

CHRONIC INFLAMMATION AT THE INTERSECTION OF RACE AND ETHNICITY, GENDER, AND SOCIOECONOMIC STATUS: THE MEDIATING EFFECTS OF PROXIMATE RISK FACTORS AS COMPOSITE LIFESTYLES

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Abstract

This paper examines proximate risk factors as composite lifestyles in order to understand how common health behavioral patterns mediate the influence of intersecting social disadvantages on C-reactive protein (CRP) concentrations. Latent class analyses enable the identification of composite lifestyles based on obesity, abdominal obesity, insufficient physical activity, alcohol use, and smoking. Lifestyles characterized by obesity, abdominal obesity, insufficient physical activity, and abstaining from alcohol account for poor white, poor black, non-poor black, and non-poor Hispanic women's elevated inflammation levels compared to non-poor white women. In the case of all male demographic subgroups as well as poor Hispanic women, inflammation disparities persist net of accounting for potentially unhealthy combinations of proximate risk factors. The extent to which latent classes explain elevated inflammation among intersecting demographic traits enables clinicians to understand how proximate risk factors cluster together to affect inflammation and identify which proximate risk factor may be most effective in creating parity in CRP levels among demographic subgroups.

Introduction

The leading causes of death in the United States are heart disease, cancer, chronic lower respiratory diseases, and stroke. Collectively, these four conditions account for more than half of all deaths each year (Murphy et al. 2012). Chronic diseases, as these common causes of death are known, are illnesses characterized by their long duration, slow onset, and inability to resolve themselves without intervention (World Health Organization 2012). Estimates show that nearly

half of all U.S. adults (141 million people) have at least one chronic condition. With this number expected to rise to 157 million by 2020 (Wu and Green 2000), the need to understand the risk factors associated with chronic conditions and implement ways to reduce the burden of chronic disease on society is paramount.

Chronic inflammation and dysregulation of the immune function are related to many chronic diseases of aging (e.g., heart disease, cancer, Alzheimer's) that are now major causes of death (Allin, Bojesen, and Nordestgaard 2009; Erlinger 2004; Harris et al. 1999; Pai et al. 2004; Ridker 2014; Rost et al. 2001). A crucial biological pathway by which immune function may become permanently altered is through chronic psychosocial stress (Segerstrom and Miller 2004), which leads to higher levels of chronic inflammation. Additionally, exposure to psychosocial stress is associated with adopting negative health behaviors, including smoking, drinking, poor diet, and physical inactivity, which are proximate risk factors for inflammation. Current research has begun examining this relationship as a potential explanation for the observed variation in health across socially disadvantaged groups. According to this research belonging to a disadvantaged group in terms of race, sex, or class leads to prolonged exposure to stress that ultimately leads to permanent changes in biological functioning resulting in worse health. Indeed, past research has demonstrated that men, non-Hispanic whites, and those with higher socioeconomic status exhibit less inflammation than their peers.

While past research has demonstrated that socially disadvantaged groups have higher levels of inflammation, research to date has not examined the intersection of how membership to these different groups interact and compound to influence measures of inflammation or the role that proximate risk factors play in these differences. Recent feminist theory, and particularly the concept of intersectionality (Collective 1986; Collins 1991; hooks 2000), suggests examining

multiple aspects of identity simultaneously to determine how privilege and disadvantage surrounding individuals' sex, race, and class interlock to produce unique experiences of psychological stress. Thus, the experience of being male, poor, or black is very different than the experience of being a poor black male. In the quantitative setting, simply controlling for anyone of these social categories may lead to erroneous conclusions concerning their impact on health, given that the experiences within these social categories is largely shaped by one's membership to other categories.

Lastly, rather than examine proximate risk factors separately, we examine latent classes of risk factors to identify different patterns of health behavior. Past research concerning health behaviors suggests that positive health behaviors tend to cluster together, such that if an individual acts healthy in one realm, such as not smoking they are also more likely to act healthy in another realm, such as refraining from heavy drinking (Cockerham 2005). However, the likelihood of having one unhealthy habit and not another likely depends on the health habits in question as well as structural and cultural factors concerning the availability and acceptability or unacceptability of those health habits. These structural and cultural factors are likely largely shaped by sex, race/ethnicity, and class. Additionally, understanding how different health habits cluster together may then direct clinicians how best to advise patients to reduce CRP levels when considering proximate risk factors.

Literature Review

Chronic exposure to stressors 'gets under the individual's skin' through altered physiological functioning including activation of the hypothalamic-pituitary-adrenocortical axis (HPA) and the sympathetic-adrenal-medullary (SAM) leading to higher levels of inflammation (Padgett and Glaser 2003) which increases the risk of mortality and morbidity (Danesh et al.

2004; Harris et al. 1999; Jenny et al. 2007; Kaptoge et al. 2010; Pai et al. 2004; Prasad 2003; Ridker 2014; Ridker et al. 2000). C-reactive protein (CRP) is released innately by the body to cope with stressors including those effecting physiological functioning such as infection, but it is also released in response to factors effecting psychosocial functioning, such as stress. CRP, as an indicator for chronic, low-grade inflammation, has been associated with elevated mortality (Harris et al. 1999; Jenny et al. 2007; Kaptoge et al. 2010) as well as increased risk of several chronic diseases, including coronary heart disease (Danesh et al. 2004; Kaptoge et al. 2010; Pai et al. 2004; Prasad 2003; Ridker 2014; Ridker et al. 2000), stroke (Kaptoge et al. 2010; Prasad 2003; Ridker 2014), cancer (Allin et al. 2009; Erlinger 2004; Siemes et al. 2006), type 2 diabetes (Pradhan 2001; Thorand et al. 2003), and hypertension (Bautista et al. 2001; Sesso 2003).

