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The Influence of Coping With Perceived Racism and Stress on Lipid Levels in African Americans

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Abstract

Background—Lipid dysregulation is a major contributor to cardiovascular disease (CVD) risk and is attributed to numerous biological, psychosocial, and behavioral risk factors. Psychological stress has been examined as a predictor of lipid dysregulation; however, the role of coping with perceived racism, a stressor unique to the African American experience, has not been addressed. The current study sought to determine the impact of behavioral coping responses to perceived racism and perceived daily stress on lipid levels in African Americans.

Methods—The sample consisted of 122 African American participants who resided in the Washington, DC, metropolitan area. Data were collected as part of an ongoing study entitled *Stress and Psychoneuroimmunological Factors in Renal Health and Disease* at Howard University Hospital.

Results—Through canonical analysis, distinct profiles of African American lipid function emerged with body mass index, age, and behavioral coping responses to perceived racism being associated with high-density lipoprotein cholesterol (HDL-C), triglycerides, and low-density lipoprotein cholesterol (LDL-C), respectively. Results from linear regression analyses showed that greater endorsement of behavioral coping responses to perceived racism items predicted higher levels of LDL ($\beta = .24, p < .05$). This relationship was not mediated by pathophysiological mechanisms associated with the stress response system such as cortisol, norepinephrine, epinephrine, and IL-6.

Conclusion—The relationship between elevated levels of LDL and behavioral coping responses to perceived racism suggests that African Americans may be at increased risk for CVD due to the unique stress encountered by racism in our culture. Behavioral pathways used to counteract the negative effects of perceived discrimination may better explain this relationship. Further research is necessary to determine other biobehavioral and pathophysiological mechanisms that explain this relationship.

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Keywords

lipids; racism; stress; African Americans

Cardiovascular disease (CVD) includes a cluster of conditions that interfere with the vascular supply of oxygen to the heart, brain, and other organs in the body. As one of the leading causes of death and disability in the United States, CVD imposes a substantially greater burden on the cardiovascular health of African Americans as compared to other ethnic groups.^{1,2} Among the numerous factors that contribute to CVD mortality and morbidity, lipid abnormalities continue to pose a significant health risk. Increased serum lipid levels give rise to the accumulation of lipids in the arterial wall, inflammation, atherosclerosis, and CVD.³ Atherosclerosis is an inflammatory condition and major risk factor for CVD. Atherosclerosis is precipitated by the buildup of plaque at damage sites along the endothelial lining of the artery, which often leads to endothelial dysfunction and subsequent rupture. These plaques rupture, forming blockages in the lumen of blood vessels, which may cause a heart attack, stroke, or CVD.³

A growing number of studies have shown that psychosocial and behavioral factors are also related to serum lipid levels. Among the biopsychosocial and behavioral risk factors that have been implicated in lipid dysregulation, body mass index (BMI),⁴ socioeconomic status,⁵ cigarette smoking, and physical inactivity are most prominent.⁶ These factors have been studied in African Americans; however, few studies have examined the relation between these factors and other novel risk factors in a community-based sample of African Americans. There are other risk factors that may further explain variability in lipid levels among African Americans. Additional research is necessary to understand what traditional and novel risk factors contribute to the development of lipid dysregulation in African Americans.

Recent studies have examined the impact of psychological stress on lipid dysregulation.^{7–10} Evidence from several research studies has shown that changes in serum concentrations of low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglyceride levels occur as a consequence of both chronic and acute psychological stress.^{11–14} The temporal parameters associated with chronic and acute stress are fundamental to understanding the stress response system and its impact on lipid levels. Under more acute situations, changes in lipid levels occur temporarily and are assumed to return to baseline, whereas under more chronic situations, this is not the case. The temporal characteristics of chronic and acute psychological stress help to explain their long-term negative health consequences.¹⁵ Chronic and acute stress conditions activate the hypothalamic-pituitary-adrenal (HPA) and sympathetic adrenal medulla (SAM) axes, 2 systems that play a vital role in the homeostatic regulatory processes that the body faces when challenged by stress.¹⁶ With the activation of the HPA and SAM axes, chronic and acute psychological stress trigger the production of proinflammatory cytokines.^{17,18} Notably, the prolonged activation of these systems under more chronic stressful situations has long-term deleterious health consequences.¹⁹ Elevated levels of LDL-C also trigger cytokine production, which play a major role in the development of atherosclerotic

plaques.²⁰ Chronic elevation of cytokines, such as IL-6, may precipitate the onset of inflammatory diseases, such as CVD.^{21–23}

