Depressive Symptomatology: Relations to C-Reactive Protein in African American Men

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Abstract

Cardiovascular disease is a major contributor to mortality among African American men. While the biological factors that increase risk are relatively well understood, the psychological factors are not. Research suggests that C-reactive protein (CRP), a marker of cardiovascular risk, is associated with psychological mood states. This study examined the relationships among CRP and two types of depressive symptomatology in African American men. Data were collected from a sample of 98, African American, community-dwelling men in the metropolitan Washington, DC, area. A nurse collected blood samples (to assess CRP levels), blood pressure, and body mass index (BMI). Participants completed the Beck Depression Inventory-II and the Revised Neuroticism, Extraversion, and Openness Personality Inventory (NEO-PI-R)

Depression Subscale. Linear regression analysis was the major statistical method used to assess the predictive relationship between CRP and depressive symptomatology. Results suggest that 1) acute depression is not predictive of CRP in African American men; 2) depressive personality style may be predictive of CRP; and 3) BMI may suggest a biobehavioral pathway that links lifestyle behaviors to CRP. Findings from the current study partially support the hypothesis that depressive symptomatology is predictive of CRP levels in African American men. Depressive personality style is possibly an underlying psychological factor that may affect CRP levels. The clinical implications of these findings suggest a need for valid depression screening and treatment of African American men at risk for cardiovascular disease and lifestyle modifications to decrease BMI.

Introduction

Cardiovascular disease (CVD) is the greatest contributor to morbidity and mortality in the United States, and African Americans are disproportionately affected.[1,2] They have higher incidences of hypertension, diabetes, and obesity, which contribute to such disorders as atherosclerosis, myocardial infarction, stroke, and coronary heart disease.[3-5] These conditions are not only more prevalent in African Americans but occur at younger ages than in other racial groups.[6] Genetic risk factors do not fully explain these disparities.[7]

Emerging research suggests that psychological mood states and individual personality traits also contribute to CVD development.[8-10] Specifically, psychological factors, such as anger, hostility, depression, and anxiety, are related to greater incidence of CVD, fatal and nonfatal cardiac events in initially healthy people, coronary heart disease, and disease mortality in cardiac patients.[8, 11-13] The relationship of psychological phenomena to CVD has been extensively studied over the past few decades, but underlying biological mechanisms that connect psychological factors with CVD have only recently been reported.

One of the biological mechanisms that may explain this association involves an inflammatory process linked to elevated levels of C-reactive protein (CRP).[14] CRP is a sensitive, acute phase, nonspecific biomarker of systemic inflammation associated with acute coronary syndromes, chronic coronary disease, peripheral vascular disease, and stroke.[15-19] The documented relationships between psychological factors and CRP suggest a need to explore individual psychological conditions and their impact on cardiovascular health in at-risk populations.

As one of the most common psychiatric conditions in the United States,[20] depression is an important variable to consider as we attempt to learn more about how psychological factors affect CVD. Recent studies have associated intense depressive symptoms with higher blood plasma levels of CRP.[3, 21, 22] When associated with higher CRP levels, evidence also links minimal depressive symptoms with increased risk for CVD.[23]

African Americans are less likely to be diagnosed with depression than Caucasians.[24] The lifetime prevalence rate for African Americans is estimated at 10 percent; 7 percent for African American men.[25] These statistics, however, do not necessarily reflect the severity of the problem. The reasons for the underdiagnosis, particularly in African American men, include differences in symptom presentation and coping styles, poor screening and treatment, lack of access to affordable health care, stigma associated with mental illness, and the use of self-medications, such as alcohol and illicit drugs, to mask symptoms.[26-29] To our knowledge, few studies have examined depressive symptomatology and its influence on CRP in African American men. The need is critical because of the psychological implications as well as the morbidity and mortality associated with CVD.

