker						0.827		0.791
						(0.122)		(0.119)
Physical Activity							0.683***	0.743***
							(0.046)	(0.051)
Control Variables:								
Age	1.006**	1.009***	1.005*	1.005*	1.007**	1.006**	1.004	1.007**
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
BP Systolic	1.009***	1.008***	1.008***	1.008***	1.009***	1.008***	1.008***	1.007***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
BP Diastolic	0.999	0.995	1.000	1.000	0.999	0.999	0.999	0.996
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
Cholesterol	0.997***	0.997***	0.997***	0.997***	0.997***	0.997***	0.997***	0.997***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Triglycerides	1.001***	1.001***	1.001***	1.001***	1.001***	1.001***	1.001***	1.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Glycated Hemoglobin	1.302***	1.260***	1.298***	1.296***	1.299***	1.297***	1.296***	1.242***
	(0.030)	(0.030)	(0.030)	(0.030)	(0.030)	(0.030)	(0.030)	(0.030)
Constant	0.006***	0.009***	0.006***	0.008***	0.007***	0.007***	0.009***	0.018***
	(0.002)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.006)

Standard errors in parentheses, *p<0.05, **p<0.01, ***p<0.001

Figure 1: Proportion of the Effect of Black*Female Explained by Each Mechanism



Future Research

The full paper will include a more extensive review of the current literature on race, gender, and inflammation. Subsequent analysis will extend the preliminary models to test a second marker of inflammation, albumin. Albumin is an acute-phase protein, like CRP, whose plasma concentrations decrease in the presence of inflammation. We expect to find lower albumin levels among black women and examine how mechanisms such as obesity, socioeconomic disadvantage, educational attainment, smoking status, alcohol consumption, and physical activity mediate or suppress this association. We will also a use cross-classified random effects model to distinguish age and cohort effects on the focal relationships and mediating pathways. Additionally, we plan to test the robustness of our findings by repeating our analyses eliminating those respondents who pass away in the five years following the survey.

References

Balkwill, Fran, and Alberto Mantovani. 2001. "Inflammation and cancer: back to Virchow?" *The Lancet* 357(9255):539–545.

Ben-Shlomo, Yoav, and Diana Kuh. 2002. "A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives." *International Journal of Epidemiology* 31(2):285–293.

Choo, Hae Yeon, and Myra Marx Ferree. 2010. "Practicing Intersectionality in Sociological Research: A Critical Analysis of Inclusions, Interactions, and Institutions in the Study of Inequalities*." *Sociological Theory* 28(2):129–149.

Chyu, Laura, and Dawn M. Upchurch. 2011. "Racial and Ethnic Patterns of Allostatic Load Among Adult Women in the United States: Findings from the National Health and Nutrition Examination Survey 1999–2004." *Journal of Women's Health* 20(4):575–583.

Deverts, Denise Janicki, Sheldon Cohen, Preety Kalra, and Karen A. Matthews. 2012. "The prospective association of socioeconomic status with C-reactive protein levels in the CARDIA study." *Brain, Behavior, and Immunity* 26(7):1128–1135.
Erlinger, T. P. 2004. "C-Reactive Protein and the Risk of Incident Colorectal Cancer." *JAMA: The Journal of the American Medical Association* 291(5):585–590.
Flegal, KM, MD Carroll, CL Ogden, and LR Curtin. 2010. "Prevalence and trends in obesity among us adults, 1999-2008." *JAMA* 303(3):235–241.
Gonçalves, R. B. et al. 2011. "Impact of smoking on inflammation: overview of molecular mechanisms." *Inflammation Research* 60(5):409–424.
Greenberg, Andrew S., and Martin S. Obin. 2006. "Obesity and the role of adipose tissue in inflammation and metabolism." *The American Journal of Clinical Nutrition* 83(2):461S–465S.
Harris, Tamara B et al. 1999. "Associations of elevated Interleukin-6 and C-Reactive protein levels with mortality in the elderly." *The American Journal of Medicine* 106(5):506–512.
Imhof, Armin et al. 2001. "Effect of alcohol consumption on systemic markers of inflammation." *The Lancet* 357(9258):763–767.
Krieger, Nancy. 2005. *Embodying inequality : epidemiologic perspectives*. Amityville, N.Y.: Baywood Pub.
Kuller, Lewis H., Russell P. Tracy, Jessica Shaten, and Elaine N. Meilahn. 1996. "Relation of C-Reactive Protein and Coronary Heart Disease in the MRFIT Nested Case-Control Study." *American Journal of Epidemiology* 144(6):537–547.
Nazmi, Aydin, and Cesar G Victora. 2007. "Socioeconomic and racial/ethnic differentials of C-reactive protein levels: a systematic review of population-based studies." *BMC Public Health* 7(1):212.
Pai, Jennifer K. et al. 2004. "Inflammatory Markers and the Risk of Coronary Heart Disease in Men and Women." *New England Journal of Medicine* 351(25):2599–2610.
Pampel, Fred C., Patrick M. Krueger, and Justin T. Denney. 2010. "Socioeconomic Disparities in Health Behaviors." *Annual Review of Sociology* 36(1):349–370.
Petersen, Anne Marie W., and Bente Klarlund Pedersen. 2005. "The anti-inflammatory effect of exercise." *Journal of Applied Physiology* 98(4):1154–1162.
Ridker, P. M. 2003. "C-Reactive Protein: A Simple Test to Help Predict Risk of Heart Attack and Stroke." *Circulation* 108(12):81e–85.
Robert, Stephanie A. 1999. "Socioeconomic Position and Health: The Independent Contribution of Community Socioeconomic Context." *Annual Review of Sociology* 25:489–516.
Rost, Natalia S. et al. 2001. "Plasma Concentration of C-Reactive Protein and Risk of Ischemic Stroke and Transient Ischemic Attack The Framingham Study." *Stroke* 32(11):2575–2579.
Visser, M, and LM Bouter. 1999. "Elevated c-reactive protein levels in overweight and obese adults." *JAMA* 282(22):2131–2135.