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Quality of Life and Cardiovascular Disease Risk: Does the SF-36v2 Predict Unhealthy Cholesterol in a Population of Female African American Healthcare Workers?

By

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Master of Public Health

Global Epidemiology

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B.S., University of Michigan, 2009

Thesis Committee Chair: Carol R. Hogue, Ph.D., M.P.H.

An abstract of
a thesis submitted to the Faculty of the
Rollins School of Public Health of Emory University
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Abstract

Quality of Life and Cardiovascular Disease Risk: Does the SF-36v2 Predict Unhealthy Cholesterol in a Population of Female African American Healthcare Workers?

By Gabriela A. Aguilar

Background: Cardiovascular diseases are the number one killer of women in the United States (1). African American women share a disproportionate disease burden with a mortality rate nearly 1.4 times greater than that of their white counterparts (4). A number of barriers prevent African American women from assessing and managing their CVD risk (31). Therefore, there is much interest in identifying an easily accessible non-laboratory-based tool for assessing CVD risk factors such as low-density lipoprotein (LDL), high-density lipoprotein (HDL), and total cholesterol levels.

Objective: This study examines the appropriateness of the Short Form-36™ version 2 (SF-36v2) Physical Component and Mental Health Component scores as a risk assessment tool for high risk LDL, HDL, and total cholesterol levels.

Methods: Secondary analysis of cross-sectional data obtained from a longitudinal physical activity intervention targeting African American female healthcare workers.

Results: Crude prevalence ratios were statistically insignificant for the six associations of interest. Odds ratios adjusted for socioeconomic, lifestyle, and sociodemographic factors were also statistically insignificant.

Discussion: The data in the present study do not support the use of the SF-36v2 as an assessor of high risk cholesterol levels in the study population. Repeat studies are needed in order to validate these results and improve generalizability to other populations.

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This thesis is dedicated to my late grandmother, Maria C. Aguilar. While many years have passed, her words still inspire me every day.

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Introduction

Rationale

Cardiovascular disease (CVD) is now widely accepted by the scientific community as the number one killer of women, affecting over one in three American women (1). Raising awareness of CVD among women, especially in those that are at high risk, is a great public health concern. The American Heart Association's *Go Red for Women* campaign is one example of the response to the data on CVD in women. The initiative hopes to address the CVD burden shared by women through empowering them to take charge of their heart health and become aware of the controllable and uncontrollable CVD risk factors, with one of the most important risk factors being race/ethnicity (2).

That African American women disproportionately share the burden of many diseases when compared with white women is widely accepted and supported by publicly available data (3). For example, CVD mortality rates in 2007 were 286.1 and 205.7 per 100,000 African American and white women, respectively (4). It is believed that later diagnosis, higher individual risk factors, having more risk factors, and the presence of multiple risk factors explain some, but not all, of the elevated risk in African Americans (5). Given that CVD mortality is highest in African Americans at all ages, it is imperative that African American women be aware of their CVD risk through risk factor assessment (6).

CVD risk can be assessed by using one or more of the available risk estimation systems that have been developed based on studies of heart health throughout the world. A widely used example is the Framingham risk score. In addition to information gathered from a physical exam and family history, many risk estimation systems require blood testing for measurement of specific biomarkers (7). Access to laboratory equipment is required to determine the levels of important CVD biomarkers such as high-density lipoprotein (HDL), low-density lipoprotein (LDL), and total cholesterol. While helpful when available, laboratories are not always accessible to clinicians and patients who are limited by time, resources, or geography. Thus, the establishment of a useful non-laboratory-based predictor of CVD risk factors would help expand risk factor assessment to women, and their clinicians, without laboratory resources.

Previous research by Gaziano et al. showed that a model using non-laboratory-based indicators, such as physical examination and history, predicted cardiovascular events as accurately as their model that relied on laboratory-based values such as blood tests (8). This application of non-laboratory based predictors as an indicator of cardiovascular events begs the investigation of other non-laboratory-based resources as a predictor of CVD risk factors. Using these results as a foundation, the goal of this study is to determine if the Short Form-36 ™ Version 2 (SF-36v2), a measure of health-related quality of life, could be an appropriate measure of underlying or unknown cardiovascular disease risk in our population of female African American healthcare workers. Discovering a

predictive association could expand the use of the SF-36v2 as a preliminary indicator of underlying cardiovascular disease risk, thus limiting time consuming and costly laboratory tests to prime candidates at higher risk of developing CVD, expanding CVD risk assessment outside of the clinic setting, and potentially increasing earlier diagnosis of CVD.

Purpose of Study

The purpose of this study is to evaluate the use of self-reported health-related quality of life, measured by the SF-36v2, as a predictor of risk factors for cardiovascular disease among female African American healthcare workers. The risk factors under investigation include HDL, LDL, and total cholesterol. Evidence from this study may be used to determine if the SF-36v2 measure is appropriate for use by researchers, clinicians, and other public health professionals as an assessment of one's cardiovascular disease risk factors.

Objective and Research Questions

The objective of this research is to improve methods used to assess CVD risk and to determine if the SF-36v2 is an appropriate predictor of known CVD risk factors. The research questions guiding this study are:

1) Does higher health-related quality of life, as determined by the SF-36v2, correlate with less cardiovascular disease risk when controlling for Body Mass Index?

2) What model best predicts the relationship between heath-related quality of life and risk factors (i.e. LDL, HDL, and total cholesterol) for cardiovascular disease?

Assumptions

This study assumes that participants completing an in-depth questionnaire, including the SF-36v2 health-related quality of life measure, have reported accurately without any bias, and that survey administrators delivered the survey in a consistent manner. Furthermore, this study assumes that participants have the literacy level to correctly interpret and respond to the items and Likert rating scale. The study also assumes that laboratory testing was done accurately and consistently, producing unbiased results.

Literature Review

Upon evaluation of the health status of African American and white women, it becomes clear that a prominent disparity exists. Data from 2004 support this claim, revealing a greater prevalence of disease risk factors, such as high blood pressure, obesity, and smoking, higher rates of mortality due to cardiovascular diseases, cerebrovascular diseases, and cancer, and a shorter life expectancy in African American women than their white counterparts (9). Part of this disparity can be attributed to differences in socioeconomic factors recognized to alter disease risk, such as education and income. Current research focuses on identifying potential contributors to disease, such as stress and quality of life, and defining their roles in the development of disease.

It is believed that African American women are further burdened by the interaction between racial and gender stressors and react more negatively to these stressors. Even with this knowledge, the relationship between stress and disease is not well understood. Identification of points in the stress-disease pathway where medical or social interventions could be applied is crucial for eliminating health disparities attributable to stress. By studying intermediaries involved in the stress-disease pathway, such as health-related quality of life and disease risk factors, an important contribution can be made in the development

of interventions aimed at improving health-related quality of life and lowering individual risk factors.

This literature review will examine the relationship between health-related quality of life and CVD risk factors by first exploring the epidemiology of CVD in women, and a description of CVD in African American women will follow. In addition, a background of the CVD risk factors pertinent to this study will be provided. Furthermore, tools used for measuring health-related quality of life, studies on health-related quality of life in African American women, and the appropriateness of health-related quality of life as a predictor will be illustrated. Finally, this review will identify the importance and implications of establishing a relationship between health-related quality of life and risk factors for CVD.

Cardiovascular Disease in Women

Cardiovascular disease is now the leading cause of death throughout the world (10). In 2004, about one in five (over 32 million) women in the United States had at least one form of CVD (11). While myocardial infarction is a prevalent manifestation in men, angina pectoris is much more common in women (12). Furthermore, 46% of women will become disabled by congestive heart failure within 6 years of a recognized myocardial infarction, while only 22% of men will experience similar disability (11). The case of CVD in women is

of particular interest because of differential clinical manifestations than those that have been traditionally observed in men.

Gender differences are observed when examining events such as myocardial infarction, angina pectoris, and congestive heart failure. An increasing amount of evidence shows that gender differences exist beyond presentation of disease, becoming apparent in clinical characteristics and health outcomes (13-15). Upon the examination of acute myocardial infarction, one sees the differences in clinical manifestations; women are much more likely to present with non-traditional symptoms such as nausea and jaw pain, while men tend to present with traditional chest pain (16). Even so, women are much more likely to experience traditional chest pain, angina pectoris, rather than acute myocardial infarction or sudden cardiac death (17). Lastly, a worse prognosis for women with congestive heart failure may be ascribed to older age at diagnosis and a greater probability of having comorbidities than men (12).

Even though there are over 200 known risk factors for coronary heart disease, tobacco use, hypertension, diabetes, dyslipidemia, obesity, diet, and physical activity stand out as well established and widely accepted risk factors for CVD (18). An important aspect of CVD in women is their differential risk among risk factors as compared to men. For example, smoking rates in the United States have been declining over the past four decades, but the rate of decline is greater for men than it is for women (19). Furthermore, women will experience an increase in cholesterol levels after menopause, while their male

counterparts exhibit a decrease in cholesterol levels as they age (20). Emerging risk factors, such as levels of C-reactive protein, homocysteine, and lipoprotein (a), appear to have an association with coronary heart disease in women and will require further investigation for their establishment as a useful risk factor (21).

Lastly, unique barriers contribute to the difficulty in addressing CVD in women. Probably the greatest barrier is the lack of awareness of CVD risk among women, as shown by the work of Mosca and Legato (22, 23). In two separate studies, these authors discovered a gap between actual and perceived risk of coronary heart disease (22, 23). In addition, missed opportunities by physicians to counsel women on risk factor modification, such as exercise, nutrition, and weight reduction, has been documented in a CDC survey on CVD prevention (24). Finally, psychosocial issues, income, and environmental influences seem to prevent many women from living a healthy lifestyle, especially low-income women (25-27).

Cardiovascular Disease in African American Women

Race and ethnicity are important risk factors for CVD. Multiple studies have shown that minority women are at increased risk of developing CVD, even when controlling for age and partial control for socioeconomic status (28-30). In addition, the CVD mortality rate for African American women is nearly 1.4 times as high as in white women (4). Sundquist et al. reported significant associations between ethnicity and CVD risk factors such as Type II diabetes, smoking,

hypertension, abdominal obesity, and physical inactivity (29). The increased prevalence of CVD risk factors in African American women raises the issue of CVD risk awareness in the African American community.

While overall awareness of CVD among women has increased over the last decade, a gap between minority and white women still exists. A recent study by Mosca et al. shows that African American women are significantly less likely to be aware of CVD risk than white women, and face substantial barriers that hinder them from seeking preventive care (31). Some of the cited barriers included caregiving responsibilities and confusion in the media. African American women were also more likely than any other group to believe that their health is determined by God or a higher power (31).