The current study examined the relationship among depression, depressive personality style, and CRP in a community-based sample of African American men. Depression was operationalized as self-reported acute depressive symptoms having occurred within the previous two weeks. Depressive personality style was operationalized as self-reported depressive symptoms characterized by more enduring personality characteristics. We

hypothesized that greater self-reported acute and lasting depressive symptoms would be predictive of higher CRP levels in African American men.

Methods

Data Source

The study was part of an ongoing five-year project aimed at testing the validity of a model that identifies medical, psychosocial, and psychoneuroimmunological risk factors that predict impaired renal function and renal disease. This project was conducted in conjunction with the Minority Organ Tissue Transplant Education Program (MOTTEP) Stress and Psychoneuroimmunological Factors in Renal Health and Disease Study and involved assessment of physiological, cognitive, psychosocial, and spiritual/religious variables.

Participants

The sample consisted of 98, community-based, African-American men between the ages of 21 and 67. The mean age was 44.09 (SD = 12.16). Participants were recruited using conventional methods, such as flyers, health fairs, and word-of-mouth; contacted by telephone; and scheduled for a data collection appointment. Exclusion criteria included current domestic, emotional, or drug abuse and a previous diagnosis of a psychological or behavioral disorder. A positive history of these conditions was based on self-report. The sample was relatively healthy, with 11 percent reporting a diagnosis of diabetes and 20 percent reporting a diagnosis of hypertension. Participants were asked about current medication use, including vitamins and other over-the-counter medications, cigarette use, and alcohol consumption. They received monetary compensation for completing study requirements.

Biological Measures

Blood serum was collected by nursing staff via venipuncture procedure to determine CRP levels. Nonfasting blood samples were drawn into four cryovials and frozen at -70° celsius. After clotting,

blood samples were centrifuged and aliquoted into six vials and stored at the University General Clinical Research Center (GCRC) until collected by Quest Laboratories, where they underwent enzyme-linked immunosorbent assaying (ELISA) to detect and quantify CRP.

Three seated blood pressure measurements were obtained. Only the initial values were used in the analysis because the second and third readings were added only after the 92nd participant in the total sample was measured. Body-mass index (BMI) was assessed as the ratio of height to weight.

Psychological Measures

Depressive symptoms related to acute depression were assessed using the Beck Depression Inventory-II (BDI-II), a well-validated, 21-item, self-report measure used to determine severity of depression with scores highly correlated with a clinical diagnosis of depression.[30] Each item is scored on a 0 to 3 Likert scale, with the total score ranging from 0 to 63. The BDI-II has been widely used with patients in medical settings.[31]

Depressive personality style was assessed using the Revised Neuroticism, Extraversion, and Openness Personality Inventory (NEO-PI-R), designed to measure the broad personality domains of neuroticism, extraversion, openness to experience, agreeableness, and conscientiousness. Each of the five domains consists of six facets, and each facet is derived from eight items in the total inventory.[32] The depression facet scale measures feelings of guilt, sadness, despondency, and loneliness and was used for the current study.[32] Within an African American sample, internal consistency coefficients for the NEO-PI-R range from .74 to .86 for the domain scales and .30 to .73 for the facet scales.[33]

Procedures

Completion of the study required four to six hours and took place at the General Clinical Research Center (GCRC). Upon arrival at the facility, participants were given an overview of the study

requirements and procedures. All provided informed consent as required by the Institutional Review Board. They completed a demographic information form and a health history questionnaire. After completing the interview, they were taken to the laboratory, seated, and a blood pressure cuff was attached. After blood pressure readings, a butterfly needle was inserted into the forearm to obtain blood samples. After physiological data were collected by GCRC nursing staff on duty, trained graduate students administered a battery of neurocognitive, psychosocial, and spiritual measures. The ethnicity of participants, graduate students, and nurse were matched. The order of the study procedures was identical for all participants and lasted from three to five hours for each participant.