Exposure to chronic stress and the internalization of these stressors is likely influenced by one's membership to socially constructed groups on dimensions such as sex, race, and class. For example, disadvantaged groups face structural barriers of making 'ends meet,' which leads to higher exposure of stress. Stressors such as the ability to pay bills on time, having sufficient and nutritious food to feed one's family, and living in a neighborhood where one does not feel like they readily face physical harm all influence exposure to stress. In addition to these factors, largely related to the economic demands of structuring an environment conducive to healthy living, exposure to stress in the form of discrimination may impact health as well. Indeed discrimination, in the form of segregation has relegated individuals of color to environments that are less conducive to healthy behaviors, such as poor urban neighborhoods. Additionally, experiences of direct or interpersonal discrimination, likely the result of membership to a disadvantaged group, result in increased stress and may increase CRP.

This idea is reflected in studies that find higher levels of CRP concentrations are most prevalent among non-Hispanic blacks, followed by Hispanics, with non-Hispanic whites having the lowest concentration of CRP (Nazmi and Victora 2007). Additionally, higher CRP is found among those with lower socioeconomic status, such that those with low education or low income have higher CRP than their better off counterparts. Past research indicates that as much of 16-18% of the race/ethnic gap in CRP can be accounted for by differences in socioeconomic status (Herd, Karraker, and Friedman 2012). However, merely controlling for differences in socioeconomic status, ignores the insight of intersectionality theory, which states the experience of class (race, or sex) is informed by your membership to other categories such as race and sex. Rather than assuming that these factors have uniform effects across groups this theory suggests that experiences are unique based on a multitude of other factors including other group membership.

In addition to experiences of stress being directly related to inflammation response, experiences of stress result in adopting negative health behaviors likely in a response to cope with elevated levels of stress. While past research has demonstrated that positive health behaviors, defined by never smoking, healthy diet, sufficient physical activity, and moderate alcohol consumption, tend to cluster together, such that being healthy in one domain increases the likelihood of being healthy in another domain, few individuals exhibit healthy lifestyles across all the domains. Data from the Behavioral Risk Factor Surveillance System (BRFSS) indicates that only 3% of adults between the ages of 18-74 years consistently practice a low-risk lifestyle (Reeves and Rafferty 2005). Adherence to low-risk lifestyles slightly increase with age, educational attainment, and household income, and is highest among women and non-Hispanic whites (Reeves and Rafferty 2005). Given that individuals often concurrently engage in both

healthy and unhealthy behaviors, it is critical to evaluate how proximate risk factors cluster together to form common lifestyle patterns, how these lifestyle patterns are related to inflammation, and how different lifestyles account for differences in inflammation across race/ethnicity, sex, and class.

Thus this project seeks to address the following research questions.

Research Question 1: What is the relationship between race and ethnicity, sex, and socioeconomic status demographic subgroups and latent class membership?

Research Question 2: To what extent does latent class membership mediate the joint effects of sex, race and ethnicity, and socioeconomic status on inflammation?

Data

This study is based on data from the National Health and Nutrition Examination Survey (NHANES). Conducted by the National Center for Health Statistics (NCHS), NHANES is a nationally representative health and nutrition study, sampling approximately 10,000 children and adults biannually, excluding those in institutional settings and all active-duty military personnel. The survey began in the 1960s and, in 1999, became an ongoing study. Biennial surveys conducted between 1999 and 2014 are referred to as “continuous NHANES.” NHANES data collection combines in-person interviews with physician performed physical examinations and laboratory testing (Johnson, Paulose-Ram, and Ogden 2013; Zipf, Chiappa, and Porter 2013).

NHANES employs a complex, multistage probability sampling design to capture a representative household sample population of the fifty states and the District of Columbia. For each two-year continuous NHANES study, NCHS researchers select approximately thirty primary sampling units (i.e. counties) across the United States, representing each quadrant of the country as well as metropolitan and nonmetropolitan areas. Within each primary sampling unit,

blocks or clusters of households are selected and then specific households are chosen to be screened (Zipf et al. 2013). On average, 1.6 persons are selected from each household. Response rates vary slightly in regard to survey year; out of the 12,000 individuals asked to participate in each continuous study, approximately 10,500 complete the household interview and 10,000 participate in the physical examinations (Zipf et al. 2013).

Additionally, we do not exclude respondents with missing data. For most study variables, the percent of respondents with missing data is minimal. More than 20 percent of the sample, however, is missing data on alcohol use (See Appendix A for the percentage of missing data for all study variables). To address missing values for all study variables, I use multivariate imputation by chained equations (MICE) in Stata 13.1 (Royston 2004). MICE generates a specified number of copies of the dataset with imputed missing values. The resulting analyses take parameter estimates for each dataset and average them across all datasets to create a single estimate. Standard error estimates take into account between- and within-imputation variation across the imputed datasets as suggested by Rubin Rules (Royston 2004; Rubin 1987). All analyses in this study are based on pooling values from ten imputed data sets.

Due to the complex survey design of NHANES, there is clustering of observations within households and broader sampling units that may downwardly bias standard errors. To correct for this potential bias, all analyses are weighted to reflect the probability of selection, to take into account survey non-response, and to apply post-stratification adjusting for underrepresented groups in the population (Johnson et al. 2013). The resulting weighted sample is representative of the U.S. Census civilian, non-institutionalized population.

Dependent Variable

The focal dependent variable in this study is C-reactive protein (CRP), an acute phase protein which marks systemic inflammation within the body. NHANES participants have their CRP levels recorded as a component of their blood tests in the MEC. CRP levels range from 0.01 mg/dL to 20 mg/dL in the sample, with extreme CRP values (CRP > 10 mg/dL) excluded because these values are generally indicative of an acute infection (Pearson et al. 2003). Additionally, I also omit pregnant women from my study as pregnancy potentially influences inflammation levels. In all analyses, CRP is log transformed to account for the variable's skewed distribution and high kurtosis.

Focal Independent Variables

My primary independent variables reflect the intersection of three social statuses: race and ethnicity, gender, and socioeconomic status. Race and ethnicity is represented with three mutually exclusive dummy variables: non-Hispanic white (referred to as white from here on), non-Hispanic black (referred to as black from here on), and Hispanic. Sex is coded 1 for female and 0 for male.¹ Socioeconomic status is measured with a poverty income ratio provided in NHANES. 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 official poverty line (>1.00). I use a dummy variable for socioeconomic disadvantage coded 1 for individuals with a poverty income ratio less than 2, to account for those who may live above the official poverty threshold yet still face substantial economic hardship.

