Social Disparities in Biological Risk Factors of Cancer in Young Adulthood: Obesity, Inflammation, and Socio-behavioral Mechanisms

Yang Claire Yang*

Moira Johnson**

Kristen Schorpp**

Courtney Boen**

Kathleen Mullan Harris**

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*Department of Sociology, Lineberger Comprehensive Cancer Center, and Carolina Population Center, University of North Carolina at Chapel Hill.
**Department of Sociology and Carolina Population Center, University of North Carolina at Chapel Hill.

Abstract

Introduction: The paper aimed to assess social disparities in the burdens of metabolic and inflammatory risks for cancer in the young adult population in the U.S. and examine psychosocial and behavioral mechanisms in such disparities.

Methods: Using data of over 7,000 individuals aged 12 to 32 from the National Longitudinal Study of Adolescent to Adult Health (Add Health) 1994 to 2009, we estimated generalized linear models to test the sex, race/ethnicity, and socioeconomic status (SES) differences in the risks of obesity and inflammation. We further tested the extent to which social isolation, smoking, physical inactivity, alcohol abuse, and illicit drug use explain social differentials in each biomarker outcome.

Results: Females, blacks and Hispanics, SES disadvantaged groups had higher risks of obesity and elevated C-reactive protein, with the SES gradients being more pronounced in females. Health related behaviors show large variation across sex, race, and SES strata. Adjusting for these behavioral variables, sex and race disparities in obesity and black excess in inflammation diminished, whereas the adolescent SES disparity in obesity remained. The effects of adolescent and young adult SES disadvantage on inflammation were also explained away by behavioral mechanisms. Behavioral factors associated with higher risks of obesity and inflammation differ, with the exception of fast food consumption, a risk factor for both.

Conclusions: The study provides new knowledge of social distribution of early-life exposures to physiological precedents to cancer development later in life with implications for prevention and early intervention of modifiable risky behaviors in adolescents and young adults.

INTRODUCTION

Cancer, accounting for more than a half million deaths annually, is a major and ever increasing public health concern as it has surpassed cardiovascular disease as the leading cause of death in 22 states in the U.S.[1, 2] Because most cancer diagnoses occur in adults 50 years of age, prior population and clinical research on cancer and its risk factors has focused on later adulthood. However, cancer is a chronic disease of aging that takes decades to develop and manifest. Research suggests that the onset of cancer is often preceded by a lengthy latency period, with clinically detectable levels of cellular dysfunction often not occurring until years after initial exposure to carcinogenic agents.[3, 4] It was further suggested that adolescent and early adult circumstances have enduring impacts on late life chronic disease outcomes, with implications for caner in particular.[5, 6] While little is known about the specific etiology linking early life circumstances to later life cancer development, previous studies indicate that young adulthood exposures to socioeconomic disadvantage, nutrition, physical activity, environmental toxin, and risky behaviors such as cigarette smoking may all play a role.[7-10] Often lagged time periods between the risk factor exposure and cancer onset are significant: rates of lung cancer incidence frequently lag population smoking rates by approximately 20 years.[3] Such findings point to a need for a life-course approach to cancer prevention by understanding the timing and duration of exposures in order to modify risk factors and delay cancer onset or slow cancer progression.

Given the low incidence rates of cancer in young adulthood, physiological pathways involved in carcinogenesis are especially important "intermediate endpoints" that signal the earliest, preclinical stage of the disease process. Obesity and inflammation are two prominent examples of pre-disease pathways amenable for intervention and hence present opportunities for research applying a life-course model to early cancer prevention. Obesity has been linked to increased risks of multiple cancers, including breast, colorectal, endometrial, kidney, pancreatic, liver, and esophageal cancers, accounting for an estimated 20% of all cancer cases.[11-13] While the

specific metabolic and hormonal mechanisms linking obesity to cancer are under investigation[14, 15] the high likelihood of adolescent obesity status persisting into adulthood suggests the unambiguous necessity of reducing obesity risk early on to curtailing the growth of cancers. Systemic inflammation can act synergistically with obesity to increase cancer risk.[16] While obesity increases low-grade inflammation, the presence of inflammation as indicated by elevated circulating levels of proinflammatory cytokines (e.g., interleukin-6) and acute phase protein (e.g., C-reactive protein or CRP) also plays a crucial role in tumorigenesis independent of obesity. Inflammation impacts every part of the cancer development process from increased likelihood of somatic mutations to the likelihood and extent of metastasis.[17, 18]