Statistical analysis

The Statistical Package for the Social Sciences (SPSS) 17.0 was used for all statistical analyses. Two hierarchical multiple regression analyses were run. For each, the first model tested whether higher levels of depressive symptomatology predicted higher levels of CRP in African American men. The second model tested whether higher levels of depressive symptomatology predicted higher CRP levels after controlling for variables known to be associated with CRP levels, including age, BMI, systolic blood pressure (SBP), smoking status, education, and income. Prior to inclusion in the regression models, CRP values were log transformed, and BDI-II scores were square-root transformed to correct for positive skew.

Results

Table 1 shows the characteristics of the sample. The first hierarchical regression tested the hypothesis that greater self-reported acute depressive symptomatology would predict higher CRP levels. This hypothesis was not supported. In the first model, BDI-II scores and CRP yielded a nonsignificant effect for the first model [$F(1, 41) = 3.75$, $p = .06$)]. BDI-II scores accounted for a nonsignificant proportion of variance within CRP levels (adjusted $R^2 = .06$). In the second model, BDI-II scores and the added covariates (age, BMI, SBP, smoking status, education, and income) yielded a significant effect (adjusted $R^2 = .36$). Driving

this effect was a significant positive association between BMI and CRP levels (ß = .66, $p < .01$).

The second hierarchical regression tested the hypothesis that enduring depressive symptomatology would predict higher CRP levels. Results from this regression for NEO Depression scores and CRP yielded a significant effect for the first model [$F(1, 37) = 4.36$, $p < .05$)]. This model accounted for 8 percent of the variance in CRP levels (adjusted $R^2 = .08$). Higher NEO Depression scores predicted higher CRP levels (ß = .33, $p < .05$). The second model was also significant [$F(7,31) = 6.72$, $p < .01$)] and accounted for 51 percent of the variance in CRP levels (adjusted $R^2 = .51$). In the second model, only BMI was a significant predictor of CRP levels, with higher BMI predicting higher CRP levels (ß = .70, $p < .01$).

Discussion

This study sought to determine if depressive symptoms in African American men predicted higher levels of CRP, an inflammatory marker used to assess risk for CVD. A depressive personality style was found to predict increased CRP levels in African American men, partially supporting our hypothesis. We did not find acute depressive symptoms to predict CRP levels. However, our findings were consistent with previous studies that demonstrated that personality traits are better predictors of inflammation and health outcomes than acute mood states.[34, 35]

Although a depressive personality style predicted elevated CRP in our sample, the relation did not remain after adjusting for age, BMI, systolic blood pressure, and smoking status. BMI accounted for elevated levels of CRP and explained much of the variability. The influence of BMI on inflammation is well documented and is associated with higher CRP levels.[22] The mean BMI in this sample was 29.6 kg/m2 and is considered overweight. Research has determined that depression is associated with inflammation through a biobehavioral pathway.[12] Previous research on our sample revealed that emotional eating and haphazard meal planning are common[37] which may provide evidence for a biobehavioral explanation. While maladaptive coping strategies were not examined here, the importance of BMI and its behavioral

predictors should be visited. The findings relating BMI and CRP also suggest that in exploring psychosocial explanations for increased CVD, researchers and practitioners should give special attention to individuals who are overweight or obese and already at greater risk for CVD due to higher CRP levels.

Even though the mechanism by which negative psychological factors influence inflammation is not fully understood or clearly explained by the current study, the significance of the findings should not be understated. How depressive symptoms manifest in African American men is not known; the overall consequences are problematic and present a serious concern for researchers, academicians, and clinical practitioners. The health consequences associated with elevated CRP and inflammation have been well documented,[38-40] and the sample in this study had a mean level that was elevated (3.04 mg/ml). This finding supports the need to investigate factors that influence elevated CRP levels in African American men and to ultimately reduce CVD risk.

The assessment of depressive personality traits is not usually conducted in primary health care settings. Findings suggest that the health care provider's understanding of how depression manifests in African American men may be critical to addressing the disparity in CVD risk between African American men and other racial groups.