¹ In this paper we are only able to measure sex dichotomously, but we understand that people transcend the categories of male/female. However, our sample did not allow individuals who self-identified as transgender, hermaphrodite, intersexed, or other sex classifications to indicate such.

To examine inequalities at the intersection of race and ethnicity, gender, and socioeconomic status, I employ a categorical approach pioneered by McCall (2005) which “focuses on the complexity of relationships among multiple social groups within and across analytical categories” (pg. 1786). This approach enables the exploration of variation in inflammation at the intersection of socioeconomic status, race and ethnicity, and gender, with sensitivity to disparate pathways that may emerge on account of sociodemographic life experiences.

Mediators

The analysis also includes proximate risk factors that are expected to mediate the effects of social statuses on CRP. The measures of proximate risk factors include body composition, physical activity, alcohol use, and smoking. Body composition is assessed with two indicators: body mass index (BMI) and waist circumference (WC). *Body mass index* (kg/m^2) is calculated using weight in kilograms divided by height in meters squared. A BMI of $30 \text{ kg}/\text{m}^2$ or above indicates *obesity* (National Heart, Lung, and Blood Institute 2014). *Waist circumference* measures the quantity of fat surrounding the waist. *Abdominal obesity* in this study is represented with a binary variable coded 1 for WC values greater than 35 inches for women and greater than 40 inches for men (National Heart, Lung, and Blood Institute 2014).

The *physical activity* section of the questionnaire addresses questions related to energy expenditure throughout a typical week. Respondents are asked to report how often they spend per week participating in vigorous work activity, moderate work activity, walking or bicycling for transportation, vigorous recreational activities, and moderate recreational activities. To summarize energy expenditure, intensity of physical activity is often defined as the metabolic equivalent of task (MET) (Bull et al. 2004). One MET reflects the energy expended while being

sedentary for one hour. NHANES provides MET scores for each measured activity. The measure of physical activity used in my analyses reflects cumulative METs expended per week. Further, the CDC recommends that healthy adults participate in 150 minutes of moderate-intensity activity weekly (U.S. Centers for Disease Control and Prevention 2011). Therefore, we use a binary measure of *insufficient physical activity* coded 1 for participants who fail to meet the CDC physical activity recommendation of 150 minutes per week (i.e. the equivalent of 175.5 METs per week [$165.5 \text{ hours} * (1 \text{ MET}) + 2.5 \text{ hours} * (4 \text{ METs})$]).

The *alcohol use* is measured by asking respondents “In the past 12 months, on those days that you drank alcoholic beverages, on the average, how many drinks did you have?” Based on the self-reported number of drinks, participants are categorized as either moderate drinkers or heavy drinkers. According to the federal dietary guidelines (U.S. Department of Agriculture and U.S. Department of Health and Human Services 2010), for women, moderate drinking is defined as one alcoholic beverage per day, with two or more alcoholic drinks per day denoting heavy drinking. For men, moderate drinking is defined as 1-2 alcoholic drinks per day, with anything in excess of two drinks signifying heavy drinking. Thus, alcohol consumption in this study is assessed with three mutually exclusive dummy variables: non-drinkers, moderate drinkers, and heavy drinkers.

We assess *smoking behavior* by grouping respondents into three categories: non-smokers, former smokers, and current smokers. Respondents who have smoked less than 100 cigarettes in their lifetime are considered non-smokers. Those participants who have smoked more than 100 cigarettes in their lives, yet no longer do so comprise former smokers. We define current smokers as those who smoke on average one or more cigarettes per day.

Social and Demographic Control Variables

Age at the time of the interview is measured in years. Indicators of *educational attainment* reflect the highest grade of school respondents completed, with responses coded as less than a high school education, high school education, and more than a high school education. A binary measure of *foreign born* is coded 1 if participants were born in a country other than the United States. Foreign-born individuals were also asked their duration of residence in the United States. *Years in the United States* is included as a part of an interaction term with *foreign born* to act as an internal moderator (Mirowsky 2013) of nativity (i.e. $\text{foreign born} \times \text{years in United States}$). Respondents' *marital status* is represented by three mutually exclusive categories: never married; currently married or living with a partner (referred to from here on as married); and divorced, separated, or widowed.

Biological Control Variables

Respondents' *total cholesterol* (mg/dL) reflect their lipid profile. *Glycohemoglobin* levels (%) indicate diabetes risk by estimating plasma glucose levels within the body over the past four months. To correct for high kurtosis, glycohemoglobin is log transformed in all analyses. Respondents undergoing MEC evaluation also had their systolic and diastolic blood pressure measured three times. I averaged respondents' three observations for diastolic and systolic blood pressure separately to create separate indicators of diastolic and systolic blood pressure. Higher values of each biological control variable reflect a riskier cardiometabolic profile.

Analyses

We employ both LCA and multivariate regression to address our research questions. Latent class analyses (LCA) is used to identify latent population subgroups ("classes") based on a number of characteristics shared by individuals within classes. We use LCA to identify

subgroups of individuals with similar characteristics in regard to the following proximate risk factors: obesity, abdominal obesity, insufficient physical activity, alcohol use, and smoking. These latent classes are then explored as potential mediators of the focal relationship between race and ethnicity \times sex \times socioeconomic status and inflammation.

LCA is based on the assumption that an underlying unobserved grouping construct can be inferred from a set of observed categorical variables (Lanza, Savage, and Birch 2010). Latent class models estimate class membership probabilities and item-response probabilities. Class membership probabilities tend to be the most informative because they reflect the proportion of the population expected to fall into each latent class (Lanza et al. 2010). Two or more observed categorical variables are used as indicators of a categorical latent variable (Lanza and Bray 2010).