In the context of the recent obesity epidemic that has affected all age groups in the U.S. population, the secular increase in obesity in younger individuals is alarming. The rate of obesity in adolescents has quadrupled over the past 30 years to 17%[19] and more recent cohorts, such as those born after 1955 and also 1980s, show increased risks of obesity than earlier cohorts, with the increase being particularly sharp for black females.[20, 21] Previous studies have documented substantial social differentials in obesity as well as biomarkers of low-grade inflammation, with women, blacks and Hispanics, and lower socioeconomic status (SES) groups having greater risks of obesity[22-24] and exhibiting higher levels of CRP.[25-28] Much less is known, however, about patterns of social disparities in the distributions of these biological risk factors for cancer in young adulthood.

Multiple behavioral and psychosocial factors have hypothesized links with inflammation, obesity, and cancer. Cigarette smoking is associated with elevated risks of CRP[29] and a well-established cause of many leading cancers and related mortality.[30, 31] Social integration has been linked to lower levels of markers of physiological stress response such as CRP, fibrinogen, metabolic syndrome[32, 33] and better cancer survival, [34, 35] whereas social isolation increases inflammation and risks of cancer mortality.[36, 37] A large body of research shows clear associations between nutrition in terms of caloric intake and diet quality with obesity

and cancer.[38, 39] In fact, obesity and the high-fat, low-vegetable western diet have been suggested to be the "largest avoidable cause of cancer in nonsmokers."[13]Physical inactivity may further heighten the risk of various cancers via its effects on adiposity and obesity, as well as immune activation and inflammation.[8] Alcohol consumption and illicit drug use (such as marijuana use and opioids) have been linked to certain cancers, although the findings are mixed and complicated by differences in the degree of alcohol use, the confounding influence of cigarette smoking, and small sample sizes.[40-43]

Individuals of lower social status are disproportionally exposed to adversities and higher levels of social stress that in turn increase disease susceptibilities through harmful behaviors and prolonged physiological stress response.[44-47]Adolescence and young adulthood are critical periods that set the stage for lifetime trajectories of social and physical well-being but are understudied in current research on cancer disparities. Cross-sectional and singular measures of SES (e.g., current education, or income) are widely used but fail to capture the dynamic and multidimensional nature of socioeconomic standing specific to each life periods. The extent to which disadvantaged and poor adolescent and young adult population in the U.S. suffer from high risks of obesity and inflammation is unknown. The role of social behavioral risk factors, all of which more modifiable early in life than later in life, in shaping social disparities in biological precursors to cancer is unclear. Our study intends to fill these gaps using the largest population-based prospective cohort study of adolescents and young adults in the U.S. We examine the sex, race/ethnicity, and life-course SES differences in obesity and CRP. We further examine six health-related behaviors, including social isolation, daily smoking, physical inactivity, consumption of fast food, alcohol abuse, and illicit drug use, as behavioral mechanisms underlying social disparities in obesity and inflammation.

METHODS

Data for our study come from the National Longitudinal Study of Adolescent and Adult

Health (Add Health), a nationally representative study of over 20,000 adolescents in grades 7-12 in the US in 1994-95 who have been followed into adulthood. Add Health used a stratified school-biased design and selected a nationally representative sample of all high schools and a feeder school in the United States. An in-school questionnaire was administered to all students who attended the selected schools during 1994-95 (wave I). An in-home sample was then selected from the school rosters for more in-depth interviews in the home setting with adolescents and a parent at wave I. The Add Health cohort were followed up in 1996 (wave II), 2001-2002 (wave III), and finally in 2008-2009 (wave IV). The study sample includes 7,889 participants aged 12-19 at wave I (adolescence) and followed up at ages 24-32 in wave IV (young adulthood). High-sensitive C-reactive protein (hsCRP) comes from assays of dried blood spots collected at wave IV. Height and weight measured at interviews at both wave I and IV were used to calculate the body mass index (BMI). For more information about Add Health biomarker collection see previous publications.[48]