Understanding the socioeconomic, religious, and cultural influences that differentially affect African American women and their ability to seek preventive care is important for developing interventions that will successfully target this high risk group. In addition, further understanding of these influences may reveal useful information on the intricacies of the association between race and CVD risk.

Cardiovascular Disease Risk Factors

Because recent data indicate that over one in three American women have some form of CVD, a substantial increase from 2004 estimates of one in five, there is a large interest in addressing this public health issue through risk factor

management (32). The extensive literature on risk factors for CVD includes discussions on laboratory and non-laboratory based predictors, risk specific to women, and high-risk groups such as minorities.

General risk factors that apply to all of the cardiovascular diseases (CVDs) include age, family history, cigarette smoking, dyslipidemia, hypertension, diabetes, inactive lifestyle, obesity, and poor nutrition (33), with racial and ethnic disparities for some, but not all, known risk factors. For example, African American women have the highest prevalence of obesity (47.3%) and hypertension (51.2% for those with less than a high school education and 37% for those with a high school degree or higher) when examining women by racial or ethnic group (6). Yet, African American women are not the only ethnic group that shares a disproportionate burden of CVD risk factors; 54% of Native American women have been diagnosed with diabetes (34).

As discussed earlier, angina pectoris is the most prevalent CVD in women. In fact, a significant sex ratio of 1.20 ($p<0.0001$) was reported by Hemingway et al., indicating a higher prevalence of this malady in women than men (35). Furthermore, they showed that the sex ratio did not differ much across regions of the world and when stratified by age. It is also important to note that this meta-analysis also found that the prevalence of angina was higher in American studies vs. other parts of the world and in non-white ethnic groups vs. whites. Risk factors for angina are those typical of many CVDs, but also include prior myocardial infarction or revascularization, resting electrocardiogram

abnormalities, carotid intima-media thickness, and coronary artery calcification (35, 36).

The death rate for coronary heart disease in black women is 140.9/100,000 person-years vs. 110/100,000 person-years in their white counterparts (33). It is also important to note that women are much more likely to suffer sudden coronary heart disease death without even knowing that they have coronary heart disease than men, 64% vs. 50% (37). Much attention is given to coronary heart disease because the mortality rate for women aged 35-44 has been increasing by just over 1% each year (38). Given this information, it is imperative to identify risk factors specific to coronary heart disease, especially those among women. One study from the 1980s indicated that bilateral oophorectomy without the use of hormones in premenopausal women increased risk of coronary heart disease (39). As a result, much research has been conducted on the effects of hormone replacement therapy on CVD risk in women (40-42).

Stroke is a major concern in the United States because of its debilitating effects. About 55,000 more women than men suffer from stroke each year, with black women aged 45-84 experiencing an incidence rate over two times higher than that of white females, 4.9/1,000 vs. 2.3/1,000 (33). This discrepancy between men and women is believed to be the result of longer life expectancy in women, but the racial disparity is not yet fully understood. Stroke risk factors unique to women include oral contraceptive use, improper stroke risk factor management, and pregnancy/postpartum period (43-45).

In 2010, approximately 2.5 million women in the United States were survivors of congestive heart failure, as measured by the number of hospital discharges for these patients (33). Although heart failure incidence rates are on the rise in the United States, this increase is thought to be attributable to the ageing American population and increased incidence rate in women (46, 47). As with other CVDs, racial disparities exist among congestive heart failure patients. 2008 estimates of the age-adjusted incidence rate of congestive heart failure in African American, white women, and white men 45-84 years of age were 8.1/1,000 person-years, 3.4/1,000 person-years, and 6.0/1,000 person-years (48). The data show that African American women fare even worse than white men, and is thought to be a result of the high prevalence of atherosclerotic risk factors in the African American community (33). The strongest risk factor for congestive heart failure among women is the presence of diabetes and obesity in postmenopausal women with known coronary heart disease (49).

Important non-modifiable risk factors for CVDs include age and family history. While men have a higher prevalence of CVDs than women in the first 40 years of life, women eventually surpass males around the seventh decade of life because of higher life expectancy. Even though most CVDs have complex genetic variants that vary by sex, some families have displayed Mendelian patterns of inheritance of coronary heart disease (32, 50, 51).

Modifiable risk factors such as tobacco use, diet, and physical activity remain as keys points of intervention. Lifestyle recommendations from the

American Heart Association include consuming an overall healthy diet, aiming for a healthy body weight, aiming for recommended levels of low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides, aiming for a normal blood pressure, aiming for a normal blood glucose level, being physically active, avoiding use of and exposure to tobacco products for decreasing overall risk of CVDs (52).

Many of the non-modifiable and modifiable risk factors discussed in this review are considered to be non-laboratory based risk factors (i.e. family history, age, diet, physical activity, and tobacco use). That is, they can be assessed without the use of laboratory blood tests (8). As of late, much focus has been given to identifying laboratory based biomarkers that indicate increased risk of CVD. Biomarkers of importance for characterizing atherosclerotic risk include LDL, C-reactive protein, interleukin (IL)-6, IL-10, IL-18, fibrinogen, and tumor necrosis factor-α, and many others have been identified for characterizing risk of unstable plaque, plaque rupture, thrombosis, and ischemia in the CVDs (53). Even so, challenges remain in identifying good biomarkers as the predictive value of many are modest at best (54).

The cholesterols have long been established as strong and independent predictors of coronary heart disease in men and women (55-57). One of the first studies to establish that an increase in total serum cholesterol level is associated with increased risk of coronary heart disease was the Framingham Heart Study (58). A substantial amount of evidence indicates that lowering levels of total

cholesterol and LDL decreases the risk of coronary heart disease, cardiovascular events, and coronary heart disease morbidity and mortality (59, 60).

Low-Density Lipoprotein

LDL level greater than or equal to 160 mg/dL is considered high risk by American Heart Association standards, and about half of older women have high risk levels of LDL (61, 62). LDL management is regarded as an important point of intervention since elevated levels of LDL are believed to play a major role in atherogenesis, giving it its nickname of 'bad' cholesterol (62). There is some evidence that indicates LDL as a better predictor of coronary heart disease than total cholesterol (63).

Similar to many of the risk factors mentioned above, LDL levels differ by racial group. Interestingly, National Health and Nutrition Examination Survey (NHANES) 2005-2006 data show that the average level of LDL in white women over 20 years of age is higher than their African American counterparts, 116.0 mg/dL and 109.7 mg/dL respectively, though both levels were not high risk (33). The average LDL level for all Americans over 20 years old was 115.0 mg/dL (33). For African American women, this is an improvement from previous NHANES data where the relationship was reversed (64).

High-Density Lipoprotein

HDL is often referred to as 'good' cholesterol because of its inverse relationship with CVD risk, where higher serum levels of HDL are associated with lower risk. The American Heart Association suggests that HDL levels below 50 mg/dL represent high risk (61). Early studies showed that young African American men and women and adult African American males had higher levels of HDL than whites, alluding to a protective effect given their excess of other CVD risk factors (62). It was hypothesized that the same effect was not seen in older African American females because of their excess of obesity; furthermore, diabetes and hypertension are also thought to lower HDL levels (62).

The latest NHANES data indicate yet another racial disparity in HDL levels. African American women were observed to have lower risk HDL levels. The national average HDL level for American adults over 20 years was 54.6 mg/dL, and 60.3 mg/dL and 62.1 mg/dL for white and African American women, respectively (33).

Total Cholesterol

The American Heart Association considers total cholesterol levels greater than or equal 240 mg/dL high risk (61). Like LDL and HDL, total cholesterol levels in the U.S. population have decreased over the past few decades (33). Even so, women still have slightly higher mean serum levels of total cholesterol,

202 vs. 198 mg/dL, and a higher population percentage of those with high risk cholesterol, 16.9% vs. 15.6%, when compared to men (65).

Given that African American women have levels of LDL and HDL that are at lower risk when compared to white women, we expect to see the same risk pattern when examining total cholesterol. The latest NHANES data indicate that this is the case; African American women have lower mean serum levels of total cholesterol when compared to white women, 195 mg/dL vs. 203 mg/dL respectively (65). Furthermore, the percentage of African American women with serum total cholesterol levels over 240 mg/dL is 13.3%, while white women have a higher proportion at 18.0% (65). Despite having overall lower risk LDL, HDL, and total cholesterol levels, African American women consistently display poorer health with regard to other risk factors for CVDs, indicating that the disparities present in these factors are substantial enough to nullify any expected effect associated with having lower risk cholesterol levels.

Health-Related Quality of Life

Health-related quality of life (HRQoL) is composed of several elements of overall quality of life that affect both physical functioning and well-being. Since the 1970s, over 20 HRQoL assessment tools have been developed by researchers. Generally speaking, assessment tools can be divided into three groups: generic, disease-specific, and preference-based. Generic assessors are very broad, with the intention to survey a variety of health concepts in populations diverse in

disease, severity, and comorbidity. Disease-specific measures are developed with disease pathology, effects of treatment, and natural history of the disease in mind. Preference-based tools are evaluators of health state value with life years, and are typically used for health planning and priority setting (66).

The past few decades have demonstrated a trend in simplifying earlier versions of generic HRQoL assessment tools. Early assessors included the Health Status Index, Health Insurance Experiment measures, and Human Population Laboratory measurement of physical, mental, and social health. The development of more practical and efficient short-form surveys resulted from concerns about cost and the burden of data collection throughout the 1980s. The Medical Outcomes Study SF-20 Health Survey was the first short form to appear, followed shortly by the Duke Health Profile, SF-36 Health Survey, Functioning and Well-Being Profile, and SF-6 (66).

Many of these HRQoL assessors have been applied to descriptive research, clinical trials, healthy policy, health planning, program evaluation, resource allocation, population surveys, and clinical practice (66). Given the variety in application setting, three criteria for selecting an appropriate HRQoL measurement tool have been developed. The first criterion is composed of conceptual considerations such as the definition health status or quality of life, determination of pertinent health constructs, and specification of associations for hypothesis testing. The second criterion, methodological considerations, focuses on reliability and power of the assessment tool. Finally, the third criterion

includes existing information on available candidate tools, such as previous use in a similar target population (67).

SF-36™ Version 2

The SF-36v2 is a generic measure of HRQoL derived from the earlier SF-20 of the Medical Outcomes Study. It is a 36 item survey that measures eight health domains: limitations in physical activities because of health problems, limitations in social activities because of physical or emotional problems, limitations in usual role activities because of physical health problems, bodily pain, general mental health (psychological distress and well-being), limitations in usual role activities because of emotional problems, vitality (energy and fatigue), and general health perceptions. The eight domains can be further simplified into physical component and mental health component summaries (68). Validity of this measure has been established using both psychometric and clinical criteria (69).