I model a categorical latent variable separately for men and women based on obesity, abdominal obesity, insufficient physical activity, alcohol use, and smoking – to categorize participants into homogeneous classes with shared profiles of proximate health risk factors. Mathematically, LCA can be expressed with the formula following Lanza and Bray (2010):

$$P(Y = y) = \sum_{c=1}^C \gamma_c \prod_{j=1}^J \prod_{r_j=1}^{R_j} \rho_{j,r_j|c}^{I(y_j=r_j)}$$

where $I(y_j = r_j)$ is an indicator function set to 1 when the response to variable $j = r_j$, γ_c is the probability of membership in latent class c , $\rho_{j,r_j|c}^{I(y_j=r_j)}$ is the probability of response r_j to item j , conditional on membership in latent class c , the γ parameters are a vector of latent class membership probabilities, and the ρ parameters are a matrix of item-response probabilities conditional on latent class membership (Lanza and Bray 2010). In other words, a person's probability to have a certain combination of health risks can be calculated as a sum of products.

Each product is a probability of the person to be in latent class c multiplied by probabilities of having a particular set of health risks conditional on being in class c . We use Mplus7 to conduct LCA.

After the latent classes are constructed we use logistic regression for females and multinomial logistic regression for males to predict latent class membership on account of demographic subgroup to address our first research question. For each latent class comparison, the first model controls for age and the second model introduces the following additional sociodemographic variables: educational attainment, nativity, and marital status. The OLS regression analyses examining the mediating effects of latent class on the focal relationship consist of three models. Model 1 examines the effects of race and ethnicity \times gender \times socioeconomic status on inflammation controlling for age. Model 2 introduces sociodemographic and biological control variables. Finally, the mediating effects of latent class are examined in Model 3.

Results

Table 1 displays summary statistics for proximate risk factors by race and ethnicity, gender, and socioeconomic disadvantage for the weighted 2007-2010 NHANES sample. Asterisks represent significant differences in proximate risk factors between each demographic subgroup and the reference group (non-poor white).

Among women, blacks and poor Hispanics have the highest prevalence of obesity and abdominal obesity. Among men, variation in obesity prevalence across demographic subgroups is minimal with the exception of non-poor black men who had the highest prevalence. However, examining abdominal obesity we find that poor black and poor Hispanic men are significantly less likely to be obese than their non-poor white counterparts ($p < .001$ and $p < .01$, respectively).

Examining insufficient physical activity we find that among women, all demographic subgroups engage in less weekly physical activity than non-poor whites, with the majority of poor and non-poor black women failing to meet this standard. Among men, poor whites and blacks are significantly less likely to engage in 150 minutes of moderate physical activity per week. With respect to alcohol use among women, non-poor white women are the most likely to be moderate drinkers. Additionally, poor Hispanic women, black women, and poor white women are the most likely to say that never drink alcohol. Lastly, non-poor Hispanic women are more likely (52% vs. 42%, $p < .01$) and non-poor black women are less likely (35% vs. 42%, $p < .05$) to be heavy drinkers compared to non-poor white women. Among men, poor and non-poor black men ($p < .01$), poor white men ($p < .001$), and poor Hispanic men ($p < .05$) are significantly more likely to never drink alcohol than non-poor white men. Similarly, poor black and poor white men and all Hispanic men are less likely to be moderate drinkers than non-poor white men ($p < .001$). In addition, poor and non-poor Hispanic men ($p < .001$) as well as poor white men ($p < .01$) are significantly more likely to be heavy drinkers than their non-poor white counterparts. Examining the last proximate risk factor, smoking, we find that poor white and black women are significantly more likely to be current smokers than non-poor white women ($p < .001$). Among men, prevalence of current smoking is highest among poor black males ($p < .001$), followed by poor white males ($p < .001$) and poor Hispanic males ($p < .01$).

To construct latent classes of proximate risk factors, several models were estimated with different numbers of latent classes and the model fit indices were compared to select the number of latent classes that produces the best-fitting model. Based on the model fit, the optimal number of latent classes is two for women and three for men. Table 2 displays class membership probabilities for each latent class based on five proximate health risk factors: obesity, abdominal

obesity, insufficient physical activity, alcohol use, and smoking. For women, 57% of NHANES 2007-2010 participants comprise latent class one. Seventy one percent of women in this class are obese and 100% have waist circumference values greater than 35 inches, indicative of abdominal obesity. The majority of respondents in this class are physically inactive (57.5%) and a minority currently smoke (17.2%). Though most participants in this class refrain from alcohol consumption (39.4%), one-third are heavy drinkers, and 27.4 percent moderately drink alcohol. Interpreting these distinctions collectively illustrates a group that may best be described as ‘obese and inactive’.

The second female latent class captures 43% of survey participants. In contrast to the first class, class two exhibits lower prevalence of obesity, abdominal obesity, and physical inactivity (8%, 24.8%, and 39.1% respectively). In terms of alcohol use and smoking, latent class two engages in more risky behavior than class one; forty four percent of those in class two are heavy drinkers and 22% smoke regularly. Class two may be summarized as ‘fit and tipsy’.

Latent class one for the men encompasses 19% of the male sample. Those in this class may be described as ‘health nuts’. Prevalence of obesity and abdominal obesity are low (2.4% and 16.2%, respectively). There are no current smokers or heavy drinkers in this class and approximately two-thirds engage in sufficient weekly physical activity. Seventy percent drink moderately and 30.5% never drink alcohol.

The second class for males is similar to the first in terms of their fitness. Four percent of this group are obese, 6.9% have high central obesity, and 27.6 % do not engage in sufficient physical activity. In terms of health behaviors, however, 66.7% are heavy drinkers and 45.6% are current smokers. This combination of proximate risk factors may best be described as ‘lean and having a good time’.

The final latent class for males accounts for 42% of the sample. The majority of participants included are obese (78.8%) and abdominally obese (98.8%), with high levels of insufficient physical inactivity (39.8%). Further, men in this group consume alcohol in high quantity. Forty one percent drink heavily, 44.8% drink moderately, and only 14.7% refrain from alcohol altogether. Nineteen percent of this class smokes, slightly below the male average. Latent class three is comprised of ‘*big drinkers*’.