The independent variables and covariates for the present study are drawn from the in-school questionnaire and the in-home interviews at wave I as well as the in-home interview at wave IV. An adolescent SES disadvantage index was compiled as a count of items reflecting parents' status at wave I including parental welfare receipt, education and/or income in the bottom quartile of the sample, parent unemployment, and single-parent household structure. The adolescent index ranges from 0-5, with 5 representing the highest level of disadvantage. A similar index was compiled based on the respondents' own status at wave IV. The young adult SES disadvantage index ranges from 0-3 with items for welfare receipt, low-education, and low-income. Adolescent social isolation is a binary indicator of no participation in any volunteer work, low levels of interaction with parents living in the household (in the bottom quartile of responses), being in the bottom quartile for number of friendship contacts, and less than monthly religious attendance at wave I. Respondents were classified as regular cigarette smokers if they had smoked at least one cigarette each day for the past 30 days. Respondents were considered to

be physically inactive if they participated in aerobic activities less than three times per week. An item for fast food consumption indicates whether respondents had eaten at a food restaurant at least once in the past seven days. Alcohol abuse was defined as having been regularly drunk three or more time per week, experiencing legal problems due to drinking, and/or having been a risk to oneself or others due to drinking. Finally, illicit drug-use was defined as the use of one or more illegal drugs or the abuse of prescription drugs at least once in the past year.

The analytic sample for each biomarker outcome (N = 7,889 for BMI; N = 6,747 for CRP) included respondents who had complete data on all covariates used in the analysis and those with valid sampling weights. Most missing data are due to respondents lacking in-school surveys at wave I for the construction of social integration score and the corresponding social isolation variable (N = 3,474). Those with missing measures of smoking and other covariates were also excluded (N = 89). The weighted descriptive statistics of all variables in the sample are reported in Table 1.

Statistical Methods

We conducted multivariate regression analyses to examine the associations between each biomarker outcome with social status characteristics and behavioral factors. We estimated both the OLS models for continuous measures of CRP (log-transformed to account for skewness) and BMI and generalized linear models for categorical outcomes of inflammation (CRP categorized as 0-1, 1-3, and >3mg/dl) and obesity based on clinical cut points. Model fit statistics such as the Bayes Information Criterion (BICs) suggest that the logistics models of obesity and OLS models of LnCRP provide the best fit to data on each outcome. We estimated models in a stepwise fashion: 1) bivariate models with no adjustment of other covariates; and 2) full models adjusting for all covariates. All analyses adjusted for survey design effects and nonresponse using sampling weights. Analyses were conducted using Stata SE 14.

RESULTS

Figure 1 shows the sex, race/ethnicity, and SES gradients in the proportions of obesity and elevated CRP in young adults in the Add Health sample. Females, blacks, and Hispanics were more likely to be obese and had elevated CRP than their male and white counterparts. The other race group, mostly Asian Americans and Native Americans, had significantly lower risks of obesity and elevated CRP. Higher SES in adulthood as indicated by lower count of SES disadvantages is related to lower proportions of obesity and elevated CRP. And these differentials are all statistically significant, as are early life SES measured for adolescents (see coefficients in Table 2). In addition, females also demonstrated a stronger negative slope in the likelihood of obesity as SES increased than males. Although the mean levels of obesity and inflammation varied by sex and race/ethnicity, the general SES gradient did not vary significantly across these groups, with a possible exception of obesity in black and Hispanic males who showed increases in obesity risk with increases in SES ($p<.064$, two-tailed).

[Insert Figure 1]

Table 2 further shows evidence for the associations of risky psychosocial and health behaviors with obesity and inflammation. The bivariate model of obesity shows that in addition to the large positive association of adolescent obesity with young adult obesity, social isolation in adolescence increased the odds of obesity in young adulthood by 23% (Odds Ratio [95%CI] = 1.23 [1.02-1.48], $p<.05$). Physical inactivity and fast food consumption are also associated with significant increases in the odds of obesity in young adulthood (OR= 1.36 and 1.35 respectively). Alcohol abuse (OR = 0.59) and illicit drug use (OR=0.71), on the other hand, have negative associations with obesity. The corresponding CRP model shows significant bivariate associations of physical inactivity and fast food consumption as well as obesity with inflammation.

