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Racial Disparities in Adverse Cardiovascular Outcomes After a Myocardial Infarction in Young or Middle-Aged Patients

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Racial Disparities in Adverse Cardiovascular Outcomes After a Myocardial Infarction in Young or Middle-Aged Patients

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An abstract of
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ABSTRACT

Background

Black patients tend to develop coronary artery disease at a younger age than other groups. Previous data on racial disparities in outcomes of myocardial infarction (MI) have been inconsistent and limited to older populations. Our objective was to investigate racial differences in the outcome of MI among young and middle-aged patients and the mediating role played by socioeconomic (SES), psychosocial and clinical differences.

Methods

We studied 313 participants (65% non-Hispanic Black) <61 years old hospitalized for confirmed type 1 MI at Emory-affiliated hospitals and followed them for 5 years. We used Kaplan Meier survival analysis and Cox proportional-hazard models to estimate the association of race with a composite endpoint of recurrent MI, stroke, heart failure or cardiovascular death after adjusting for demographic, SES, psychological and clinical risk factors.

Results

The mean age was 50 years and 50% were women. Compared with non-Black patients, Black patients had lower SES and more clinical and psychosocial risk factors, but less angiographic coronary artery disease. The 5-year incidence of cardiovascular events was higher in Black (35%) compared to non-Black patients (19%): hazards ratio (HR) 2.1, 95% confidence interval (CI), 1.3-3.6. Adjustment for SES weakened the association (HR 1.3, 95% CI, 0.8-2.4) more than adjustment for clinical and psychological risk factors.

Low income, which was defined as earning less than 35 thousand dollars a year, explained 46% of the race-related disparity in outcome.

Conclusion

Among young and middle-aged adult survivors of an MI, Black patients have a two-fold higher risk of adverse outcomes, which is largely driven by upstream socioeconomic factors, rather than downstream psychological and clinical risk factors. This suggests the need for greater emphasis on policy-level interventions, rather than biomedical or behavioral ones.

Abbreviations	
BMI	body mass index
CAD	coronary artery disease
HR	hazard ratio
MI	myocardial infarction
MIMS2	myocardial infarction and mental stress 2
NSTEMI	non-ST-elevation myocardial infarction
PTSD	post-traumatic stress disorder
SES	socioeconomic status
SD	standard deviation
STEMI	ST-elevation myocardial infarction

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INTRODUCTION

The Black population in the United States has worse cardiovascular health and higher rates of cardiovascular morbidity and mortality compared with other racial groups (1). Of further concern is the fact that downward trends in cardiovascular-related mortality in the past 4-5 decades have been less pronounced in Black individuals compared to other groups, leading to an increase in disparity over time (2,3). Understanding and eliminating such health inequalities has long been recognized as a national priority (4). Black adults in the United States overall have a more unfavorable cardiometabolic risk factor profile than their White counterparts, but whether these risk factors fully explain race-related disparities is controversial (5–7). Many studies have evaluated the contribution of low socioeconomic status (SES) to health inequalities by race across medical conditions and healthcare settings (5,8). However, disentangling the effect of race from that of SES has proven to be challenging. Nearly every indicator of SES is highly related to race, with US Black individuals bearing a disproportionate burden of poverty and other indicators of social disadvantage in comparison with Whites (9).

In this study we sought to investigate race differences in the outcome of MI among young and middle-aged survivors of MI, and the relative role played by SES, traditional risk factors and psychosocial factors. We were especially interested in the relative importance of SES versus other individual patient characteristics including psychosocial and traditional clinical risk factors in explaining racial disparities in MI outcomes. Our hypothesis was that a more adverse socioeconomic and psychosocial profile among Black patients would play a key role in explaining differences in the outcomes after MI between Black and non-Black patients.

BACKGROUND

Racial disparities in cardiovascular care in the United States have been well documented (10–14). Black patients with acute MI are less likely to receive guideline directed care before the event(15) or coronary revascularization procedures (16), and suffer worse outcomes when compared to their White counterparts (17). Some have suggested that race serves as a marker for SES and other health characteristics related to worse outcomes that are beyond the control of professionals involved in medical care (18–20). However, few studies have had sufficient numbers of Black participants and detailed socioeconomic and clinical information to evaluate the influence of both race and SES on myocardial infarction (MI) (21–26).

In a large study of hospitalized patients with MI, the excess mortality in Black patients compared to White patients was observed only among patients younger than 65 years of age, and differences in mortality by race diminished as age increased(This phenomenon, known as "racial crossover," has been reported before in population studies, and has been attributed to higher mortality among high-risk Black individuals who never reach the oldest ages (28–30). This earlier study, however, lacked information on SES, which has important applications on policy. Nonetheless, this study highlights the importance of examining young and middle-aged individuals when investigating health disparities by race. To date, few studies have focused on the young and middle-aged post-MI population to understand the reasons behind race-related differences in outcomes. Black individuals tend to be disadvantaged socioeconomically, but they also have more cardiometabolic risk factors and more psychosocial stressors compared with other groups (31). The relative importance of all these factors in explaining race differences in the risk of adverse outcomes among young patients with MI is currently unexplored.