Another cause for concern directly involves the future of the health care field and how it moves forward in addressing gaps in diagnosing and treating psychological disorders in African American men. The education and training of the next generation of health care practitioners requires a shift to recruit more people of color. The underdiagnosis and misdiagnosis of depression and other psychological conditions in African American men may reflect the reality that African American men are underrepresented in the field. The need for better diagnostic criteria, assessments, and treatment approaches will be significantly improved by the inclusion of more African American men in health care. As we move forward as researchers, academicians, and practitioners, it will be imperative that we consider the full scope of factors that contribute to poor health outcomes, including biological,

psychological, and social factors and how they may interact uniquely for patients of color.

Further research is needed to delineate these relationships to inform psychological treatment and recommendations for African American men. Specifically, attempts by health care providers to identify depressive symptomatology in African American men should not rely solely on state measures of depression, but examine for enduring depressive characteristics that may predict negative cardiovascular outcomes.

Future Directions

Findings indicated that the average participant had a high BMI and was overweight or obese. Future studies should investigate the relationship between overweight and obesity, depressive states and personality style, and CRP levels. Another future direction will determine whether the hypothesized relationships in the current analysis may operate in the inverse direction. Evidence suggests that inflammatory markers may affect psychological outcomes like depression. Although findings have been mixed, the scientific community recognizes systematic inflammation as a trigger for depressive symptoms. Specifically, cytokine communication with the brain has been implicated in the pathogenesis of depression.[43-45] Future studies should use longitudinal designs to examine the directionality of the depression/CRP link

Limitations

The small sample size in the current analysis may have limited significance; however, this convenience sample provided valuable initial findings for a population that has been understudied in the areas of mood, personality, and inflammation. In addition, the availability of more ideal measures of depression was limited. The BDI-II is a clinical tool used to help diagnose depression. Other measures might have been more relevant for a nonclinical sample; for example, the Center for Epidemiological Studies Depression Scale (CES-D)[46]. Last, blood samples were drawn at varying times during the day, which did not control for diurnal CRP variation.

Table 1: Sample characteristics		
	M	**SD**
Age	44.09	12.16
Education	13.40	2.25
Body-mass index (kg/m^2)	29.64	7.55
Systolic blood pressure (mmHg)	134.21	17.38
Diastolic blood pressure (mmHg)		79.82
12.98		
C-reactive protein		
(mg/mL)	3.04	4.08
	% endorsed	
Self-reported hypertension	20.4	
Self-reported diabetes	10.7	
Current smoker	18.4	
Annual income		
≤$10,000	21.4	
$10,001 - $20,000	17.5	
$20,001 - $50,000	51.5	
>$50K	9.7	
Marital status		
Single	64.1	
Married	17.5	
Divorced	10.7	
Separated	2.9	

Table 2: Hierarchical multiple regression: BDI-II scores and CRP levels

	Step 1			Step 2		
Predictors	B	SE	β	B	SE	β
BDI-II	.11	.06	.29	.03	.05	.09
Age				.001	.01	.03
BMI				.04	.01	.66**
SBP				.003	.003	.15
Smoking status				.21	.16	.23
Education				.04	.03	.21
Income				-.01	.05	-.02
R2	.06			.36		
ΔR2				.30		

Note. * p <. 05; **p < .01; BDI-II = Beck Depression Inventory II score, BMI = body-mass index, SBP = systolic blood pressure

Table 3: Hierarchical multiple regression: NEO Depression scores and CRP levels

	Step 1			Step 2		
Predictors	B	SE	β	B	SE	β
NEO Depression	.04	.02	.33*	.02	.01	.15
Age				.002	.01	.06
BMI				.04	.01	.70**
SBP				.003	.003	.14
Smoking status				.09	.14	.09
Education				.04	.03	.19
Income				.02	.05	.06
R2	.08			.51		
ΔR2				.43		

Note. * p <. 05; **p < .01; NEO Depression = NEO-PI-R Depression facet score, BMI = body-mass index, SBP = systolic blood pressure

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