[Insert Table 2]

To examine the extent to which social gradients in obesity and inflammation may be

due to corresponding gradients in these behavioral factors, we compared patterns of variation risky psychosocial and health behaviors by social status characteristics. Figure 2 illustrates that the proportions of unhealthy behaviors declined as SES increased in general. However, there are significant sex differences in both the mean levels and SES differentials in these behaviors. Males had higher levels of social isolation, cigarette smoking, fast food consumption, alcohol abuse, and drug use, but lower rates of physical inactivity than females. The sex gaps declined among those with lower SES disadvantage. Figure 3 presents distributions of each behavioral factor by race among males (results for females similar). White males had lower levels of physical inactivity and fast food consumption than the other race groups, but the highest rates of cigarette smoking and drug use. Black males had the highest levels of physical inactivity, fast food consumption, and alcohol abuse, but the lowest rates of drug use. Hispanic males and the other race category were similar in levels of smoking, physical inactivity, and alcohol abuse, falling between the rates for whites and blacks. And the other race group had lower rates of fast food consumption. Race differences in the prevalence of social isolation and alcohol abuse were not statistically significant.

[Insert Figures 2 & 3]

Multivariate results in Table 2 show that adjusting for all social and behavioral variables, social disparities in obesity and inflammation were attenuated. In the full model for obesity, the ORs for sex and race effects were no longer statistically significant, suggesting that SES disadvantage and other social behavioral factors accounted for much of the sex and racial differences in obesity risk. While the adolescent SES disadvantage remained a significant predictor of obesity risk in young adulthood, the effect of young adult SES disadvantage was explained away by health behaviors. Net of the other factors, obesity in adolescence, fast food consumption, and physical inactivity are all significantly associated with the likelihood of obesity in young adulthood, while smoking and drug use are negatively associated with the likelihood of obesity.

In the full model for CRP, sex and race coefficients remained statistically significant, but blacks no long showed more inflammation than whites after adjusting for behavioral risk factors. SES disadvantage in either adolescent or young adulthood was no longer predictive of CRP levels. The significant coefficients for social isolation, cigarette smoking, physical inactivity, fast food consumption, and obesity suggest that they are potentially important mechanisms underlying the SES-inflammation link.

DISCUSSION

Our study makes unique contributions to extant literature on cancer disparities and prevention. First, although the social gradient in cancer risk is well documented, its underlying mechanisms are not. Metabolic dysregulation and inflammatory processes are integral parts of the initiation and progression of many cancers. Our study of the biosocial linkages shows how social status "gets under the skin" to influence cancer biology. Second, we adopt a life course approach that helps to illuminate points of intervention in early life periods that can more effectively curtail the emergence of adverse bodily change relevant to cancer and delay or prevent the onset of malignancy. Third, substantial heterogeneity in biological risk factors by sex, race, and SES in the current study sheds light on the early life origins of social disparities in cancer risks later in life. An open question that remains in previous research on social factors at a point in time is when a particular social factor would matter and for how long. The specification of SES disadvantage as well as other environmental exposures at different points in time, i.e., adolescence and young adulthood, allows the examination of the timing and duration of their harmful effects in early life. In support of a sensitive period model,[4] for instance, our findings indicate the lasting influences of the adolescent SES on obesity risk and social isolation on inflammation in early adulthood. Other behavioral risk factors are contemporaneously associated with obesity and inflammation and contribute to the social stratification of these biomarkers in young adults. These findings aid the ascertainment of the specific time window in which modification would provide maximum benefits.

Limitations

The study has limitations for future investigations to consider. First, there are few other biomarkers of immune functions that may be important to include in studies of cancer biology. Second, CRP is not available at wave I to permit a longitudinal analysis of change in inflammation over time. The findings in this study are thus best interpreted as prospective associations of baseline social status and inflammation at the follow-up. Third, the Add Health is an on-going study and has yet to provide more longitudinal follow-up data on the current cohort of young adults as they age into mid adulthood when cancer incidence starts to increase. Until then, there can be no definitive conclusion about the life course pathways linking social status, inflammation and related biological mechanisms, and cancer outcomes.