The generalizability of the SF-36v2 was assessed using 24 subgroups of the Medical Outcomes Study that varied greatly in diagnosis, disease severity, and sociodemographic characteristics. This sample exhibited high item completion rates, passed tests of item-internal consistency and item-discriminant validity, high reliability coefficients (median = 0.85). Furthermore, floor effects were negligible in six of the eight scales. Ceiling effects were observed in the role-physical, role-emotional, and social functioning scales. In consideration of this

information, the creators of the SF-36v2 determined that its use in a variety of populations is appropriate (70).

HRQoL is typically used to assess the effectiveness of a new treatment or intervention, but there exists an increased interest in its use as a predictor of disease risk or events associated with disease. Many studies describe HRQoL as an indicator of hospitalization and mortality among a variety of patients (71-74). A substantial amount of research is lacking on the use of HRQoL as a predictor of disease risk. Thus, it is of interest to contribute to the literature, generally, on HRQoL as a risk factor for disease risk, and specifically as a risk factor for unhealthy cholesterol, a well-established risk factor for CVD.

Quality of Life in African American women

Constant across different measures of HRQoL is the result that African Americans have lower self-reported HRQoL. In the Study of Women's Health Across the Nation, five of the eight SF-36 scale scores were assessed in a multiethnic sample of women. Unadjusted and multiple adjusted odds ratios indicated significant differences between African American and white women in the bodily pain, vitality, and social functioning scales (75).

Another study comprised of people over 65 years of age, the Chicago Health & Ageing Project, found similar differences. The HRQoL measure used in this study was made up of two questions used by the Centers for Disease Control and Prevention's Behavioral Risk Factor Surveillance System and the

National Health and Nutrition Examination Survey. The question asked each subject to indicate the number of days during the past 30 days that their physical and mental health was poor. African Americans had increased odds of reporting poor HRQoL compared with whites (OR = 1.72; 95% CI: 1.50 - 1.98) when adjusting for sex and age (76).

Lastly, a recent study conducted using data from the National Health Measurement Study demonstrated that African American women consistently reported considerably lower HRQoL than white women. This study used five different measures of HRQoL: EuroQol EQ-5D, Health Utilities Index Mark 2, Health Utilities Index Mark 3, Quality of Well-Being Scale, and SF-6D. Furthermore, the authors found that much of the difference in HRQoL can be explained by socioeconomic and sociodemographic factors (77).

Summary

As illustrated by the information included in this literature review, there exists a need for a CVD risk assessment tool that is easily accessible to African American women despite barriers that they face. By increasing CVD risk awareness among the female African American community and reducing the prevalence, severity, and number of CVD risk factors in these women, a decrease in mortality due to CVD in this high risk group may follow. Thus, risk awareness and risk factor management in African American women may directly contribute to a decrease in the overall burden of CVD in women. This thesis posits that the

SF-36v2 may be an appropriate measure of CVD risk, as assessed by unhealthy levels of HDL, LDL, and total cholesterol, in African American women because of its validity, reliability, and predictive nature of cardiovascular events.

Methods

Target Population and Sample Participants

The following analyses were conducted using data from the P.R.I.S.E.®: PREPS (preparedness through individual and group counseling), REPS (weight training), INCREASED STEPS (increasing physical activity through walking/steps), and ENCOURAGEMENT (social support mechanisms) study conducted between 2005 and 2007 by Emory University faculty. The objective of the P.R.I.S.E.® study was to test a sustainable intervention geared toward improving long-term physical activity among female African American healthcare workers employed through the Grady Health System in Atlanta, GA and at Meharry Medical College in Nashville, TN. The sites were chosen because of their demographic makeup; Grady Health System estimates suggest that 80% of their employees are African American and Meharry Medical College is recognized by the U.S. Department of Education as a Historically Black College. After screening for previous cardiovascular disease or other contraindications for a non-clinical physical activity intervention study, potentially eligible non-pregnant women of any race between the ages of 18 and 55 who performed poorly on a cardiac stress test or walked less than 10,000 steps per day without any medical history of heart problems were invited to participate in the P.R.I.S.E.® study.

In total, 239 participants were enrolled in the study. For the purpose of the following analyses, only Non-Hispanic African American women were included in the sample population (n=208). Analyses were conducted using only baseline data collected from both sites.

Procedure

All study protocols were approved by the respective Institutional Review Boards at Emory University and Meharry Medical College. Supervisor permission was required for participation of randomly selected departments within the Grady Health System and Meharry Medical College. After supervisory permission was obtained, study staff recruited participants by phone or during department-wide meetings. Interested participants were screened for eligibility by age and health criteria. Eligible employees were given a supervisor consent form and a study consent form, allowing the study staff to notify the supervisor of the subject's participation in the study and begin baseline testing, respectively.

After signed informed consent was obtained using standard procedures, premenopausal women were asked to provide a urine sample for the purpose of verifying reported pregnancy status. Self-reported history of disease information collected during eligibility screening was reconfirmed by the completion of a medical disease history questionnaire before initiation of baseline data collection. After the pregnancy test and disease history questionnaire were administered,

non-pregnant healthy women were asked to complete baseline tests and an in-depth questionnaire.

One-time baseline tests included completion of a standard cardiac stress test, calculation of anthropometrics (weight, height, hip, waist, thigh, calf, forearm, and arm measurements) using a calibrated scale, stadiometer, and a standard tape measure, blood pressure measurement, laboratory analysis of biomarkers of obesity and cardiovascular risk factors, twice in a single day salivary cortisol measurement, and a lipid and comprehensive metabolic panel using blood serum samples from each study participant. For consistency and accuracy, only trained staff members performed baseline tests and analyses.

The in-depth questionnaire was designed to obtain diet, physical activity, emotional state, medical history, and demographic data. Embedded within the questionnaire are the State, Trait and Anger Expression Inventory (STAXI-2) measure of current anger (state anger), anger expression, and disposition towards anger (trait anger), Pender Exercise Benefits/Barriers Scale (EBBS) measure of perceived benefits and barriers to physical activity, Jackson, Hogue, Phillips Contextualized Stress Measure (JHP) for perceived stress, Hardiness Index for transformational coping, Stressful Life Events Scale for stress assessment for the previous three months, Brief Symptom Inventory (BSI-18)® index for depression, Self-Efficacy for Exercise Behaviors Scale for exercise self-efficacy, Weight Efficacy Lifestyle (WEL) Questionnaire for weight management self-efficacy, and Short Form 36 Version 2 (SF-36v2) for quality of life

assessment. Participants anonymously completed the questionnaire in private rooms.

Measures

The cross-sectional data used for the following analyses were obtained during baseline data collection of the P.R.I.S.E.® study. Baseline data included responses to an extensive questionnaire, blood serum analyses, anthropometric measurement, salivary cortisol analysis, and blood pressure and heart rate measurement. The questionnaire collected data on diet and physical activity, demographics (i.e. race, age, place of birth, household size, marital status, highest degree earned, hours worked per week, income), disease history, and included the STAXI-2, EBBS, JHP Measure, Hardiness Index, Stressful Life Events Scale, BSI-18, Self-Efficacy for Exercise Behaviors Scale, WEL questionnaire, and SF-36v2.

SF-36™ Version 2

The SF-36v2 was used to measure HRQoL, the exposure of interest. This survey is a multi-purpose short-form that yields eight functional health and well-being scale scores, as well as psychometrically-based physical and mental health summary scores. The eight scale scores in the second version of the SF-36 are physical functioning, role-physical, bodily pain, general health, vitality, social functioning, role-emotional, and mental health. Reliability statistics, such as the

Cronbach's alphas calculated in this study, have exceeded 0.70 in more than 25 studies (78). The validity of this measure was demonstrated through the International Quality of Life Assessment Project in 1991(79). The SF-36v2 is a generic measure, appropriate for use in general and specific populations, that provides useful information on HRQoL as a result of a wide range of different treatments for many diseases and interventions. Few studies have investigated the use of the SF-36v2 as a predictor. Rodriguez-Artalejo et al. used the SF-36v2 in a survival analysis of HRQoL on time to hospital readmission and death among congestive heart failure patients (80). The hypothesis guiding this study is that higher SF-36v2 scores, which represent good health, will reflect lower risk of CVD in the three CVD risk factors of interest, LDL, HDL, and total cholesterol.

Demographics

During baseline testing, detailed demographic data were collected on each participant. This included self-reported race/ethnicity, birthplace, age, marital status, education, employment status (hours worked per week), household income, composition, and size, tobacco use, drinking habits, and use of vitamins or supplements. In a few instances when self-reported race and ethnicity were missing, race and ethnicity were classified by the interviewer. For the purpose of this study, only age, smoking status, current use of alcohol, education, income, employment status, birthplace, and marital status were treated as potential confounders or effect modifiers. Though socioeconomic

status explains much of the differential risk in CVD, race and ethnicity are still very important indicators. This point is illustrated by Boykin et al; their data show that the most advantaged African American women, those with higher socioeconomic status, had similar or worse risk profiles than the most disadvantaged white women, indicating a substantial gap between African American of lower socioeconomic status and their white counterparts (81). It is hypothesized that less favorable socioeconomic factors among our group of African American women will associate with lower HRQoL scores and higher risk of unhealthy levels of LDL, HDL, and total cholesterol.

Body Mass Index

A relationship between adiposity, as measured by body mass index, and quality of life is well established. Hopman et al. found a decrease in SF-36 scores as one's body mass index increased, especially in the physical functioning scale score (82). Body mass indices were estimated using the height and weight measurements obtained during baseline testing and derived from the standard weight in kilograms divided by height in meters-squared equation. It is hypothesized that a higher body mass index will correlate with lower SF-36 v2 scores.

Furthermore, previous research shows that body mass index is positively correlated with unhealthy cholesterol. This includes an increase in LDL and total cholesterol and a decrease in HDL (62). Given that body mass index is an

independent risk factor for the exposure, HRQoL, and the outcomes, LDL, HDL, and total cholesterol levels, it will be controlled for as a confounder in the following analyses.

Cardiovascular Risk Factors

Serum samples from all participants were used to determine biomarker levels for CVD. This includes adiponectin, PAI-1, TNF-α, IL-6, IL-10, C-reactive protein, LDL, HDL, and total cholesterol. Little evidence exists on the predictive nature of HRQoL in relation to CVD risk factors such as HDL, LDL, and total cholesterol, the outcomes of interest for these analyses. The main hypothesis of this study is that poor HRQoL will correlate with unhealthy levels of HDL, LDL, and total cholesterol, thus indicating an increased risk for CVD among study participants with low SF-36v2 scores.

Data Handling & Analysis

All data from the questionnaire, anthropometric, and lab results forms were entered using DATAFAX (83). This involved faxing data collection forms to a computer system that converted the raw information into statistical data. SAS® Version 9.2 was used for data cleaning and analysis (84). Preliminary descriptive analyses for all variables of interest were performed in order to check for outliers and missing values. All biologically implausible data were set to missing.