Despite evidence to suggest the negative impact of psychological stress on lipids, its influence on lipid function in African Americans has not been extensively studied. The psychological stress associated with the daily hassles in the lives of African Americans needs to be better understood in the context of lipid function. Moreover, the psychological stress associated with racism also needs to be investigated. Racism is a psychological stress unique to many in the African American community. Research has suggested that perceptions of racism and exposure to racist events remain prevalent in the lives of African Americans.²⁴ This reality is a chronic and significant stressor for many African Americans.^{24,25} African Americans are exposed to racism and discrimination on many levels of society. The psychological stress associated with racism is experienced in the workplace, at school, and other public settings.

Experience with racism throughout one's lifetime arguably demands that African Americans develop a chronic anticipatory state to prepare for the possibility of a racist threat or situation. As a consequence, the body's physiological response to an objective or perceived threat increases levels of cortisol and blood pressure, as well as compromises immune function. This chronic anticipatory state can remain present even when racist or discriminatory acts do not occur. The adverse health consequences of experiencing racism have been well documented.^{26–28} Research has demonstrated the adverse impact of perceived racism on blood pressure and cardiovascular reactivity.²⁹ Many of these studies have primarily examined health outcomes and exposure to types of racist events. It is critical to examine not only exposure to racism but also the coping responses to perceived racism. The authors of the current study argue that it is the coping responses to perceived racism that elicit physiological responses. The impact of behavioral coping responses to perceived racism is assumed to follow the pathophysiologic pathways seen in previous research on psychological stress and lipid level dysregulation.

The current study examined the relation between perceived daily stress and lipid levels in African Americans. In addition, the behavioral coping responses to perceived racism were also included to assess the influence of a distinct psychological stressor that is qualitatively different from everyday hassles African Americans experience in life. Therefore, the study sought: (1) to determine if behavioral coping responses to perceived racism and perceived daily stress are associated with a specific profile of lipid levels in African Americans; (2) to test the associations between behavioral coping responses to perceived racism and perceived daily stress and independent lipid indices; and (3) to determine if the effects of behavioral responses to coping with perceived racism and perceived daily stress on lipid levels are reduced when the biological mechanisms involved in the stress response process are introduced into the statistical models.

METHODS

Participants

Data were collected in conjunction with the Minority Organ Tissue Transplant Education Program (MOTTEP) *Stress and Psychoneuroimmunological Factors in Renal Health and Disease Study*. MOTTEP is a national organization founded at Howard University Hospital whose aim is to promote and facilitate organ and tissue donation within the African American community. The current study is part of an ongoing project aimed at identifying and reducing biological and psychosocial predictors of renal disease as well as devising interventions to reduce the risk of renal disease in the African American population. The study was conducted in the metropolitan Washington, DC, area, an area with a large concentration of African Americans. The *Stress and Psychoneuroimmunological Factors in Renal Health and Disease Study* employs a design consisting of physiological, cognitive, and psychosocial assessment.

The sample consisted of 122 African American males and females (47% male). The mean age for the sample was 43.61 years (SD = 12.60; range, 21–73 y). The mean years of education for the sample was 14.12 (SD = 2.42). Exclusion criteria for the overall study included a history of psychiatric illness or report of current illicit drug use. Neither lipid dysregulation nor a family history of lipid dysregulation was included as exclusion criteria. The sample was relatively healthy, with 11 % reporting a diagnosis of diabetes and 20% reporting a diagnosis of hypertension; however, the cardiovascular profiles of participants suggest that many were at risk for developing CVD. Participants were asked about current medication use, including vitamin supplements and over-the-counter medications; cigarette use; and alcohol consumption. Only 5% of participants reported taking lipid medications. They received monetary compensation for completion of the study requirements.