Conclusions

Our study has provided new knowledge about differential exposures early in life to physiological precedents to cancer development later in life in the general population. Many social and cultural changes occurring in the U.S. related to gender, race, and SES based exposures to risk factors for cancer (e.g., educational attainment, poverty, perceived stress and discrimination, substance use, etc.) in younger adults may continue to shape and modify the projections of cancer burden on the aging society in the future. Social structural and behavioral mechanism examined in the life course context here provide some tangible steps to take because they evidently influence the rate of development of the underlying cancer pathology and are preventable and modifiable years before they eventuate in cancers.

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Table 1. Sample Characteristics: Weighted Descriptive Statistics, Add Health: 1994 - 2009.

Variable	All Mean (SD)	Male Mean (SD)	Female Mean (SD)	P value
Age, years	27.8 (1.7)	27.9 (1.6)	27.7 (1.6)	**0.002**
Race/Ethnicity, %				
White	74.1	74.5	73.6	0.51
Black	10.7	8.9	12.5	**0.01**
Hispanic	10.3	10.1	10.4	0.58
Others	4.9	5.3	4.7	0.09
SES Disadvantage				
Adolescent (0 - 5)	.7 (1.1)	.66 (1.0)	.72 (.9)	0.05
Young Adult (0 - 3)	.7 (.9)	0.67(.6)	0.71 (.9)	
Social Isolation, % (adolescent)	17.7	19.7	15.9	**0.004**
Current Cigarette Smoker, %	23.8	25.8	21.9	**0.008**
Physically Inactive, %	15.8	14.5	16.9	**0.000**
Fast Food Consumption, %	74.6	76.4	75.2	**0.009**
Alcohol Abuse, %	4.9	6.9	2.9	**0.000**
Illicit Drug Use, %	11.1	13	9	**0.000**
Obesity, % (BMI>=30)				
Adolescent	9.5	9.6	9.2	0.557
Young Adult	34.1	33	36.5	**0.029**
CRP, mg/dL	3.7 (5.3)	3.0 (6.5)	4.3 (9.4)	**0.000**
N[a]	7,889	4,138	3,751	

Note: Boldface indicates statistical significance for sex difference (t-test for mean and Chi-squared test for proportions; two-tailed). Unless otherwise specified, variables were measured in 2009 when respondents were ages 24-32 (young adulthood).

[a]N's based on the sample size for the obesity outcome and slightly smaller for the CRP sample (N = 6,747)