Of the 239 participants in the P.R.I.S.E.® study, only African American women were included in the sample population. In addition, two African American women that identified themselves as Hispanic were excluded from analyses. Furthermore, one woman with an underweight body mass index was removed from the study sample, leaving n=207 participants for analyses.

SF-36v2 scale and component summary scores were calculated using QualityMetric Health Outcomes™ Scoring Software Version 4.0 (85). Cronbach's alphas were calculated to demonstrate SF-36v2 reliability. Age was treated both as a continuous and multilevel categorical variable. Body mass index was treated as a continuous variable and categorized as normal (18.5 – 25.0 kg/m^2), overweight (25.0 – 30.0 kg/m^2), and obese (> 30.0 kg/m^2). Current smoking status, current alcohol use, site, and birthplace were treated as dichotomous categorical variables, while income, marital status, education, and employment status were initially multilevel categorical variables. HDL, LDL, and total cholesterol were initially treated as continuous variables.

Univariate analyses were conducted by running frequencies on age, body mass index, income, employment status, birthplace, marital status, smoking status, alcohol use, and education by study site. In addition, the central tendencies of the SF-36v2 scale and component summary scores were calculated using standard procedures in the SAS program. Lastly, the distribution of those with above median scale and component summary scores were determined by site. After these analyses, all remaining multilevel categorical and continuous

variables were made dichotomous. Age was dichotomized by the median, while HDL, LDL, and total cholesterol were dichotomized by American Heart Association standards, where unhealthy serum levels were < 50mg/dL, ≥ 160 mg/dL, and ≥ 240 respectively (61). Marital status was dichotomized as married or living with partner (married) vs. single, widowed, divorced, separated, or not living with partner (unmarried), education as high school or less vs. > high school, income as < $60,000 U.S. dollars (USD)/year vs. ≥ $60,000 USD/year, and employment status as part-time (<30 hours/week) vs. full-time (≥ 30 hours/week).

Bivariate associations between the main outcomes (HDL, LDL, and total cholesterol) and the HRQoL physical and mental health component summary scores (PCS and MCS) were examined using Cochran-Mantel-Haenszel odds ratios. Stratified analyses were conducted, and Breslow-Day p-values were used to identify significant interaction terms. The correlations between independent variables and HRQoL scores were also examined and the final models were built based on the observed associations and theory; body mass index was determined to be a confounder *a priori*.

Multivariate models were tested using logistic regression with total cholesterol, LDL, and HDL as the outcome variables. Body mass index, other potential confounders, and the two exposure variables were in the full model for each outcome. Forward regression techniques were used to construct the reduced model. The final models for each outcome included body mass index,

PCS, MCS, and variables that remained significant at $p < 0.20$. The two exposure variables and body mass index were forced back into the model if eliminated.

Results

Table 1 lists the univariate results comparing women at both study sites for general characteristics. Significant differences in age, birthplace, and education were observed between the two study sites (p = 0.0201, 0.015, and 0.0041 respectively). As indicated, the majority of participants were obese, between 30 and 49 years of age, full-time employees, unmarried, born in the United States, non-smokers, use alcohol, and had higher than a high school education.

The means, medians, standard deviations, and number of observations missing for SF-36v2 scale and component summary scores are described in Table 2. The highest scale score was physical functioning (mean = 85.48) and the lowest was vitality (mean = 57.71). Cronbach's alphas were greater than 0.70 for every scale except bodily pain (data not shown).

As illustrated in Table 3, the proportion of participants with above median SF-36v2 scale and summary scores (exposed) did not differ by site. The physical functioning and role-physical scales had the largest proportion of participants with above median scores (48.3%). Bodily pain had the lowest proportion of women with above median scores (33.3%). The proportion of exposed participants was 48.8% and 49.3% in the PCS and MCS, respectively.

Table 4 shows the crude prevalence ratios for the exposures and the outcomes. There was no statistically significant difference between those with above median and less than or equal to median PCS or MCS with respect to LDL, HDL, and total cholesterol. Each 95% confidence interval contained the null value of 1.00.

Breslow-Day p-values were calculated for the association between both exposures, independently, and each outcome, independently, controlling for potential confounders. In cases where 0 was a cell value, 0.5 was added to each cell and the Logit aOR was calculated. The Brewslow-Day test for intrastratum homogeneity did not identify any effect modifiers; all Breslow-Day p-values were greater than 0.05. These data are shown in Table 5 in the appendix.

Independent variables were examined for statistically significant associations with all exposures and outcomes. There was a significant association between alcohol use and above median MCS (PR = 0.74; 95% CI: 0.56 - 0.97), education and high risk HDL levels (PR = 1.49; 95% CI: 1.09 – 2.03), and marital status and high risk total cholesterol levels (PR = 0.06; 95% CI: 0.00 – 0.99). No independent variables were found to have a statistically significant relationship with both an exposure and outcome. Even so, body mass index was controlled for in all models because of its documented association with both quality of life and cholesterol.

Table 7 shows collinearity diagnostics for the full models for each outcome. The criterion for collinearity was a conditional index greater than 30

accompanied by at least two variance decomposition proportions greater than 0.5. No collinearity issues were identified in any of the full models.

Tables 8 - 10 describe the characteristics of the full and reduced models for each outcome. The criterion for inclusion in the forward regression was a p-value less than 0.2. For the LDL model, only alcohol use and education were significant predictors (p = 0.042 and 0.1801, respectively); PCS, MCS, and body mass index were forced back into the model (Table 8). Education, marital status, employment status, and smoking status were significant predictors of high risk LDL levels (p = 0.0025, 0.0203, 0.0526, and 0.2063, respectively). As in the LDL model, PCS, MCS, and body mass index were forced into the model after initial elimination (Table 9). Marital status, employment status, education, and birthplace were significant predictors of high risk total cholesterol levels (p = 0.0044, 0.0603, 0.0533, and 0.1157, respectively) at alpha=0.20 (Table 10). PCS, MCS, and body mass index were also forced back into the total cholesterol model.

The reduced models from tables 8 - 10 were used to calculate adjusted odds ratios for the association between PCS and the three outcomes, as well as MCS and the outcomes of interest. Table 11 demonstrates these findings alongside the crude prevalence ratios from Table 4. Each of the six adjusted odds ratios were statistically insignificant; the null value of 1.00 was included in every 95% confidence interval.

Discussion

This study attempted to provide evidence for the use of the SF-36v2 as a predictor of the following cardiovascular risk factors: LDL, HDL, and total cholesterol. Results from this study did not support the hypothesis that higher HRQoL scores would have a protective effect on high risk cholesterol levels.

1) *Does higher health-related quality of life, as determined by the SF-36v2, correlate with less cardiovascular disease risk when controlling for Body Mass Index?*

   The data in the present study do not appear to support this hypothesis at a statistically significant level. The null value of 1.00 was included in each of the 95% confidence intervals for the adjusted odds ratios for both exposures and each of the outcomes of interest.

2) *What model best predicts the relationship between heath-related quality of life and risk factors (i.e. LDL, HDL, and total cholesterol) for cardiovascular disease?*

   Interestingly, the best models for predicting high risk LDL, HDL, and total cholesterol did not include the PCS, MCS, or body mass index. The best predictors for cardiovascular risk attributable to cholesterol levels were socioeconomic, lifestyle, and sociodemographics factors such as marital status, employment status, education, alcohol use, smoking status, and birthplace. The two exposures and *a priori* confounder had to be forced back into the reduced models for adjusted odds ratio ascertainment.

Strengths & Limitations

A major strength of this study is its contribution to the literature on HRQoL. The use of the SF-36v2, and HRQoL in general, as a predictor of disease risk is greatly under researched. Limitations of this study include *a priori* assumptions about study participants, reliability of scale scores, potential for bias, and limited external validity.

As mentioned above, the study participants were assumed to have the literacy and capacity to interpret each of the 36 items on the SF-36v2 and their accompanying Likert scale. The Cronbach's α for the bodily pain scale score did not demonstrate sufficient reliability, affecting the resulting PCS score. Furthermore, the questionnaire relied on self-reported information that is vulnerable to recall bias. This leads to the potential for exposure misclassification, biasing our results in either direction. Additionally, inaccuracy in laboratory measurement of the outcomes could also lead to bias in either direction. Lastly, the external validity of this study is limited because of the focus on such a specific population. These results, though insignificant, are not appropriate for application to other groups of women or the general population.

Future Research

Further investigation of the relationship between SF-36v2 scores and cholesterol levels is needed before a definitive conclusion can be reached

regarding a lack of association between the two. Similarly, future studies should investigate other CVD risk factors such as hypertension and biomarkers of atherosclerosis. Investigating risk factors for other diseases would also benefit HRQoL literature. Replication studies in other groups are also needed to establish the validity of these results to broader populations. Finally, longitudinal studies of SF-36v2 scores and CVD incidence could determine the predictive capability of this HRQoL measure, independent of known CVD risk factors.

Conclusions

The data present in this study are inadequate for providing a definitive conclusion on the relationship between SF-36v2 scores and serum levels of LDL, HDL, and total cholesterol when controlling for body mass index. Further investigation of this association in both similar and different populations is needed in order to draw any conclusions. Use of the SF-36v2 as a predictor of cholesterol risk is not supported by the data in this study, yet investigation of its use for evaluating other CVD risk factors is still warranted.

References

1. MedlinePlus. Heart Disease in Women. Bethesda: National Institutes of Health; 2011. (http://www.nlm.nih.gov/medlineplus/heartdiseaseinwomen.html). (Accessed April 10 2011).
2. American Heart Association. Go Red for Women: About the Movement. Dallas: American Heart Association; 2011. (http://www.goredforwomen.org/about_the_movement.aspx). (Accessed April 10 2011).
3. National Center for Health Statistics. Health, United States, 2007 With Chartbook on Trends in the Health of Americans. Hyattsville: National Center for Health Statistics, 2008, (Department of Health and Human Services)
4. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart disease and stroke statistics--2011 update: a report from the American Heart Association. *Circulation* 2011;123(4):e18-e209.
5. Williams RA, Gavin JR, 3rd, Phillips RA, et al. High-risk African Americans with multiple risk factors for cardiovascular disease: challenges in prevention, diagnosis, and treatment. *Ethn Dis* 2006;16(3):633-9.
6. Mensah GA, Mokdad AH, Ford ES, et al. State of disparities in cardiovascular health in the United States. *Circulation* 2005;111(10):1233-41.
7. Cooney MT, Dudina AL, Graham IM. Value and limitations of existing scores for the assessment of cardiovascular risk: a review for clinicians. *J Am Coll Cardiol* 2009;54(14):1209-27.
8. Gaziano TA, Young CR, Fitzmaurice G, et al. Laboratory-based versus non-laboratory-based method for assessment of cardiovascular disease risk: the NHANES I Follow-up Study cohort. *Lancet* 2008;371(9616):923-31.
9. National Center for Health Statistics. Health, United States, 2007 With Chartbook on Trends in the Health of Americans. Hyattsville: National Center for Health Statistics, 2007, (Department of Health and Human Services)
10. Yusuf S, Reddy S, Ounpuu S, et al. Global burden of cardiovascular diseases: part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization. *Circulation* 2001;104(22):2746-53.
11. American Hearth Association. Heart Disease and Stroke Statistics -2003 Update [electronic article].