Research Question 1: What is the relationship between race and ethnicity, gender, and socioeconomic status demographic subgroups and latent class membership?

Table 3 presents the proportion of each demographic subgroup in each latent class for men and women. Among women, the obese and inactive class is primarily composed of non-poor black, non-poor Hispanic, poor white, poor black, and poor Hispanic women. The majority of non-poor white women exhibit attributes consistent with the fit and tipsy class. Despite the majority belonging to the obese and inactive class, forty six percent of non-poor Hispanic women and 43.7% of poor white women are characterized as fit and tipsy. Among males, most non-poor white, non-poor black, non-poor Hispanic, and poor white men fall within the *big drinkers* class. Approximately half of poor black and poor Hispanic men fit within the lean and having a good time class. Although there is no dominant presence of any male demographic subgroup in the health nut class, non-poor men comprise the highest proportion of those included.

Table 4 reports the odds of being in the obese and inactive class (vs. the fit and tipsy class) for each female demographic subgroup controlling for age (Model 1) and sociodemographic control variables (Model 2). Compared to non-poor white women, all female

demographic subgroups have increased odds of belonging to the obese and inactive class than to the fit and tipsy class. In particular, poor black women are three times as likely to exhibit health risk factors consistent with the obese and inactive class ($p < .001$), non-poor black women are 2.5 times as likely ($p < .001$), poor Hispanic women are 2.4 times as likely ($p < .001$), non-poor Hispanic women are 1.5 times as likely ($p < .001$), and poor white women are 1.4 times as likely ($p < .05$). These findings persist net of sociodemographic controls.

Table 5 presents relative risk ratios for men examining the odds of each demographic subgroup belonging to the lean and having a good time class (Models 1 and 2) or the big drinkers class (Models 3 and 4) compared to the health nut class. Poor white ($p < .05$), poor black ($p < .05$), and poor Hispanic men ($p < .001$) are significantly more likely than non-poor white men to belong to the lean and having a good time class than the health nut class. This finding largely reflects higher rates of heavy drinking and smoking among poor men of all race and ethnicities. Comparing the *big* drinkers class to the health nut class, both poor and non-poor Hispanic men have significantly higher odds of belonging to the *big* drinkers than non-poor white men, given their higher rates of heavy drinking and poor Hispanic men's higher rates of abdominal obesity ($p < .001$ and $p < .05$, respectively). These demographic subgroup latent class distinctions persist when accounting for the sample's sociodemographic characteristics.

Research Question 2: To what extent does latent class membership mediate the joint effects of race and ethnicity and socioeconomic status on inflammation?

Ordinary least squares regression analyses examining the mediating effect of latent class on the focal relationship between demographic subgroup and CRP are presented in Table 6 for women and Table 7 for men. Model 1 examines the main effect of demographic subgroup on

CRP controlling for age; Model 2 introduces social and biological control variables, while Model 3 explores the effects of latent class.

Among females, poor white ($p < .05$), poor black ($p < .01$), non-poor black ($p < .01$), and poor Hispanic women ($p < .001$) have significantly elevated CRP levels compared to non-poor white women. Controlling for sociodemographic and biological factors reduces, but does not diminish, the significant magnitude of these findings (Model 2). As Model 3 indicates, being in the obese and inactive class is associated with elevated inflammation levels ($p < .001$) compared to the fit and tipsy class. With the exception of poor Hispanic women ($p < .05$), belonging to the obese and inactive class explains the elevated inflammation levels associated with poor white, poor black, and non-poor black women reported in Model 2. Inflammation disparities between poor Hispanic and non-poor white women remain, as belonging to the obese and inactive latent class does not fully explain this variation.

Among men, all demographic subgroups experience significantly elevated inflammation levels compared to non-poor white men. These findings persist net of all sociodemographic and biological factors (Model 2). As shown in Model 3, belonging to the lean and having a good time class or the *big* drinkers class are associated with higher CRP levels compared to the health nut class ($p < .001$ and $p < .001$, respectively). Though the latent classes are significantly related to inflammation, this relationship does not strongly mediate the focal demographic subgroup and CRP relationship. Higher inflammation levels among poor white men ($p < .001$), poor black men ($p < .01$), non-poor black men ($p < .05$), poor Hispanic men ($p < .01$), and non-poor Hispanic men ($p < .05$) remain compared to non-poor white men. Further, we observe a suppression effect among poor and non-poor black males when accounting for latent class membership and this is especially true among poor black men. This suppression effect suggests that poor black men

would actually have *higher* levels of CRP if they had health behaviors that were similar to those found among non-poor whites. Overall, belonging to either the lean and having a good time class or *big* drinkers class does not account for variation in inflammation levels among men.

Conclusions

Among women of all ages in the NHANES 2007-2010 analytic sample, elevated inflammation is partially or fully explained by belonging to the obese and inactive class compared to the fit and tipsy class. Poor white, poor black, non-poor black, and non-poor Hispanic women's elevated inflammation levels compared to non-poor white women are a result of higher rates of obesity, abdominal obesity, insufficient physical activity, and never drinking alcohol. Although these proximate risk factors reduce the inflammation disparity between poor Hispanic and non-poor white women, elevated inflammation experienced by poor Hispanic women persists net of class membership.

Among men ages 20 and older, higher CRP levels among poor white, poor black, non-poor black, poor Hispanic, and non-poor Hispanic males are partially mediated or suppressed by belonging to the lean and having a good time class or the *big* drinkers class compared to the health nut class. Membership to the lean and having a good time class or the *big* drinkers class partially explains elevated inflammation among poor white, poor Hispanic, and non-poor Hispanic men. Poor and non-poor black men, however, experience a slight suppression effect. In the case of all male demographic subgroups, inflammation disparities compared to non-poor white males remains despite accounting for potentially unhealthy combinations of proximate risk factors.