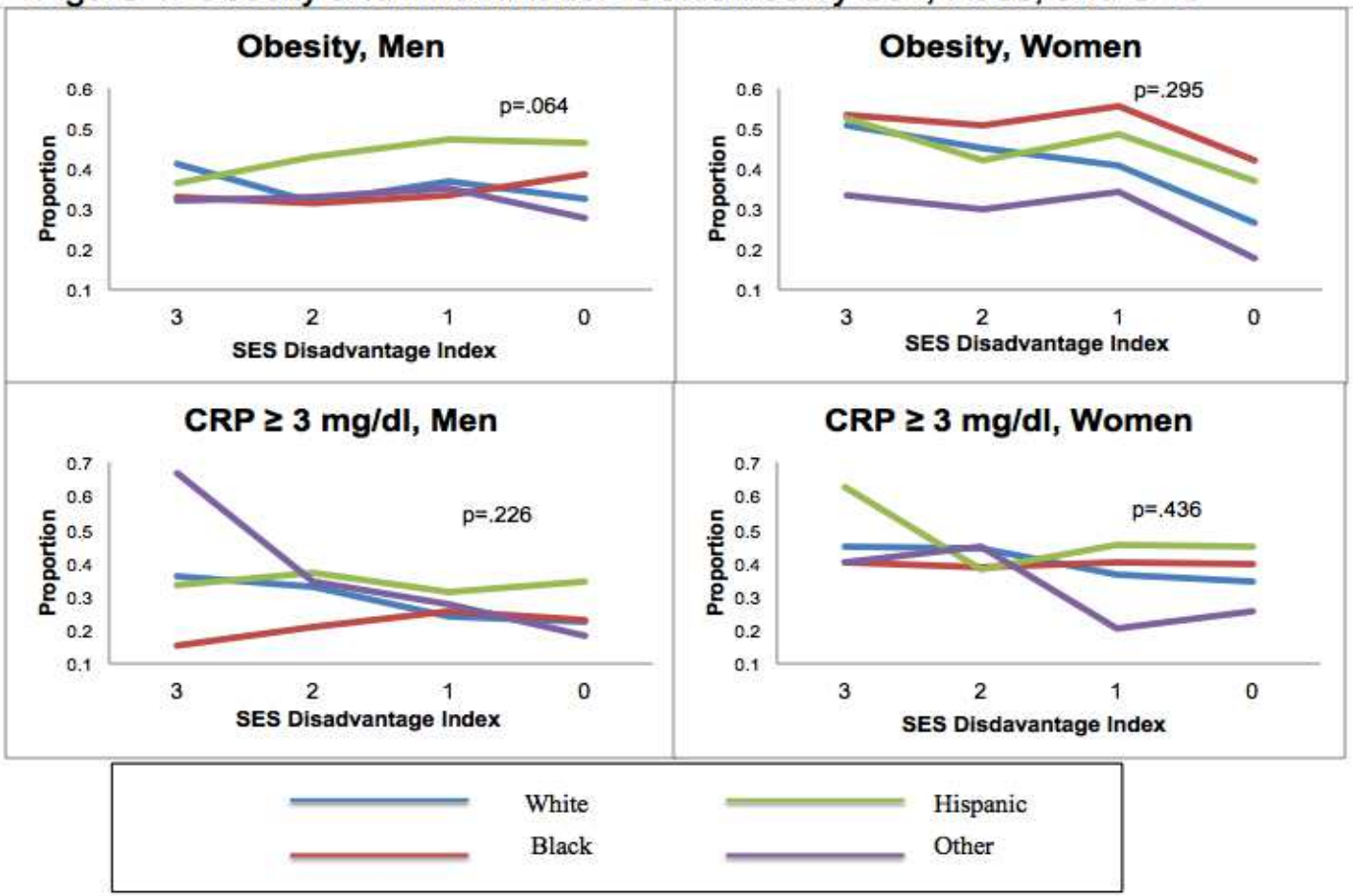
Abbreviations:
CRP, C-Reactive Protein;
SES, Socioeconomic Status

Table 2. Estimated Associations of Social Status and Health Behaviors with Biomarkers of Cancer Risk

Variable	Obesity (N = 7,889)				Ln(CRP) (N = 6,747)			
	Unadjusted Odds Ratio	(95% CI)	Fully Adjusted Odds Ratio	(95% CI)	Unadjusted Coefficient	(s.e.)	Fully Adjusted Coefficient	(s.e.)
Sex (Female = 1)	**1.16***	1.01-1.34	1.10	0.92-1.31	**0.57*****	0.05	**0.56*****	0.07
Race/Ethnicity								
White (ref.)								
Black	**1.67*****	1.40-1.95	1.22	0.91-1.64	**0.19***	0.08	0.04	0.1
Hispanic	**0.68***	0.49-0.95	1.31	0.93-1.85	0.16	0.09	**0.27***	0.12
Others	1.23+	0.98- 1.56	0.70	0.43-1.16	**-0.54*****	0.11	**-0.41****	0.13
SES Disadvantage								
Adolescent	**1.18*****	1.09-1.25	**1.13***	1.01-1.26	**0.09****	0.027	0.01	0.03
Young Adult	**1.21*****	1.11-1.32	1.11	0.98-1.27	**0.11****	0.034	0.03	0.04
Age	**1.05***	1.00-0.47	1.00	0.94-1.06	0.02	0.016	0.01	0.02
Social Isolation (adolescent)	**1.23***	1.02-1.48	1.19	0.91-1.57	-0.005	0.07	**0.122***	0.05
Current Cigarette Smoker	0.87	0.73-1.03	0.77	0.59-1.00	-0.031	0.068	**0.109***	0.05
Physically Inactive	**1.36****	1.12-1.65	1.31	0.95-1.79	**0.24****	0.078	0.18	0.09
Fast Food Consumption	**1.35*****	1.14-1.60	**1.64*****	1.28-2.08	**0.26*****	0.07	**0.24****	0.08
Alcohol Abuse	**0.59***	0.38-0.88	0.71	0.43-1.16	0.007	0.15	0.24	0.17
Illicit Drug Use	**0.71****	0.56-0.89	**0.71***	0.51-.99	-0.037	0.1	0.15	
Obesity								
Adolescent	**22.88*****	14.55-35.98	**19.33*****	10.81-34.55	**1.03*****	0.09	**0.62*****	0.62
Young Adult					**1.1*****	0.05	**0.96*****	0.96