12. Bello N, Mosca L. Epidemiology of coronary heart disease in women. *Prog Cardiovasc Dis* 2004;46(4):287-95.
13. Goldberg RJ, O'Donnell C, Yarzebski J, et al. Sex differences in symptom presentation associated with acute myocardial infarction: a population-based perspective. *Am Heart J* 1998;136(2):189-95.
14. Penque S, Halm M, Smith M, et al. Women and coronary disease: relationship between descriptors of signs and symptoms and diagnostic and treatment course. *Am J Crit Care* 1998;7(3):175-82.
15. Wenger NK. Clinical characteristics of coronary heart disease in women: emphasis on gender differences. *Cardiovasc Res* 2002;53(3):558-67.
16. Milner KA, Funk M, Richards S, et al. Gender differences in symptom presentation associated with coronary heart disease. *Am J Cardiol* 1999;84(4):396-9.
17. Lerner DJ, Kannel WB. Patterns of coronary heart disease morbidity and mortality in the sexes: a 26-year follow-up of the Framingham population. *Am Heart J* 1986;111(2):383-90.
18. Hopkins PN, Williams RR. A survey of 246 suggested coronary risk factors. *Atherosclerosis* 1981;40(1):1-52.
19. Sauer WH, Berlin JA, Strom BL, et al. Cigarette yield and the risk of myocardial infarction in smokers. *Arch Intern Med* 2002;162(3):300-6.
20. Plasma lipid distributions in selected North American populations: the Lipid Research Clinics Program Prevalence Study. The Lipid Research Clinics Program Epidemiology Committee. *Circulation* 1979;60(2):427-39.
21. Mosca L. Novel cardiovascular risk factors: do they add value to your practice? *Am Fam Physician* 2003;67(2):264, 6.
22. Legato MJ, Padus E, Slaughter E. Women's perceptions of their general health, with special reference to their risk of coronary artery disease: results of a national telephone survey. *J Womens Health* 1997;6(2):189-98.
23. Mosca L, Jones WK, King KB, et al. Awareness, perception, and knowledge of heart disease risk and prevention among women in the United States. American Heart Association Women's Heart Disease and Stroke Campaign Task Force. *Arch Fam Med* 2000;9(6):506-15.
24. Centers for Disease Control and Prevention. Missed Opportunities in Preventive Counseling for Cardiovascular Disease - United States 1995. *MMWR Morb Mortal Wkly Rep* 1998;47:91-5.
25. Gettleman L, Winkleby MA. Using focus groups to develop a heart disease prevention program for ethnically diverse, low-income women. *J Community Health* 2000;25(6):439-53.
26. Mosca L, McGillen C, Rubenfire M. Gender differences in barriers to lifestyle change for cardiovascular disease prevention. *J Womens Health* 1998;7(6):711-5.
27. Baturka N, Hornsby PP, Schorling JB. Clinical implications of body image among rural African-American women. *J Gen Intern Med* 2000;15(4):235-41.

28. Gerhard GT, Sexton G, Malinow MR, et al. Premenopausal black women have more risk factors for coronary heart disease than white women. *Am J Cardiol* 1998;82(9):1040-5.
29. Sundquist J, Winkleby MA, Pudaric S. Cardiovascular disease risk factors among older black, Mexican-American, and white women and men: an analysis of NHANES III, 1988-1994. Third National Health and Nutrition Examination Survey. *J Am Geriatr Soc* 2001;49(2):109-16.
30. Winkleby MA, Kraemer HC, Ahn DK, et al. Ethnic and socioeconomic differences in cardiovascular disease risk factors: findings for women from the Third National Health and Nutrition Examination Survey, 1988-1994. *JAMA* 1998;280(4):356-62.
31. Mosca L, Mochari-Greenberger H, Dolor RJ, et al. Twelve-year follow-up of American women's awareness of cardiovascular disease risk and barriers to heart health. *Circ Cardiovasc Qual Outcomes* 2010;3(2):120-7.
32. Lloyd-Jones D, Adams R, Carnethon M, et al. Heart disease and stroke statistics--2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation* 2009;119(3):480-6.
33. Zhang Y. Cardiovascular diseases in American women. *Nutr Metab Cardiovasc Dis* 2010;20(6):386-93.
34. Lee ET, Howard BV, Savage PJ, et al. Diabetes and impaired glucose tolerance in three American Indian populations aged 45-74 years. The Strong Heart Study. *Diabetes Care* 1995;18(5):599-610.
35. Hemingway H, Langenberg C, Damant J, et al. Prevalence of angina in women versus men: a systematic review and meta-analysis of international variations across 31 countries. *Circulation* 2008;117(12):1526-36.
36. Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension* 2003;42(6):1206-52.
37. O'Rourke RA. *Hurst's the Heart: Manual of Cardiology*. 12th ed.: Mc-Graw Hill; 2008.
38. Ford ES, Capewell S. Coronary heart disease mortality among young adults in the U.S. from 1980 through 2002: concealed leveling of mortality rates. *J Am Coll Cardiol* 2007;50(22):2128-32.
39. Colditz GA, Willett WC, Stampfer MJ, et al. Menopause and the risk of coronary heart disease in women. *N Engl J Med* 1987;316(18):1105-10.
40. Anderson GL, Limacher M, Assaf AR, et al. Effects of conjugated equine estrogen in postmenopausal women with hysterectomy: the Women's Health Initiative randomized controlled trial. *JAMA* 2004;291(14):1701-12.
41. Grady D, Herrington D, Bittner V, et al. Cardiovascular disease outcomes during 6.8 years of hormone therapy: Heart and Estrogen/progestin Replacement Study follow-up (HERS II). *JAMA* 2002;288(1):49-57.

42. Manson JE, Hsia J, Johnson KC, et al. Estrogen plus progestin and the risk of coronary heart disease. *N Engl J Med* 2003;349(6):523-34.
43. Barrett KM, Worrall BB. Sex and stroke: are they really different in midlife? *Neurology* 2007;69(20):1894-5.
44. Kittner SJ, Stern BJ, Feeser BR, et al. Pregnancy and the risk of stroke. *N Engl J Med* 1996;335(11):768-74.
45. Towfighi A, Saver JL, Engelhardt R, et al. A midlife stroke surge among women in the United States. *Neurology* 2007;69(20):1898-904.
46. Barker WH, Mullooly JP, Getchell W. Changing incidence and survival for heart failure in a well-defined older population, 1970-1974 and 1990-1994. *Circulation* 2006;113(6):799-805.
47. Rich MW. Heart failure in the 21st century: a cardiogeriatric syndrome. *J Gerontol A Biol Sci Med Sci* 2001;56(2):M88-96.
48. Loehr LR, Rosamond WD, Chang PP, et al. Heart failure incidence and survival (from the Atherosclerosis Risk in Communities study). *Am J Cardiol* 2008;101(7):1016-22.
49. Bibbins-Domingo K, Lin F, Vittinghoff E, et al. Predictors of heart failure among women with coronary disease. *Circulation* 2004;110(11):1424-30.
50. Wang L, Fan C, Topol SE, et al. Mutation of MEF2A in an inherited disorder with features of coronary artery disease. *Science* 2003;302(5650):1578-81.
51. Yamada Y, Izawa H, Ichihara S, et al. Prediction of the risk of myocardial infarction from polymorphisms in candidate genes. *N Engl J Med* 2002;347(24):1916-23.
52. Lichtenstein AH, Appel LJ, Brands M, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation* 2006;114(1):82-96.
53. Vasan RS. Biomarkers of cardiovascular disease: molecular basis and practical considerations. *Circulation* 2006;113(19):2335-62.
54. May A, Wang TJ. Biomarkers for cardiovascular disease: challenges and future directions. *Trends Mol Med* 2008;14(6):261-7.
55. Bush TL, Fried LP, Barrett-Connor E. Cholesterol, lipoproteins, and coronary heart disease in women. *Clin Chem* 1988;34(8B):B60-70.
56. Rifkind BM. High-density lipoprotein cholesterol and coronary artery disease: survey of the evidence. *Am J Cardiol* 1990;66(6):3A-6A.
57. Wilson PW. High-density lipoprotein, low-density lipoprotein and coronary artery disease. *Am J Cardiol* 1990;66(6):7A-10A.
58. Kannel WB. High-density lipoproteins: epidemiologic profile and risks of coronary artery disease. *Am J Cardiol* 1983;52(4):9B-12B.
59. The Lipid Research Clinics Coronary Primary Prevention Trial results. II. The relationship of reduction in incidence of coronary heart disease to cholesterol lowering. *JAMA* 1984;251(3):365-74.

60. The Lipid Research Clinics Coronary Primary Prevention Trial results. I. Reduction in incidence of coronary heart disease. *JAMA* 1984;251(3):351-64.
61. American Heart Association. What do My Cholesterol Levels Mean? [electronic article].
62. Harris-Hooker S, Sanford GL. Lipids, lipoproteins and coronary heart disease in minority populations. *Atherosclerosis* 1994;108 Suppl:S83-104.
63. Anderson KM, Castelli WP, Levy D. Cholesterol and mortality. 30 years of follow-up from the Framingham study. *JAMA* 1987;257(16):2176-80.
64. Sempos C, Fulwood R, Haines C, et al. The prevalence of high blood cholesterol levels among adults in the United States. *JAMA* 1989;262(1):45-52.
65. National Center for Health Statistics. Health, United States, 2008, With Chartbook. Hyattsville: National Center for Health Statistics, 2009, (Department of Health and Human Services)
66. McHorney CA. Health status assessment methods for adults: past accomplishments and future challenges. *Annu Rev Public Health* 1999;20:309-35.
67. Bergner M, Rothman ML. Health status measures: an overview and guide for selection. *Annu Rev Public Health* 1987;8:191-210.
68. Ware JE, Jr., Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med Care* 1992;30(6):473-83.
69. McHorney CA, Ware JE, Jr., Raczek AE. The MOS 36-Item Short-Form Health Survey (SF-36): II. Psychometric and clinical tests of validity in measuring physical and mental health constructs. *Med Care* 1993;31(3):247-63.
70. McHorney CA, Ware JE, Jr., Lu JF, et al. The MOS 36-item Short-Form Health Survey (SF-36): III. Tests of data quality, scaling assumptions, and reliability across diverse patient groups. *Med Care* 1994;32(1):40-66.
71. Konstam V, Salem D, Pouleur H, et al. Baseline quality of life as a predictor of mortality and hospitalization in 5,025 patients with congestive heart failure. SOLVD Investigations. Studies of Left Ventricular Dysfunction Investigators. *Am J Cardiol* 1996;78(8):890-5.
72. Mapes DL, Lopes AA, Satayathum S, et al. Health-related quality of life as a predictor of mortality and hospitalization: the Dialysis Outcomes and Practice Patterns Study (DOPPS). *Kidney Int* 2003;64(1):339-49.
73. Rumsfeld JS, MaWhinney S, McCarthy M, Jr., et al. Health-related quality of life as a predictor of mortality following coronary artery bypass graft surgery. Participants of the Department of Veterans Affairs Cooperative Study Group on Processes, Structures, and Outcomes of Care in Cardiac Surgery. *JAMA* 1999;281(14):1298-303.