Membership to socially disadvantaged groups is associated with higher levels of mortality and morbidity. One potential mechanism that may lead to these higher levels of

mortality and morbidity is chronic exposure to stress leading to adopting unhealthy behaviors and higher levels of inflammation. Intersectionality theory stresses that membership (or non-membership) to one of these socially disadvantaged groups is informed by one's membership to other groups. Indeed, we find distinct patterns of CRP in relation to sex, race/ethnicity, and socioeconomic status. These differences would not be apparent if controls for gender, race/ethnicity, or socioeconomic status were only employed, rather than looking at the intersection of these factors. Additionally, the latent class analysis demonstrates that proximate risk factors to health cluster together to form different health lifestyles and these different health lifestyles vary by sex, race, and socioeconomic status. Among women, blacks, Hispanics, and poor white women were all more likely to belong to the obese and inactive group. In fact only non-poor white women had a majority of their group in the fit and tipsy category. Men, on the other hand, were most likely to belong to the big drinkers group across sociodemographic characteristics, with the exception of poor black and poor Hispanic men who were more likely to belong to the fit and having a good time group. This study highlights the importance of examining how membership to different socially disadvantaged groups impacts measures of inflammation and also demonstrates that health behaviors can be examined as clusters or groups.

Table 1. Descriptive Statistics for the Study Variables by Race and Ethnicity, Gender, and Socioeconomic Status, NHANES 2007-2010 (Weighted)

Variable Name	Full Sample	Non-Poor			Poor		
		White	Black	Hispanic	White	Black	Hispanic
C-Reactive Protein	.390						
Male	.330	.295	.347**	.341	.370***	.445**	.324*
Female	.440***	.411	.495***	.423	.440**	.666***	.508***
Obese	.358						
Male	.346	.350	.446***	.393	.330	.313	.326
Female	.369	.315	.521***	.371	.364	.564***	.447***
Abdominal Obesity	.540						
Male	.463	.494	.442	.435	.461	.338***	.369**
Female	.626***	.573	.705**	.586	.637	.773***	.715***
Insufficient Physical Activity	.350						
Male	.268	.232	.301*	.267	.329**	.350***	.271
Female	.431***	.365	.530***	.468**	.486***	.542***	.485**
Alcohol Use							
Non-Drinker	.188						
Male	.119	.094	.152**	.107	.191***	.166**	.113
Female	.254***	.172	.328***	.225	.338***	.341***	.410***
Moderate Drinker	.386						
Male	.445	.528	.497	.333***	.329***	.351***	.219***
Female	.330***	.409	.325**	.257***	.229***	.216***	.197***
Heavy Drinker	.426						
Male	.436	.377	.351	.560***	.481***	.483**	.668***
Female	.416	.419	.346*	.519***	.433	.443	.393
Current Smoker	.224						
Male	.251	.187	.205	.188	.388***	.466***	.283**
Female	.199***	.148	.138	.119	.343***	.313***	.148
Male <i>n</i>	5101	1650	553	561	1008	482	847
Female <i>n</i>	5237	1499	491	524	1158	581	984

Note: Analyses conducted separately by gender, with non-poor white males and females serving as reference categories. Asterisks denote significant differences between each subgroup and the reference category: * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2. Probabilities of Latent Class Membership by Proximate Risk Factors

Variable Name	Females		Males		
	Latent Class 1	Latent Class 2	Latent Class 1	Latent Class 2	Latent Class 3
	Obese and Inactive	Fit and Tippy	Health Nut	Lean and Having a Good Time	<i>Big</i> Drinkers
	57%	43%	19%	39%	42%
Body Composition					
Obese	.714	.008	.024	.043	.788
Abdominal Obesity	1.000	.248	.162	.069	.988
Insufficient Physical Activity	.575	.391	.322	.276	.398
Alcohol Use					
Non-Drinker	.394	.249	.305	.060	.147
Moderate Drinker	.274	.308	.695	.273	.448
Heavy Drinker	.331	.443	.000	.667	.405
Current Smoker	.172	.223	.000	.456	.190
Log Likelihood (df)	-17665 (13)		-16416 (20)		
AIC	35359		32872		
BIC	35404		32941		

Table 3. Latent Class Membership by Demographic Subgroup

Latent Class	Female			Male			
	1	2		1	2	3	
Variable Name	Obese and Inactive	Fit and Tippy	<i>n</i>	Health Nut	Lean and Having a Good Time	<i>Big</i> Drinkers	<i>n</i>
Non-Poor							
White	.481	.519	1499	.240	.274	.485	1650
Black	.669	.331	491	.223	.331	.446	553
Hispanic	.536	.464	524	.196	.372	.432	561
Poor							
White	.563	.437	1158	.149	.405	.446	1008
Black	.712	.288	581	.162	.495	.343	482
Hispanic	.648	.352	984	.124	.511	.365	847
Total			5237				5101

Table 4. Logistic Regression Models Predicting Obese and Inactive Latent Class Membership for Females (Odds Ratios), NHANES 2007-2010

Latent Class	Class 2	
	Fit and Tipsy	
Variable Name	Model 1	Model 2
Poor White Female	1.411** (.175)	1.345* (.150)
Poor Black Female	3.102*** (0.424)	2.986*** (.398)
Non-Poor Black Female	2.407*** (.391)	2.501*** (.392)
Poor Hispanic Female	2.535*** (.340)	2.385*** (.372)
Non-Poor Hispanic Female	1.455*** (.148)	1.516*** (.156)
Age	1.030*** (.002)	1.029*** (.003)
Less than High School		1.442*** (.136)
High School		1.285** (.110)
Foreign Born		.855 (.101)
Foreign Born * Years in U.S.		.994 (.006)
Widowed, Divorced, or Separated		.847^ (.077)
Never Married		.882 (.104)
Constant	.226*** (.032)	.222*** (.031)
N	5819	5819

p-value, ^ 0.10 * 0.05 ** 0.01 *** 0.001

Table 5. Multinomial Logistic Regression Models Predicting Health Nut Latent Class Membership for Males (Relative Risk Ratios), NHANES 2007-2010