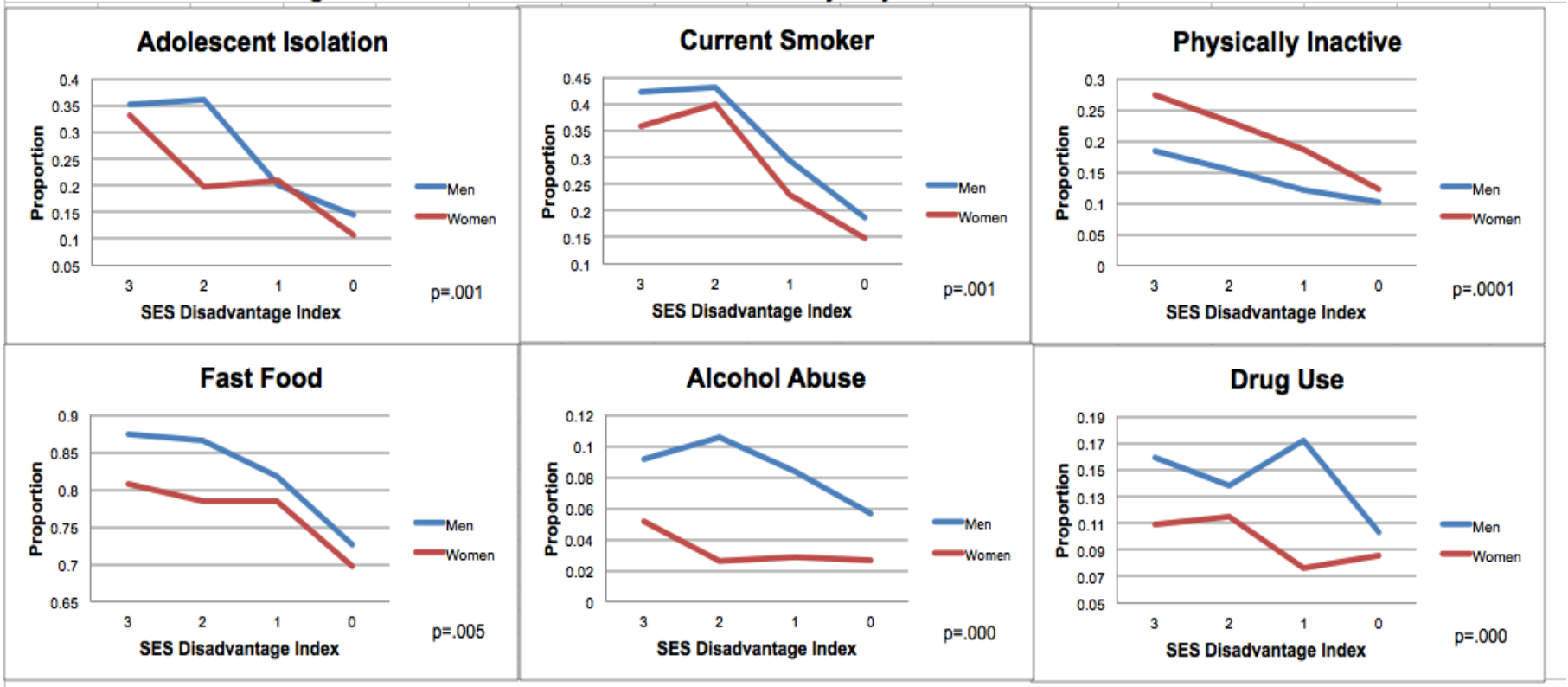
Note: boldface indicates statistical significance
(*p<.05; **p<.01; ***p<.00, two-tailed)
Abbreviations:
CRP, C-reactive protein
SES, Socioeconomic Status