74. Stull DE, Clough LA, Van Dussen D. Self-report quality of life as a predictor of hospitalization for patients with LV dysfunction: a life course approach. *Res Nurs Health* 2001;24(6):460-9.
75. Avis NE, Ory M, Matthews KA, et al. Health-related quality of life in a multiethnic sample of middle-aged women: Study of Women's Health Across the Nation (SWAN). *Med Care* 2003;41(11):1262-76.
76. Skarupski KA, de Leon CF, Bienias JL, et al. Black-white differences in health-related quality of life among older adults. *Qual Life Res* 2007;16(2):287-96.
77. Pereira CC, Palta M, Mullahy J, et al. Race and preference-based health-related quality of life measures in the United States. *Qual Life Res* 2010.
78. Tsai C, Bayliss, MS, Ware, JE SF-36® Health Survey Annotated Bibliography: Second Edition (1988-1996) [electronic article].
79. Ware JE, Jr., Gandek B. Overview of the SF-36 Health Survey and the International Quality of Life Assessment (IQOLA) Project. *J Clin Epidemiol* 1998;51(11):903-12.
80. Rodriguez-Artalejo F, Guallar-Castillon P, Pascual CR, et al. Health-related quality of life as a predictor of hospital readmission and death among patients with heart failure. *Arch Intern Med* 2005;165(11):1274-9.
81. Boykin C, Zhang G, Chen YH, et al. Cucurbitacin IIa: a novel class of anti-cancer drug inducing non-reversible actin aggregation and inhibiting survivin independent of JAK2/STAT3 phosphorylation. *Br J Cancer* 2011;104(5):781-9.
82. Hopman WM, Berger C, Joseph L, et al. The association between body mass index and health-related quality of life: data from CaMos, a stratified population study. *Qual Life Res* 2007;16(10):1595-603.
83. Datafax. Montevallo: Datafax, 2005.
84. SAS. Cary: SAS Institute Inc.
85. QualityMetric Health Outcomes™ Scoring Software. Lincoln: QualityMetric Incorportated, 2010.

Appendix

Table 1. Characteristics of Female African American P.R.I.S.E.® Study Participants[a]

Variable	Total Participants (n=207)		Grady (n=152)		Meharry (n=55)		p-value[b]
	No.	%	No.	%	No.	%	
Age, years							
20-29	42	20.3	33	21.7	9	16.4	
30-39	69	33.3	55	36.2	14	25.5	
40-49	78	37.7	56	36.8	22	40.0	
50+	18	8.7	8	5.3	10	18.2	0.0201*
BMI Category, kg/m2							
Normal	21	10.1	17	11.2	4	7.3	
Overweight	61	29.5	44	28.9	17	30.9	
Obese	125	60.4	91	59.9	34	61.8	0.7092*
Income, USD							
< $60,000	124	59.9	91	59.9	33	60.0	
$60,000 - $100,000	47	22.7	36	23.7	11	20.0	
> $100,000	19	9.2	12	7.9	7	12.7	
Missing	17	8.2	13	8.6	4	7.3	0.7174*
Hours Worked Per Week							
< 10	1	0.5	1	0.7	0	0.0	
10 - 20	2	1.0	2	1.3	0	0.0	
21 - 30	4	1.9	3	2.0	1	1.8	
31 - 40	91	44.0	73	48.0	18	32.7	
> 40	108	52.2	73	48.0	35	63.6	
Missing	1	0.5	0	0.0	1	1.8	0.1364
Birthplace							
United States	180	87.0	127	83.6	53	96.4	
Other	24	11.6	23	15.1	1	1.8	
Missing	3	1.4	2	1.3	1	1.8	0.0105
Marital Status							
Married/Living With Partner	87	42.0	68	44.7	19	34.5	
Separated/Widowed/Divorced	46	22.2	31	20.4	15	27.3	
Single/Never Married	73	35.3	52	34.2	21	38.2	
Missing	1	0.5	1	0.7	0	0.0	0.5230
Current Smoker							
Yes	9	4.3	6	3.9	3	5.5	
No	197	95.2	146	96.1	51	92.7	
Missing	1	0.5	0	0.0	1	1.8	0.2714
Currently Drink Alcohol							
Yes	141	68.1	104	68.4	37	67.3	
No	64	30.9	47	30.9	17	30.9	
Missing	2	1.0	1	0.7	1	1.8	0.6746
Highest Degree Earned							
Less than HS/HS equivalent	40	19.3	32	21.1	8	14.5	
Some College/College	113	54.6	90	59.2	23	41.8	
Graduate/Professional/Other	53	25.6	29	19.1	24	43.6	
Missing	1	0.5	1	0.7	0	0.0	0.0041

[a]Participants met age and health eligibility criteria for the 2004-2007 P.R.I.S.E.® Study conducted by Emory University and Meharry Medical College

[b]p-values were calculated using Fisher's Exact Test, unless indicated by an asterisk

*Calculated using chi-square test

Table 2. Central Tendencies of SF-36v2 Scale and Component Summary Scores for Female African American P.R.I.S.E.® Study Participants, n=207[a, b]

SF-36 Scale	Mean	Median	Standard Deviation	n=missing
Physical Functioning	*85.48*	*90.00*	*18.45*	*2*
Role-Physical	*84.27*	*93.75*	*21.59*	*3*
Bodily Pain	*75.29*	*84.00*	*22.97*	*1*
General Health	*72.71*	*77.00*	*17.17*	*0*
Vitality	*57.71*	*56.25*	*17.44*	*1*
Social Functioning	*82.04*	*87.50*	*21.75*	*1*
Role-Emotional	*83.05*	*91.67*	*22.95*	*2*
Mental Health	*75.79*	*80.00*	*16.84*	*1*
Physical Component	*51.76*	*53.02*	*6.65*	*4*
Mental Health Component	*48.57*	*51.10*	*10.16*	*3*

[a]Participants met age and health eligibility criteria for the 2004-2007 P.R.I.S.E.® study conducted by Emory University and Meharry Medical College

[b]SF-36v2 scale and component summary score means, medians, and standard deviations were calculated by standard procedures

Table 3. Proportion of Participant SF-36v2 Scale and Summary Scores Above the Median for Female African American P.R.I.S.E.® Study Participants[a]

SF-36 Scale Total & by Site	No.	% Above Median Score	p-value[b]
Physical Functioning			
Total (n=207)	***100***	***48.3***	
Grady (n=152)	*70*	*46.1*	
Meharry (n=55)	*30*	*54.5*	***0.3173***
Role-Physical			
Total	***100***	***48.3***	
Grady	*71*	*46.7*	
Meharry	*29*	*52.7*	***0.4220***
Bodily Pain			
Total	***69***	***33.3***	
Grady	*47*	*30.9*	
Meharry	*22*	*40.0*	***0.2325***
General Health			
Total	***85***	***41.1***	
Grady	*63*	*41.4*	
Meharry	*22*	*40.0*	***0.8517***
Vitality			
Total	***93***	***44.9***	
Grady	*68*	*44.7*	
Meharry	*25*	*45.5*	***0.9571***
Social Functioning			
Total	***99***	***47.8***	
Grady	*70*	*46.1*	
Meharry	*29*	*52.7*	***0.4182***
Role-Emotional			
Total	***99***	***47.8***	
Grady	*69*	*45.4*	
Meharry	*30*	*54.5*	***0.2133***
Mental Health			
Total	***88***	***42.5***	
Grady	*59*	*38.8*	
Meharry	*29*	*52.7*	***0.0797***
Physical Component			
Total	***101***	***48.8***	
Grady	*71*	*46.7*	
Meharry	*30*	*54.5*	***0.3196***
Mental Health Component			
Total	***102***	***49.3***	
Grady	*71*	*46.7*	
Meharry	*31*	*56.4*	***0.2042***

[a]Participants met age and health eligibility criteria for the 2004-2007 P.R.I.S.E.® Study conducted by Emory University and Meharry Medical College

[b]p-values were calculated using chi-square test

Table 4. Crude Prevalence Ratios for Exposures (PCS & MCS) and Outcomes (LDL, HDL, & Total Cholesterol)

Above Median SF-36 Component Summary Scores	LDL		
	Unhealthy	Healthy	PR (95% CI)
Physical Component	1 (0.49%)	100 (49.26%)	0.51 (0.05 - 5.48)
Mental Health Component	1 (0.49%)	101 (49.50%)	0.50 (0.05 - 5.43)

Above Median SF-36 Component Summary Scores	HDL		
	Unhealthy	Healthy	PR (95% CI)
Physical Component	45 (22.17%)	56 (27.59%)	0.97 (0.71 - 1.31)
Mental Health Component	45 (22.55%)	56 (27.45%)	1.00 (0.74 - 1.35)

Above Median SF-36 Component Summary Scores	Total Cholesterol		
	Unhealthy	Healthy	PR (95% CI)
Physical Component	3 (1.48%)	98 (48.28%)	1.51 (0.26 - 8.87)
Mental Health Component	2 (0.98%)	100 (49.02%)	0.67 (0.11 - 3.91)

Table 5. Adjusted Odds Ratios for the Assocation Between Exposures (PCS & MCS) and Outcomes (LDL, HDL, & Total Cholesterol) Controlled for Potential Confounders

Adjusted Odds Ratios for the Association Between SF-36v2 Physical Component Score and LDL Controlled for Potential Confounders

Crude OR = 0.51 (0.05 - 5.48)

Variable	aOR	95% CI	Breslow-Day P-Value
Age	0.46	0.04 - 5.65	0.46
Education	0.64	0.07 - 5.89	0.30
Income	0.50	0.05 - 4.83	0.32
Current Alcohol Use	0.57	0.06 - 5.61	0.34
Current Smoking Status	0.51*	0.05 - 5.48	.
Body Mass Index	0.49*	0.05 - 5.30	.
Birthplace	0.49	0.04 - 5.41	0.38
Marital Status	0.49	0.05 - 5.40	0.39
Employment Status	0.49*	0.05 - 5.37	.
Mental Health Component	0.51	0.05 - 4.80	0.06