Latent Class	Class 2		Class 3	
	Lean and Having a Good Time (vs. Health Nut)		<i>Big Drinkers</i> (vs. Health Nut)	
Variable Name	Model 1	Model 2	Model 3	Model 4
Poor White Male	2.017*** (.301)	1.516* (.254)	1.453** (.191)	1.295^ (.185)
Poor Black Male	2.200*** (.491)	1.519* (.308)	1.066 (.192)	.970 (.155)
Non-Poor Black Male	1.072 (.207)	.960 (.186)	1.018 (.163)	1.004 (.151)
Poor Hispanic Male	2.571*** (.424)	2.161*** (.409)	1.595* (.310)	2.503*** (.563)
Non-Poor Hispanic Male	1.285 (.197)	1.284^ (.186)	1.161 (.223)	1.592* (.310)
Age	.970*** .003	.966*** (.004)	1.013*** (.003)	1.008* (.003)
Less than High School		2.639*** (.254)		1.520*** (.182)
High School		2.112*** (.379)		1.724** (.281)
Foreign Born		.661** (.086)		.388*** (.052)
Foreign Born * Years in U.S.		1.002 (.009)		1.013^ (.007)
Widowed, Divorced, or Separated		1.962*** (.330)		1.258 (.193)
Never Married		1.144 (.191)		.750^ (.124)
Constant	5.100*** (1.110)	4.246*** (1.156)	1.085 (.235)	1.290 (.338)
N	5623	5623	5623	5623

p-value, ^ 0.10 * 0.05 ** 0.01 *** 0.001

Table 6. Ordinary Least Squares Regression Models Predicting C-Reactive Protein (logged) for Females, NHANES 2007-2010

Variable Name	Model 1	Model 2	Model 3
Poor White Female	.254** (.082)	.173* (.076)	.112 (.068)
Poor Black Female	.543*** (.085)	.303** (.084)	.126 (.084)
Non-Poor Black Female	.404*** (.081)	.267** (.087)	.110 (.072)
Poor Hispanic Female	.464*** (.089)	.366*** (.090)	.201* (.075)
Non-Poor Hispanic Female	.184^ (.104)	.181^ (.104)	.099 (.096)
Class 1: Obese and Inactive			1.039*** (.041)
Class 2: Fit and Tipsy (reference)			
Age	.008*** (.001)	-.005* (.002)	-.008*** (.002)
Less than High School		.132* (.064)	.072 (.057)
High School		.110* (.047)	.057 (.044)
Foreign Born		-.158* (.066)	-.125* (.061)
Foreign Born * Years in U.S.		-.001 (.003)	.001 (.003)
Widowed Divorced or Separated		.028 (.032)	.065^ (.034)
Never Married		-.016 (.060)	.015 (.054)
Cholesterol		.002* (.001)	.002* (.001)
Diastolic Blood Pressure		.006* (.002)	.002 (.002)
Systolic Blood Pressure		.006** (.002)	.004* (.002)
Glycohemoglobin (logged)		2.441*** (.193)	1.426*** (.188)
Constant	-2.122*** (.099)	-7.174*** (.400)	-5.271*** (.376)
N	5819	5819	5819

p-value, ^ 0.10 * 0.05 ** 0.01 *** 0.001

Table 7. Ordinary Least Squares Regression Models Predicting C-Reactive Protein (logged) for Males, NHANES 2007-2010

Variable Name	Model 1	Model 2	Model 3
Poor White Male	.279*** (.048)	.212*** (.044)	.206*** (.045)
Poor Black Male	.276*** (.066)	.151* (.066)	.212** (.067)
Non-Poor Black Male	.232** (.065)	.126* (.062)	.152* (.060)
Poor Hispanic Male	.300*** (.075)	.284*** (.073)	.220** (.066)
Non-Poor Hispanic Male	.256** (.091)	.237* (.098)	.198* (.095)
Class 1: Health Nut (reference)			
Class 2: Lean and Having a Good Time			.224*** (.059)
Class 3: <i>Big</i> Drinkers			.851*** (.054)
Age	.015*** (.002)	.008*** (.002)	.006** (.002)
Less than High School		.164** (.053)	.179** (.050)
High School		.212** (.059)	.186** (.054)
Foreign Born		-.194** (.060)	-.075 (.063)
Foreign Born * Years in U.S.		.000 (.003)	-.000 (.002)
Widowed Divorced or Separated		.104 (.064)	.122^ (.063)
Never Married		-.101 (.067)	-.054 (.064)
Cholesterol		.002** (.001)	.002** (.000)
Diastolic Blood Pressure		.005^ (.002)	.002 (.002)
Systolic Blood Pressure		.002 (.002)	.002 (.002)
Glycohemoglobin (logged)		1.435*** (.183)	.934*** (.177)
Constant	-2.712*** (.087)	-5.873*** (.364)	-5.131*** (.323)

N	5623	5623	5623
p-value, ^ 0.10 * 0.05 ** 0.01 *** 0.001			

Appendix A.

Summary Statistics Comparison of Study Variables for Non-Imputed and Imputed, Weighted NHANES 2007-2010

Variable Name	Non-Imputed Data w/ Survey Weights						Imputed Data w/ Survey Weights	
	N	Mean / Proportion	Standard Error	Min.	Max.	% Missing	Mean / Proportion	Standard Error
C-Reactive Protein	10411	.39	.01	.01	9.51	9.01	.39	.01
Males	5111	.33	.01	.01	9.51	9.11	.32	.01
Females	5300	.44 ***	.01	.01	9.49	8.92	.45 ***	.01
Age	11442	47.28	.34	20	80	0	47.28	.34
Males	5623	46.31	.38	20	80	0	46.31	.38
Females	5819	48.20 ***	.40	20	80	0	48.20 ***	.40
Age								
20-44	4,611	.46		0	1	0	.46	
Males	2,265	.48		0	1	0	.48	
Females	2,346	.45 *		0	1	0	.45 *	
45-64	3,863	.36		0	1	0	.36	
Males	1,921	.36		0	1	0	.36	
Females	1,942	.36		0	1	0	.36	
65+	2,968	.18		0	1	0	.18	
Males	1,437	.16		0	1	0	.16	
Females	1,531	.20 ***		0	1	0	.20 ***	
Sex								
Male	5,623	.49		0	1	0	.49	
Female	5,819	.51		0	1	0	.51	
Race and Ethnicity								
Non-Hispanic White	5,703	.74		0	1	0	.74	
Males	2,851	.73		0	1	0	.73	
Females	2,852	.74		0	1	0	.74	
Non-Hispanic Black	2,323	.12		0	1	0	.12	