Biological Measures

Nonfasting blood samples were collected by nursing staff at the General Clinical Research Center (GCRC) via venipuncture using 21- and 23-gauge syringes to obtain nonfasting LDL-C, HDL-C, and triglycerides. Cholesterol and triglycerides were assayed at the GCRC using standard enzymatic techniques. Blood samples were also used to determine levels of cortisol, epinephrine, and IL-6. Samples were drawn into 4 cryovials and frozen at -70°C . After clotting, blood samples were centrifuged and aliquoted into 6 vials and stored at Howard University Hospital until collected by Quest Laboratories, where samples underwent enzyme-linked immunosorbent assaying to detect and quantify cortisol, epinephrine, and IL-6. Nursing staff also used a balance scale to measure weight and height of the participants. Weight and height measurements were used to calculate BMI by converting weight from pounds to kilograms and converting height from inches to meters. The participants wore clothing during the measurement. Three seated blood pressure measurements were obtained by nursing staff using a Criticare Model 506DXNT sphygmomanometer (Criticare Systems, Waukesha, Wisconsin). The average of the 3 blood pressure measurements was utilized in the analyses. BMI was measured.

Psychological Measures

A team of trained graduate research assistants administered an extensive battery of psychological instruments to participants. Among the assessments were the Perceived Racism Scale, the Perceived Stress Scale, and the 36-Item Short Form Health Survey (SF-36). The Perceived Racism Scale³⁰ is an instrument consisting of 51 items, allowing respondents to describe their experience with and reaction to racist incidents both recently and throughout their lifetimes. The scale has an internal consistency of 0.87 to 0.95 for a community-based sample. Responses are categorized into distinct domains. Only the behavioral coping responses to perceived racism subscale were used in the current analysis. Behavioral coping responses were selected to capture not simply the perception of racist experiences but the individual's behavioral response to the act, which ultimately determines the physiological response and consequences of the act. The Perceived Stress Scale³¹ is a 10-item instrument designed to measure the degree to which an individual perceives and appraises life events as stressful. The total score was utilized in the analysis. Both test-retest reliability and predictive validity of the scale have a coefficient³¹ of 0.85. The SF-36 is a 36-item instrument³² that examines general concepts of health related to functional status and well-being. Only the Physical Functioning subscale was used here.

Procedure

Data were collected at the GCRC in Howard University Hospital. The protocol for the study was approved by the Howard University institutional review board (IRB). The study required 4 to 6 hours for completion. Following a general presentation of the global project, each participant completed an IRB-approved consent form. Demographic information and health history were recorded for each participant. After completing the preliminary forms, participants were taken to the laboratory for blood pressure measurements and venous blood draws. Participants were seated and a blood pressure cuff was attached to the upper arm. After the baseline blood pressure reading, blood samples were obtained. After physiological data were obtained, a battery of neurocognitive, psychosocial, and spiritual measures was administered. A second blood pressure reading followed the neurocognitive battery, and the final reading followed the psychosocial and spiritual measures. All participants completed the study in the same order. The ethnicity of participants, graduate students, and the nurse were matched.

Statistical Approach

The statistical analyses were executed in 3 stages: a canonical analysis, a multiple regression analysis, and a mediation analysis. The dependent variables were 3 components of the lipid panel: HDL-C, LDL-C, and triglycerides. The primary independent variables for this study were perceived stress and behavioral coping responses to perceived racism. Age, gender, and income (used as a proxy for socioeconomic status) were covariates. Physical functioning, BMI, and smoking habits, which tend to have proximal influences on lipid levels, were introduced as additional covariates.

When the goal of a research study is to study the relationships between 2 sets of variables, as is the case in this study, it is preferable to use a type of statistical analysis known as canonical analysis. Canonical analysis maximally correlates a linear combination of

dependent variables with a linear combination of independent variables. Both correlations between variables within a set of variables and between sets of variables are examined. Variables within each set are clustered to form canonical variates, a linear combination of the independent variables maximally correlated with a linear combination of the dependent variables. The canonical variate is a representation of an underlying construct, or *latent variable*, which is made up of relationships between the sets of variables. Once the canonical variates are derived, those which are made up of statistically significant pairings from both sets of variables are examined. The next step is to examine and interpret the composition of the canonical variates to yield conclusions about the underlying relationships. Each variate is characterized by a canonical weight contributed by each independent and dependent variable. The weights are interpreted as factor loadings are interpreted within a factor analysis. Larger weights connote a larger contribution of a variable to the variate than smaller weights. A canonical analysis was carried out to study an initial, social-behavioral model of interrelations between the set of 8 independent variables and the set of 3 dependent variables simultaneously. The aim at this stage was to see if distinct problems in lipid regulation could be linked to distinct psychosocial profiles.