Adjusted Odds Ratios for the Association Between SF-36v2 Mental Health Component Score and LDL Controlled for Potential Confounders

Crude OR = 0.50 (0.05 - 5.43)

Variable	aOR	95% CI	Breslow-Day P-Value
Age	0.55	0.06 - 5.16	0.30
Education	0.43	0.04 - 4.87	0.43
Income	0.51	0.04 - 6.23	0.46
Current Alcohol Use	0.38	0.03 - 4.89	0.50
Current Smoking Status	0.50*	0.05 - 5.42	.
Body Mass Index	0.54*	0.05 - 5.85	.
Birthplace	0.55	0.04 - 7.41	0.52
Marital Status	0.48	0.04 - 5.35	0.40
Employment Status	0.48*	0.04 - 5.21	.
Physical Component	0.51	0.05 - 4.80	0.06

Adjusted Odds Ratios for the Association Between SF-36v2 Physical Component Score and HDL Controlled for Potential Confounders

Crude OR = 0.97 (0.71 - 1.31)

Variable	aOR	95% CI	Breslow-Day P-Value
Age	0.95	0.71 - 1.29	0.19
Education	1.02	0.75 - 1.37	0.35
Income	0.96	0.69 - 1.32	0.26
Current Alcohol Use	0.97	0.72 - 1.32	0.41
Current Smoking Status	0.95	0.70 - 1.29	0.81
Body Mass Index	0.96	0.71 - 1.30	0.37
Birthplace	0.98	0.72 - 1.33	0.63
Marital Status	0.98	0.72 - 1.32	0.39
Employment Status	0.97	0.72 - 1.31	0.20
Mental Health Component	0.97	0.71 - 1.31	0.99

Adjusted Odds Ratios for the Association Between SF-36v2 Mental Health Component Score and HDL Controlled for Potential Confounders

Crude OR = 1.00 (0.74 - 1.35)

Variable	aOR	95% CI	Breslow-Day P-Value
Age	1.02	0.75 - 1.39	0.30
Education	0.99	0.74 - 1.34	0.86
Income	0.96	0.70 - 1.31	0.43
Current Alcohol Use	1.01	0.75 - 1.37	0.47
Current Smoking Status	1.02	0.75 - 1.38	0.07
Body Mass Index	1.02	0.75 - 1.38	0.76
Birthplace	0.99	0.73 - 1.35	0.75
Marital Status	1.01	0.74 - 1.36	0.95
Employment Status	0.95	0.70 - 1.29	0.67
Physical Component	1.01	0.74 - 1.36	0.99

Adjusted Odds Ratios for the Association Between SF-36v2 Physical Component Score and Total Cholesterol Controlled for Potential Confounders

Crude OR = 1.51 (0.26 - 8.87)

Variable	aOR	95% CI	Breslow-Day P-Value
Age	1.67	0.25 - 11.11	0.19
Education	1.60	0.31 - 8.26	0.07
Income	1.40	0.25 - 7.95	0.56
Current Alcohol Use	1.56	0.26 - 9.28	0.82
Current Smoking Status	1.52*	0.26 - 8.87	.
Body Mass Index	1.59	0.25 - 10.05	0.22
Birthplace	1.48	0.26 - 8.54	0.72
Marital Status	1.47*	0.26 - 8.33	.
Employment Status	1.60	0.25 - 10.13	0.20
Mental Health Component	0.97	0.71 - 1.31	0.99

Adjusted Odds Ratios for the Association Between SF-36v2 Mental Health Component Score and Total Cholesterol Controlled for Potential Confounders

Crude OR = 0.67 (0.11 - 3.91)

Variable	aOR	95% CI	Breslow-Day P-Value
Age	0.63	0.11 - 3.52	0.52
Education	0.62	0.11 - 3.68	0.81
Income	0.72	0.13 - 4.06	0.53
Current Alcohol Use	0.64	0.1 - 3.82	0.98
Current Smoking Status	0.67*	0.11 - 3.90	.
Body Mass Index	0.64	0.12 - 3.32	0.20
Birthplace	0.80	0.11 - 5.90	0.24
Marital Status	0.64*	0.11 - 3.62	.
Employment Status	0.84	0.12 - 5.78	0.67
Physical Component	0.70	0.13 - 3.84	0.10

*Logit OR calculated by adding 0.5 to each cell

Table 6. Crude Prevalence Ratios for Assocations between Potential Confounders and Exposures (PCS & MCS) and Outcomes (LDL, HDL, & Total Cholesterol)

Demographic Characteristics	Physical Component	Mental Health Component
	PR (95% CI)	
Age	0.81 (0.61 - 1.08)	1.27 (0.96 - 1.66)
Education	0.78 (0.51 - 1.18)	1.13 (0.81 - 1.56)
Income	0.84 (0.64 - 1.12)	1.26 (0.90 - 1.76)
Current Alcohol Use	1.13 (0.83 - 1.58)	0.74 (0.56 - 0.97)
Current Smoking Status	0.89 (0.42 - 1.88)	1.11 (0.61 - 2.03)
Body Mass Index	1.18 (0.71 - 1.97)	0.72 (0.51 - 1.01)
Birthplace	0.99 (0.65 - 1.51)	0.63 (0.35 - 1.14)
Marital Status	0.98 (0.74 - 1.29)	0.97 (0.74 - 1.28)
Employment Status	1.17 (0.49 - 2.79)	3.57 (0.58 - 22.04)

Demographic Characteristics	LDL	HDL	Total Cholesterol
	PR (95% CI)		
Age	1.16 (0.17 - 8.05)	0.85 (0.62 - 1.16)	2.31 (0.43 - 12.35)
Education	4.15 (0.60 - 28.57)	1.49 (1.09 - 2.03)	2.08 (0.39 - 10.93)
Income	0.53 (0.08 - 3.69)	1.01 (0.72 - 1.41)	0.53 (0.11 - 2.56)
Current Alcohol Use	0.45 (0.07 - 3.15)	0.94 (0.68 - 1.29)	0.91 (0.17 - 4.83)
Current Smoking Status	2.20 (0.13 - 38.10)	0.49 (0.14 - 1.69)	1.52 (0.09 - 25.18)*
Body Mass Index	1.06 (0.06 - 19.02)*	1.37 (0.73 - 2.56)	0.56 (0.07 - 4.61)
Birthplace	2.50 (0.27 - 23.08)	1.04 (0.66 - 1.66)	3.75 (0.73 - 19.39)
Marital Status	0.24 (0.03 - 2.30)	0.75 (0.55 - 1.01)	0.06 (0.00 - 0.99)*
Employment Status	0.36 (0.02 - 6.13)	3.17 (0.51 - 19.56)	0.18 (0.02 - 1.31)

Statistically significant

* Logit OR calculated by adding 0.5 to each cell

Table 7. Collinearity Assessment for LDL, HDL, and Total Cholesterol

A. LDL

VARIABLE	VDP1	VDP2	VDP3	VDP4	VDP5	VDP6	VDP7	VDP8	VDP9	VDP10	VDP11	VDP12
EIGENVAL	0	0	0.0729	0.1294	0.2686	0.3224	0.5413	0.7269	0.8853	0.9997	1.0136	7.04
CONDINDX	3157.6	1464.9	9.8264	7.3759	5.12	4.673	3.6065	3.1122	2.8199	2.6538	2.6354	1
Intercept	0.97	0.03	0	0	0	0	0	0	0	0	0	0
PCS	0	0	0.0089	0.6202	0.0054	0.0316	0.0286	0.1051	0.0122	0.0048	0.1806	0.0025
MCS	0	0	0.0886	0.078	0.033	0.0181	0.5234	0.2017	8E-05	0.0017	0.0509	0.0046
Birthplace	0	0	0.0137	0.4584	0.0462	0.4587	0.0013	0.013	0.0044	1E-05	0.0006	0.0039
Smoking Status	0	0	0	0	0	0	6E-05	1E-05	0.0001	0.9736	0.0262	0
Alcohol Use	0	0	0.1156	0.0589	0.0026	0.1975	0.2091	0.3506	0.0078	0.0014	0.0524	0.0042
Age	0	0	3E-05	0.1914	0.0797	0.2878	0.0385	0.0003	0.3887	9E-05	0.0101	0.0035
Body Mass Index	0.13	0.87	0	0	0	0	0	0	0	0	0	0
Income	0	0	0.8176	0.05	0.1017	0.0051	0.0104	0.0016	0.0105	3E-05	0.0009	0.002
Empoyment Status	0.87	0.13	0	0	0	0	0	0	0	0	0	0
Marital Status	0	0	0.0111	0.4383	0.2384	0.0535	0.0915	0.0114	0.0549	0.0027	0.095	0.0032
Education	0	0	0.8317	0.0165	0.1328	0.0058	0.0003	7E-05	0.0094	5E-05	0.0013	0.002

B. HDL

VARIABLE	VDP1	VDP2	VDP3	VDP4	VDP5	VDP6	VDP7	VDP8	VDP9	VDP10	VDP11	VDP12
EIGENVAL	0.0085	0.0504	0.0815	0.1855	0.2286	0.3651	0.4265	0.5162	0.5962	0.8783	1.0194	7.6438
CONDINDX	30.027	12.311	9.6874	6.4193	5.7821	4.5754	4.2335	3.8482	3.5806	2.9501	2.7383	1
Intercept	0.9465	0.0458	0.0029	0.0029	0.0009	0.0006	2E-05	1E-05	0	0.0002	1E-05	0.0002
PCS	0.0149	0.0084	0.0321	0.0673	0.0047	0.1888	0.4858	0.0288	0.0867	0.0767	0.0018	0.0042
MCS	0.0019	0.0468	0.0288	0.0235	0.1975	0.0006	0.0952	0.3183	0.283	6E-05	6E-05	0.0042
Birthplace	0.0084	0.096	0.8349	0.0414	0.0015	0.0137	0.0013	0.0003	0	0.0009	1E-05	0.0015
Smoking Status	0.0045	0.0005	0.0066	0.0168	0.0004	0.0002	0.0189	0.0838	0.1094	0.0366	0.7215	0.0009
Alcohol Use	0.0411	0.0118	0.0364	0.4127	0.221	0.1973	0.0306	0.0013	0.0374	0.0072	1E-05	0.0033
Age	0.0339	0.0041	0.026	0.2106	0.0068	0.0332	0.0115	0.3707	0.2513	0.0008	0.0472	0.0039
Body Mass Index	0.0227	0.6733	0.2307	0.0458	0.0198	0.004	3E-05	0.0017	1E-05	0.0007	5E-05	0.0012
Income	0.0504	0.0002	0.005	0.3483	0.4484	0.0382	0.0262	0.0562	0.0064	0.0121	0.0052	0.0032
Empoyment Status	0.8601	0.123	0.0061	0.0086	0.0009	0.0008	1E-05	0	0	0.0002	1E-05	0.0003
Marital Status	0.005	0.0118	0.0031	0.0756	0.3437	0.3603	0.1668	0.0003	0.0172	0.0099	0.0025	0.004
Education	0.0001	0.0041	0.0009	0.0038	0.0367	0.0545	0.2375	0.0537	0.0121	0.5621	0.0313	0.003