Males	1,141	.11		0	1	0	.11	
Females	1,182	.13	**	0	1	0	.13	**
Hispanic	3,416	.14		0	1	0	.14	
Males	1,631	.15		0	1	0	.15	
Females	1,785	.13	***	0	1	0	.13	***
Educational Attainment								
Less than High School	11424	.20		0	1	.16	.20	
Males	5617	.20		0	1	.11	.20	
Females	5807	.20		0	1	.21	.20	
High School	11424	.25		0	1	.16	.25	
Males	5617	.26		0	1	.11	.26	
Females	5807	.24	*	0	1	.21	.24	*
More than High School	11424	.55		0	1	.16	.55	
Males	5617	.54		0	1	.11	.54	
Females	5807	.57	*	0	1	.21	.57	*
Marital Status								
Never Married	11437	.18		0	1	.04	.18	
Males	5620	.20		0	1	.05	.20	
Females	5817	.16	**	0	1	.03	.16	**
Divorced, Widowed, or Separated	11437	.19		0	1	.04	.19	
Males	5620	.14		0	1	.05	.14	
Females	5817	.24	***	0	1	.03	.24	***
Married	11437	.63		0	1	.04	.63	
Males	5620	.67		0	1	.05	.67	
Females	5817	.59	***	0	1	.03	.59	***
Foreign Born	11437	.14		0	1	.04	.14	
Males	5620	.15		0	1	.05	.15	
Females	5817	.13	***	0	1	.03	.13	***

Foreign Born * Years in U.S. (Mean Centered)	11278	-.25	.09	-21.40	27.60	1.43	-.26	.09
Males	5544	-.38	.12	-21.40	27.60	1.40	-.39	.12
Females	5734	-.13 *	.08	-21.40	27.60	1.46	-.14 *	.08
Socioeconomic Disadvantage	10338	.35		0	1	9.65	.35	
Males	5101	.32		0	1	9.28	.32	
Females	5237	.37 ***		0	1	10.00	.38 ***	
Body Mass Index	10921	28.74	.09	13.18	50	4.55	28.74	.09
Males	5358	28.71	.12	14.20	50	4.71	28.71	.12
Females	5563	28.77	.13	13.18	50	4.40	28.77	.13
Obese	10921	.36		0	1	4.55	.36	
Males	5358	.35		0	1	4.71	.35	
Females	5563	.37		0	1	4.40	.37	
Waist Circumference (Inches)	10437	38.74	.12	23.27	56	8.78	38.80	.12
Males	5147	39.87	.15	25.08	56	8.47	39.91	.14
Females	5290	37.66 ***	.14	23.27	56	9.09	37.74 ***	.14
Abdominal Obesity	10437	.54		0	1	8.78	.55	
Males	5147	.46		0	1	8.47	.46	
Females	5290	.62 ***		0	1	9.09	.63 ***	
Physical Activity (METs/Week)	11411	227.19	1.32	168	646	.27	227.20	1.32
Males	5604	248.44	1.92	168	646	.34	248.46	1.92
Females	5807	207.03 ***	1.24	168	646	.21	207.03 ***	1.24
Insufficient Physical Activity	11411	.35		0	1	.27	.35	
Males	5604	.27		0	1	.34	.27	
Females	5807	.43 ***		0	1	.21	.43 ***	
Alcohol Use								
Non-Drinker	8718	.19		0	1	23.81	.19	
Males	4209	.12		0	1	25.15	.12	
Females	4509	.25 ***		0	1	22.51	.26 ***	

Moderate Drinker	8718	.39		0	1	23.81	.38		
Males	4209	.45		0	1	25.15	.45		
Females	4509	.33	***	0	1	22.51	.32	***	
Heavy Drinker	8718	.43		0	1	23.81	.42		
Males	4209	.44		0	1	25.15	.43		
Females	4509	.42		0	1	22.51	.41		
Smoking Behavior									
Non-Smoker	11156	.54		0	1	2.50	.54		
Males	5455	.48		0	1	2.99	.48		
Females	5701	.61	***	0	1	2.03	.60	***	
Former Smoker	11156	.23		0	1	2.50	.23		
Males	5455	.27		0	1	2.99	.27		
Females	5701	.20	***	0	1	2.03	.20	***	
Current Smoker	11156	.22		0	1	2.50	.23		
Males	5455	.25		0	1	2.99	.25		
Females	5701	.20	***	0	1	2.03	.20	***	
Cholesterol	10381	196.44		.59	90	313	9.27	196.29	.57
Males	5106	193.85		.82	90	313	9.19	193.71	.79
Females	5275	198.91	***	.76	91	313	9.35	198.75	***
Diastolic Blood Pressure	10502	70.70		.35	40	132	8.22	70.68	.34
Males	5197	72.35		.34	40	121	7.58	72.33	.33
Females	5305	69.12	***	.38	40	132	8.83	69.11	***
Systolic Blood Pressure	10581	121.66		.31	80	185	7.52	121.64	.31
Males	5226	123.42		.29	82	185	7.06	123.39	.29
Females	5355	119.99	***	.41	80	185	7.97	119.98	***
Glycohemoglobin	10457	5.60		.02	2.00	10.70	8.61	5.55	.00
Males	5132	5.62		.02	3.70	10.70	8.73	5.57	.00
Females	5325	5.58	*	.02	2.00	10.70	8.49	5.54	.00

Note: Table 3.1 contains means/proportions, standard errors for means, and minimum and maximum values for each variable. Asterisks denote significant differences between men and women: * $p < .05$. ** $p < .01$. *** $p < .001$

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