Significant canonical correlations were followed up by multiple regression analyses. Regression analysis was applied to determine how much variance in each lipid index could be accounted for and which predictors were comparatively most salient within each clinical profile. Finally, the regression analysis was extended to the study of a second, more inclusive model, which added cortisol, norepinephrine, epinephrine, and IL-6 as possible biomarkers, which may mediate the relationship between psychosocial factors and lipid regulation. These biomarkers were selected because of their reported role in the stress response. In the absence of standardized cortisol testing, cortisol values were adjusted for the time assessed due to fluctuations of cortisol levels throughout the day. The time of day that cortisol was assessed was utilized to create predicted, time-adjusted cortisol values for the analysis.

RESULTS

The means, standard deviations, and frequencies for demographic characteristics of the sample, including age, education, and income, are provided in Table 1. In addition, the means, standard deviations, and ranges of the independent and dependent variables included in the analyses are presented in Table 1.

The results of the canonical analysis are summarized in Table 2, where correlations between each variable and the canonical variates are presented. As mentioned earlier, the aim at this stage was to see if variability in lipid levels was linked to distinct biopsychosocial factors. Three canonical variates reached the level of significance. HDL-C figured most prominently on the first canonical variate, with a coefficient of -0.94 . Equally important on the predictor side of the canonical variate was the independent variable BMI with a coefficient of 0.86 . The weight of these 2 variables suggested a strictly metabolic process represented by this variate. Triglycerides figured most prominently on the second canonical variate, with a coefficient of -0.76 . Equally important on the predictor side of the canonical variate was the independent variable age, with a coefficient of -0.80 . Taking the weight of this pair of variables into consideration, one could infer that this variate accounted for a developmental

aspect of lipid regulation. LDL-C figured most prominently on the third canonical variate, which had the highest coefficient of 0.92. The salient independent variable on the other side of the equation was coping with racism, with a coefficient of 0.74. The coupling of these 2 variables suggested that this canonical variate reflected a biopsychosocial phenomenon. Upon closer examination of the dependent variables, the variates appeared to represent 3 distinct clinical profiles. The first profile, with a high negative coefficient on HDL-C, but relatively moderate levels of LDL-C and triglycerides, provided the closest picture of lipid dysregulation. The profile suggested by the second canonical variate was characterized by low levels of triglycerides and LDL-C, which would be a desirable condition if it were not for the relatively low level of HDL-C. The third canonical variate, with a high positive value on LDL-C, seemed to define a typical profile of elevated cholesterol. The canonical correlations were equal to 0.65 for the first variate, 0.45 for the second, and 0.41 for the third variate.

The results of the initial stage of the regression analysis are presented in Table 3 under model 1. The predictors for HDL-C were, in order of importance, BMI ($\beta = -.54$) and gender ($\beta = -.39$). In other words, being male and having lower BMI were associated with higher levels of HDL-C. Together, all of the independent variables explained 35.2% of the variance HDL-C. The most salient predictors for triglycerides were BMI ($\beta = .46$) and age ($\beta = .31$). In other words, the older a person was and the higher the BMI, the more elevated the triglyceride levels. The independent variables accounted for 35.1% of the variance in triglycerides. The significant predictor for LDL-C was coping with perceived racism ($\beta = .28$). In this instance, having to cope with racism contributed to elevated levels of LDL-C. This variable helped to explain 4.9% of the variance in LDL-C.

The final stage of the analysis broadened the model with the addition of 4 new variables to the regression equation: cortisol, epinephrine, norepinephrine, and IL-6. These biological predictors did not significantly change the amount of variance explained in HDL-C; however, higher levels of IL-6 were significantly associated with lower levels of HDL-C ($\beta = -.24$). There was also no significant change in the amount of variance explained in triglycerides. The amount of variance in LDL-C was not significantly changed by the addition of the biological predictors; however, the relation between perceived coping with racism and LDL-C persisted after the introduction of these predictors ($\beta = .24$).