C. Total Cholesterol

VARIABLE	VDP1	VDP2	VDP3	VDP4	VDP5	VDP6	VDP7	VDP8	VDP9	VDP10	VDP11	VDP12
EIGENVAL	0.0145	0.0721	0.1225	0.2364	0.3251	0.3882	0.5079	0.7287	0.8175	1	1	6.7872
CONDINDX	21.667	9.7034	7.4428	5.3587	4.5695	4.1813	3.6554	3.052	2.8814	2.6052	2.6052	1
Intercept	0.9849	0.0014	0.0084	3E-05	0.003	0.0008	0.0005	0.0007	0	0	0	0.0004
PCS	0.0285	0.1979	0.5572	0.0052	0.0093	0.0288	0.1407	0.0109	0.0184	0	0	0.0031
MCS	0.0057	0.0881	0.0031	0.5025	0.0347	0.0255	0.1754	0.056	0.1046	0	0	0.0046
Birthplace	0.0405	0.0499	0.0228	0.6224	0.0622	0.0695	0.1233	0.001	0.0039	0	0	0.0046
Smoking Status	0	1E-05	0	0	0	1E-05	0	1E-05	4E-05	0.1786	0.8213	0
Alcohol Use	0.2557	0.0043	0.068	0.1136	0.0476	0.389	0.0051	0.0814	0.0314	0	0	0.004
Age	0.2475	0.3063	0.1419	0.0005	0.0577	0.0789	0.0385	0.1079	0.0181	0	0	0.0027
Body Mass Index	0.0836	0.8687	2E-05	0.0024	0.0135	0.0162	0.0123	0.0003	0.0014	0	0	0.0018
Income	0.6441	0.001	0.0611	0.0636	0.1026	0.047	0.0002	3E-05	0.0785	0	0	0.0019
Empoyment Status	0.68	0.0821	0.2202	0.0038	0.0017	0.0032	0.0044	0.0014	0.0022	0	0	0.0011
Marital Status	0	1E-05	2E-05	2E-05	1E-05	0	1E-05	1E-05	2E-05	0.8213	0.1786	0
Education	0.3774	0.127	0.0053	0.0001	0.3449	0.0039	0.0087	0.0951	0.0346	0	0	0.003

Table 8. Logistic Regression Models for LDL

A. Full Model

Parameter Estimates						
Variable	Comparison	DF	Parameter Estimate	Standard Error	t Value	Pr > |t|
Intercept		1	0.0584	0.0680	0.86	0.3915
PCS	Above Median : Below Median	1	-0.0108	0.0196	-0.55	0.5822
MCS	Above Median : Below Median	1	-0.0135	0.0202	-0.67	0.5069
Birthplace	Outside of U.S. : U.S.	1	-0.0331	0.0319	-1.04	0.3013
Smoking Status	Yes : No	1	0.0031	0.0464	0.07	0.9468
Alcohol Use	Yes : No	1	-0.0254	0.0223	-1.14	0.2571
Age	Above Median : Below Median	1	-0.0101	0.0213	-0.48	0.6346
Body Mass Index	Overweight/Obese : Normal	1	0.0114	0.0335	0.34	0.7331
Income	<$60,000 : ≥$60,000	1	-0.0041	0.0231	-0.18	0.8580
Employment Status	Full-time : Part-time	1	0.0165	0.0565	0.29	0.7701
Marital Status	Married : Unmarried	1	-0.0207	0.0212	-0.98	0.3297
Education	≤High School : >High School	1	0.0573	0.0263	2.18	0.0309

B. Reduced Model

Parameter Estimates						
Variable	Comparison	DF	Parameter Estimate	Standard Error	t Value	Pr > |t|
Intercept		1	0.0276	0.0340	0.81	0.4173
PCS	Above Median : Below Median	1	-0.0068	0.0173	-0.39	0.6956
MCS	Above Median : Below Median	1	-0.0146	0.0175	-0.83	0.4051
Education	≤High School : >High School	1	0.0460	0.0223	2.07	0.0402
Alcohol Use	Yes : No	1	-0.0254	0.0189	-1.35	0.1801
Body Mass Index	Overweight/Obese : Normal	1	0.0080	0.0283	0.28	0.7785

Table 9. Logistic Regression Models for HDL

A. Full Model

Parameter Estimates						
Variable	Label	DF	Parameter Estimate	Standard Error	t Value	Pr > |t|
Intercept		1	0.1685	0.2591	0.65	0.5162
PCS	Above Median : Below Median	1	-0.0142	0.0748	-0.19	0.8502
MCS	Above Median : Below Median	1	-0.0059	0.0770	-0.08	0.9389
Birthplace	Outside of U.S. : U.S.	1	-0.0212	0.1216	-0.17	0.8616
Smoking Status	Yes : No	1	-0.1835	0.1766	-1.04	0.3002
Alcohol Use	Yes : No	1	0.0112	0.0849	0.13	0.8957
Age	Above Median : Below Median	1	-0.0525	0.0809	-0.65	0.5177
Body Mass Index	Overweight/Obese : Normal	1	0.1395	0.1275	1.09	0.2756
Income	<$60,000 : ≥$60,000	1	0.0010	0.0878	0.01	0.9913
Employment Status	Full-time : Part-time	1	0.2797	0.2150	1.30	0.1951
Marital Status	Married : Unmarried	1	-0.1820	0.0807	-2.25	0.0255
Education	≤High School : >High School	1	0.2482	0.1003	2.47	0.0143

B. Reduced Model

Parameter Estimates						
Variable	Comparison	DF	Parameter	Standard	t Value	Pr > |t|
Intercept		1	0.09867	0.20621	0.48	0.6328
PCS	Above Median : Below Median	1	-0.00208	0.06892	-0.03	0.9759
MCS	Above Median : Below Median	1	-0.01112	0.06989	-0.16	0.8737
Eduation	≤High School : >High School	1	0.27563	0.08985	3.07	0.0025
Marital Status	Married : Unmarried	1	-0.16433	0.07022	-2.34	0.0203
Employment Status	Full-time : Part-time	1	0.37166	0.19057	1.95	0.0526
Smoking Status	Yes : No	1	-0.20979	0.16546	-1.27	0.2064
Body Mass Index	Overweight/Obese : Normal	1	0.05368	0.11422	0.47	0.6389

Table 10. Logistic Regression Models for Total Cholesterol

A. Full Model

Parameter Estimates						
Variable	Comparison	DF	Parameter Estimate	Standard Error	t Value	Pr > |t|
Intercept		1	0.2259	0.0850	2.66	0.0087
PCS	Above Median : Below Median	1	0.0154	0.0245	0.63	0.5320
MCS	Above Median : Below Median	1	-0.0078	0.0253	-0.31	0.7580
Birthplace	Outside of U.S. : U.S.	1	-0.0604	0.0399	-1.51	0.1320
Smoking Status	Yes : No	1	-0.0217	0.0580	-0.37	0.7082
Alcohol Use	Yes : No	1	-0.0052	0.0279	-0.19	0.8534
Age	Above Median : Below Median	1	0.0269	0.0266	1.01	0.3133
Body Mass Index	Overweight/Obese : Normal	1	-0.0326	0.0419	-0.78	0.4375
Income	<$60,000 : ≥$60,000	1	0.0154	0.0288	0.53	0.5946
Employment Status	Full-time : Part-time	1	-0.1046	0.0706	-1.48	0.1401
Marital Status	Married : Unmarried	1	-0.0772	0.0265	-2.91	0.0040
Education	≤High School : >High School	1	0.0566	0.0329	1.72	0.0875

B. Reduced Model

Parameter Estimates						
Variable	Label	DF	Parameter	Standard	t Value	Pr > |t|
Intercept		1	0.2362	0.0714	3.31	0.0011
PCS	Above Median : Below Median	1	0.0115	0.0217	0.53	0.5968
MCS	Above Median : Below Median	1	-0.0038	0.0222	-0.17	0.8652
Marital Status	Married : Unmarried	1	-0.0642	0.0223	-2.88	0.0044
Employment Status	Full-time : Part-time	1	-0.1220	0.0646	-1.89	0.0603
Education	≤High School : >High School	1	0.0562	0.0289	1.94	0.0533
Birthplace	Outside of U.S. : U.S.	1	-0.0533	0.0338	-1.58	0.1157
Body Mass Index	Overweight/Obese : Normal	1	-0.0245	0.0361	-0.68	0.4976

Table 11. Crude Prevalence & Adjusted Odds Ratios for Exposures (PCS & MCS) and Outcomes (LDL, HDL, & Total Cholesterol)

Above Median SF-36 Component Summary Scores	**LDL**			
	Unhealthy	**Healthy**	**PR (95% CI)**	**aOR (95% CI)***
Physical Component	1 (0.49%)	100 (49.26%)	0.51 (0.05 - 5.48)	0.75 (0.06 - 9.45)
Mental Health Component	1 (0.49%)	101 (49.50%)	0.50 (0.05 - 5.43)	0.35 (0.03 - 4.53)

*Adjusted for education, current alcohol use, and body mass index

Above Median SF-36 Component Summary Scores	**HDL**			
	Unhealthy	**Healthy**	**PR (95% CI)**	**aOR (95% CI)***
Physical Component	45 (22.17%)	56 (27.59%)	0.97 (0.71 - 1.31)	1.00 (0.55 - 1.80)
Mental Health Component	45 (22.55%)	56 (27.45%)	1.00 (0.74 - 1.35)	0.94 (0.52 - 1.71)

*Adjusted for education, marital status, employment status, current smoking status, and body mass index

Above Median SF-36 Component Summary Scores	**Total Cholesterol**			
	Unhealthy	**Healthy**	**PR (95% CI)**	**aOR (95% CI)***
Physical Component	3 (1.48%)	98 (48.28%)	1.51 (0.26 - 8.87)	*1.68 (0.18 - 16.06)*
Mental Health Component	2 (0.98%)	100 (49.02%)	0.67 (0.11 - 3.91)	*1.13 (0.09 - 14.02)*

*Adjusted for marital status, employment status, education, birthplace, and body mass index