Figure 1. Obesity and Inflammation Outcomes by Sex, Race, and SES



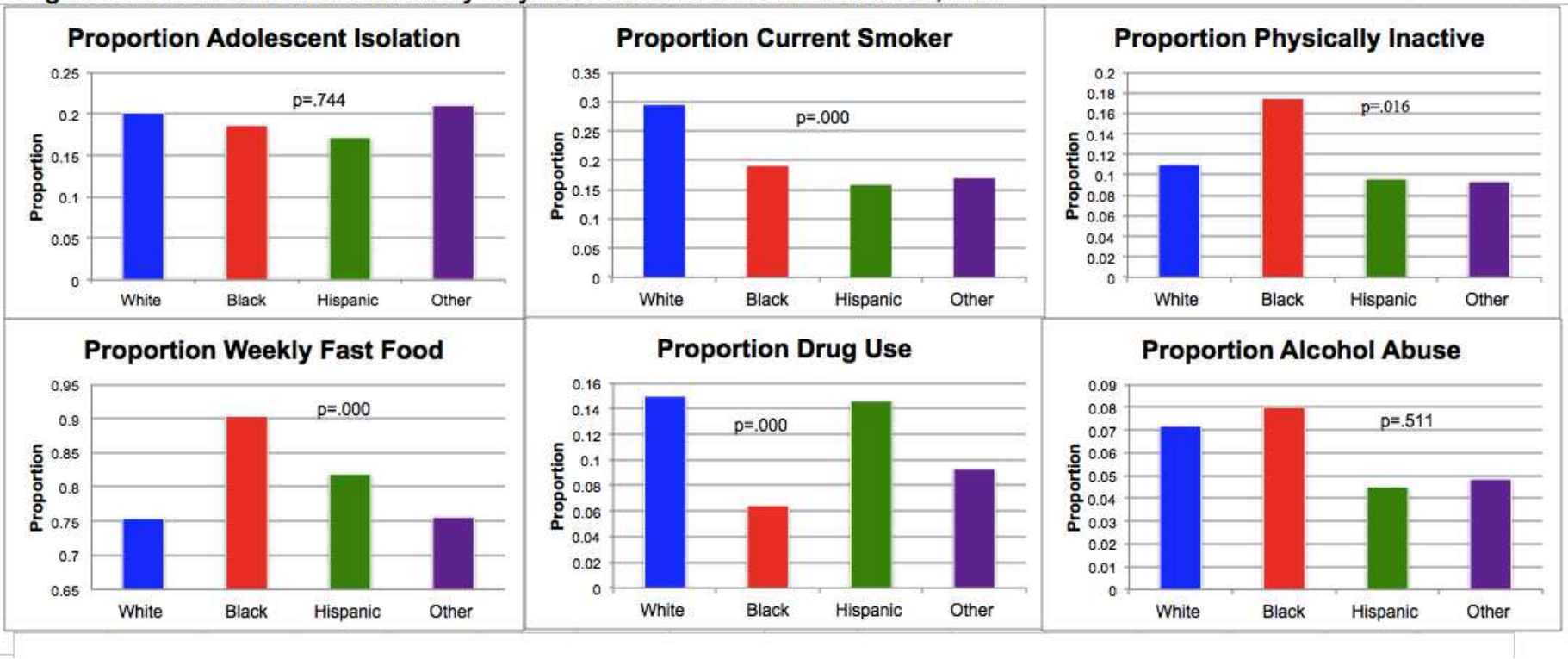
Note: Wald test for equality of coefficients calculated using logistic regression models adjusting for age, sex, race, and SES; two-tailed.
SES Disadvantage Index (for young adults) comprises items for respondent welfare receipt, respondent low-education, and respondent low-income and ranges from 0 (no disadvantage) to 3 (most disadvantage).
Abbreviations: CRP, C-reactive protein; SES, Socioeconomic Status

Figure 2. Sex and SES Differentials in Risky Psychosocial and Health Behaviors



Note: Wald test for equality of coefficients calculated using logistic regression models adjusting for age, sex, race, and SES; two-tailed.
SES Disadvantage Index (for young adults) comprises items for respondent welfare receipt, respondent low-education, and respondent low-income and ranges from 0 (no disadvantage) to 3 (most disadvantage).
Abbreviations: SES, Socioeconomic Status

Figure 3. Race Differentials in Risky Psychosocial and Health Behaviors, Men



Note: Wald test for equality of coefficients calculated using logistic regression models adjusting for age, sex, race, and SES; two-tailed.