DISCUSSION

The aim of this study was to determine: (1) if distinct lipid profiles are associated with behavioral coping responses to perceived racism and perceived daily stress in African Americans; (2) if behavioral coping responses to perceived racism and perceived daily stress are predictive of specific lipid indices; and (3) if the statistical introduction of biological factors such as cortisol, epinephrine, norepinephrine, and IL-6 would reduce the association between behavioral coping responses to perceived racism and perceived daily stress. The canonical analysis identified 3 distinct profiles in lipid function in African Americans. The first canonical variate revealed the closest clinical picture of unhealthy lipid function. In this profile, higher BMI yielded lower levels of HDL-C and moderate levels of triglycerides and LDL-C. This finding has been supported in the research on BMI and lipid levels.³³ BMI is

associated with higher LDL-C and triglycerides and lower HDL-C and predictive of conditions such as atherosclerosis and CVD.^{34,35} Behavioral coping responses to perceived racism and perceived daily stress were not prominent in this profile. This finding is not unusual given previous research that demonstrated similar findings, where psychological stressors did not yield changes in LDL-C, HDL-C, or total cholesterol.⁸ The second variate identified age as a significant factor in the regulation of triglycerides. Higher triglycerides as a function of greater age is a developmental phenomenon that is not unexpected. Previous research has shown a positive relationship between age and triglyceride levels.³⁶ Again, behavioral coping responses to perceived racism and perceived daily stress did not figure prominently in this profile. The third canonical variate yielded the only psychosocial variable. Behavioral coping responses to perceived racism influenced lipid levels. Behavioral coping responses to perceived racism were associated with higher LDL-C levels. Chronic psychological stress has been previously associated with higher levels of LDL-C.¹⁴ This finding is not completely unexpected given previous research that has shown the deleterious health consequences associated with perceived racism.^{26,27}

This study also wanted to determine whether behavioral coping responses to perceived racism and perceived daily stress specifically predicted lipid levels. Perceived daily stress did not emerge as a significant predictor of HDL-C, triglycerides, or LDL-C. This finding was somewhat unexpected; however, prior research on current psychological stress and lipid function has yielded inconsistent results.⁸ However, independent of other psychosocial factors, behavioral coping responses to perceived racism were associated with higher levels of LDL-C. This relationship persisted even when epinephrine, norepinephrine, cortisol, and IL-6 were taken into consideration. Given this finding, it is not possible to account for the direct effect of behavioral coping responses to perceived racism on LDL-C simply by the introduction of biological factors. Since Cannon, the response to stress has been evaluated in terms of fight or flight. Coping with perceived racism seems to call for a third type of response that results from a constant heightened state of vigilance or arousal. It is our judgment that the perception of having to deal with racism creates a chronic anticipatory state, whereby having to cope requires a psychological state of preparedness for any future racist events. Our findings suggested that a stress activation mechanism other than the HPA and SAM axes may be responsible for the relation between behavioral coping responses and LDL-C.

An alternative explanation that may shed light on the relation between behavioral coping responses to perceived racism and LDL-C includes a behavioral pathway. It is possible that elevated LDL-C is the result of negative health behaviors that are used as behavioral coping responses.¹⁴ Research has suggested the negative health consequences of racism may be explained by a complex set of psychosocial and biobehavioral mechanisms that include psychosocial adversities, lack of access to health care, cognitive-emotional processes, and negative health behaviors.³⁷ Studies have indicated that African Americans use negative health behaviors such as poor eating habits, smoking, alcohol, and illicit drug use to cope with racism and discrimination.^{38,39} It is worth noting, in a previous analysis of this sample, that perceived daily stress was associated with unhealthy eating behaviors such as emotional eating and haphazard meal planning.⁴⁰ Consistent with poor eating habits, participants were,

on average, obese. These findings lend support for the existence of a behavioral pathway between behavioral coping responses to perceived racism and LDL-C.

Clinical Implications

Many of the findings in this study are similar to those found in previous studies on lipid regulation. The canonical analysis identified 3 profiles consistent with common lipid dysfunction. BMI emerged as significant in the profiles and provides support for implementing prevention and intervention treatment programs that target weight management, healthy eating choices, and moderate exercise. In addition, our findings on coping with racism suggest that this type of stress is unique to African Americans and impacts the physiological system in a manner that falls outside of the traditional conceptualization of stress and the stress response. The significance of this finding cannot be understated due to the importance of developing stress management programs that focus on dealing effectively with a stress response approach that arguably may serve a paradoxically “protective” function. African Americans have a historical precedence that has taught them to be prepared to cope with racism. This anticipatory state has arguably served as a protective mechanism against a social ill but has potentially had long-term health consequences. Further research is necessary to clarify the health consequences of having to cope with perceived racism.

Limitations

One of the limitations of the present study is that it did not include lifestyle factors, including diet and eating habits, that are related to lipid levels. Nevertheless, by analyzing lipid regulation in some detail, the main findings shed some light on a risk factor for the leading cause of mortality in the African American community. Another possible limitation of the current study was that lipids were measured in nonfasting individuals. According to the National Cholesterol Education Program Adult Treatment Panel III, guidelines suggest that lipids should be assayed after patients have been fasting. However, recent evidence suggests that there is not a clinically significant difference between fasting and nonfasting lipid panels in the general population.^{41,42} Furthermore, nonfasting lipids have been shown to be as good or better predictors of cardiovascular risk.^{41,43,44}

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Table 1

Descriptive Statistics

	M	SD
Age	43.86	12.22
High-density lipoprotein cholesterol, mg/dL	51.63	15.58
Low-density lipoprotein cholesterol, mg/dL	116.56	34.37
Triglycerides, mg/dL	90.79	60.85
Body mass index	31.58	9.47
Systolic blood pressure, mm Hg	132.92	17.70
Diastolic blood pressure, mm Hg	78.41	11.88
Physical functioning (SF-36)	24.99	5.82
Perceived stress	15.67	6.67
Coping with racism	8.22	5.08
Time-adjusted cortisol, nmol/L	9.39	4.13
Norepinephrine, nmol/L	33.88	16.87
Epinephrine, pmol/L	6.71	4.29
IL-6, pg/mL	2.80	4.43
	%	
Current smoker	25.4	
Yearly income		
\$10 000	20.5	
\$10 001–\$20 000	15.6	
\$20 001–\$50 000	47.5	
>\$50 000	16.4	

Table 2

Correlations Between Variables and Canonical Variates

	Canonical Variate 1	Canonical Variate 2	Canonical Variate 3
Dependent variables			
Low-density lipoprotein cholesterol	0.33	−0.20	0.92
High-density lipoprotein cholesterol	−0.94	−0.34	0.01
Triglycerides	0.65	−0.76	−0.06
Independent variables			
Age	0.15	−0.80	−0.15
Sex	0.25	0.65	0.04
Income	−0.39	−0.02	−0.05
Body mass index	0.86	−0.20	0.08
Physical functioning	−42	0.22	−0.50
Smoking	0.06	−0.17	−0.27
Perceived Stress Scale	0.35	0.23	0.07
Coping with racism	0.11	−0.13	0.74
Canonical R	0.65 ^a	0.45 ^a	0.41 ^a
Explained variance in dependent variables, %	47.00	24.54	28.54
Explained variance in independent variables, %	6.83	3.13	1.92

^a*p* < .01.

Table 3

Standardized Coefficients Between Dependent Variables and Predictors

Independent Variables	High-Density Lipoprotein Cholesterol		Triglycerides		Low-Density Lipoprotein Cholesterol	
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Age	-0.03	-0.10	0.31 ^a	0.29 ^a	0.06	-0.01
Sex	-0.39 ^a	-0.44 ^a	0.06	0.09	0.07	0.06
Income	0.11	0.07	-0.04	-0.02	0.08	0.07
BMI	-0.54 ^a	-0.50 ^a	0.46 ^a	0.46 ^a	0.07	0.17
Physical functioning (SF-36)	-0.06	-0.06	0.04	0.05	-0.24	-0.20
Smoking	0.11	0.16	0.13	0.12	-0.07	-0.07
Perceived stress	-0.14	-0.20	0.15	0.16	0.03	0.03
Coping with racism	-0.01	-0.07	0.09	0.11	0.28 ^b	0.24 ^b
Cortisol		0.05		-0.13		0.03
Epinephrine		-0.03		0.03		0.23
Norepinephrine		0.14		0.11		0.12
IL-6		-0.24 ^b		0.09		-0.12
% of variance explained	35.2	39.5	35.1	38.3	4.9	10.0

^a $p = | < .01$.^b $p < .05$.