In a study that sought to examine the degree to which nonrace characteristics explain observed survival differences between White and Black patients following acute MI, characteristics differed significantly between both groups. Characteristics such as socioeconomic and social factors, rather than race itself, were associated with an approximately 3-fold difference in 5-year mortality rate following acute MI and mediated most of the observed mortality rate difference between racial groups. However, this study included patients with no upper age limit with a mean age of 60 years, and most importantly did not present results in younger and older patients separately (32).

In a third study, investigators sought to examine the association between Black race and low SES with long-term outcomes of patients after acute MI. Post–MI life expectancy estimates were shorter for Black patients than for White patients across all SES levels in patients ≤75 years of age; yet was more pronounced in those younger than 68 years. Their sample was limited to patients aged between 65-90 years (6), and highlights the fact that younger Black patients are at a disproportionately higher risk after an MI.

METHODS

Study Aims

1. Investigate racial differences in the occurrence of cardiovascular events at 5 years in a population of young and middle-aged survivors of MI, using data from the Myocardial Infarction and Mental Stress 2 (MIMS2) study
2. Understand whether a more adverse socioeconomic and psychosocial profile explains any differences found in the outcomes after MI, between Black and non-Black patients

Study Design

Between August 2012 and March 2016 a total of 313 adult men and women were enrolled from the Myocardial Infarction and Mental Stress Study 2 (MIMS2), a prospective cohort study of patients 18 to 60 years of age with a documented history of MI in the previous eight months at Emory-affiliated hospitals in Atlanta, Georgia (33). MI case diagnosis (type 1) was verified with medical record review based on standard criteria of troponin elevation, symptoms of ischemia, and changes in the electrocardiogram or other evidence of myocardial necrosis (34). Exclusion criteria included unstable angina, acute coronary syndrome or decompensated heart failure in the previous week, severe comorbid medical or psychiatric disorder that could interfere with the study assessments, pregnancy or breastfeeding, or the use of immunosuppressant or psychotropic medications other than anti-depressants. Each participant underwent an assessment protocol that included a blood draw, measured height and weight, and clinic tests of myocardial perfusion imaging. A research nurse obtained sociodemographic, medical history and body measurements, and participants completed standardized questionnaires on behavioral, social, and health status information. After the baseline visit, patients were followed for 5 years for adverse events,

including cardiovascular death, recurrent (type 1) MI, stroke, and heart failure hospitalization. All events were independently adjudicated. The Emory University Institutional Review Board approved the protocol and all participants provided written informed consent.

Baseline Study Measures

Demographic and SES information included sex, race/ethnicity, age, educational attainment, employment status and income. Race/ethnicity was self-reported. Participants who self-reported as neither Black nor White, were few, thus they were grouped together as "non-Black." Educational attainment was assessed as years of education and dichotomized as <12 years or ≥12 years. Annual household income was categorized as <$35,000, $35,000 to $75,000, and >$75,000. Body mass index (BMI) was calculated as measured weight divided by the square of measured height (kg/m^2). History of cardiovascular risk factors was ascertained by chart review and by standardized questionnaires and included history of smoking, diabetes mellitus, hypertension, and dyslipidemia. Characteristics of the index MI were abstracted from the medical records and included type of MI (ST-elevation myocardial infarction [STEMI] vs. non-ST-elevation myocardial infarction [NSTEMI]), left ventricular ejection fraction, preventive medication use (eg, aspirin, beta blockers) and angiographic data, the latter obtained from the coronary angiogram associated with the index MI. CAD severity was quantified using the Gensini Score (35).

We obtained six scales of psychological characteristics with known association with cardiovascular disease or prognosis. Current depressive symptoms were assessed with the Beck Depression Inventory, a 21-item self-administered scale (36). PTSD symptoms were assessed

using the civilian version of the PTSD Symptom Checklist a 17-item scale (37). Trait anxiety was measured with the State-Trait Anxiety Inventory (38). To measure trait anger symptoms, we used the Spielberger's State-Trait Anger Expression Inventory (39); to measure hostility, we administered the Cook-Medley Hostility Scale (40), and to assess general perceived stress, we used the Perceived Stress Scale (41).

Outcomes

Participants were followed prospectively for adverse cardiovascular outcomes for a median time of 5 years after the baseline visit. Follow-up information was collected through patient contacts, medical record review, and by querying the Social Security Death Index. Patients were contacted at their approximate 3-year and 5-year anniversary from their initial visit. If hospitalizations or procedures were reported, patients' physicians were contacted, and hospital records were obtained. Follow-up was virtually complete, with only 5 (1.6%) patients lost to follow-up. Ascertained cardiovascular events included cardiovascular death, recurrent MI (type 1), stroke and heart failure hospitalization. All events were adjudicated by consensus by study investigators (AJS, AAQ, VV), who were blinded to other study data. Cardiovascular death was defined as death attributable to an ischemic cardiovascular cause (fatal MI), cardiac arrhythmia (including cardiac resuscitation), or heart failure. The endpoint of the study was a composite outcome of adverse cardiovascular events, including cardiovascular death, recurrent MI, stroke, or hospitalization for heart failure.

Statistical Analysis

We calculated descriptive statistics of the sample and compared them by race using chi-squared tests for categorical variables and t-tests for continuous variables. Next, we used Kaplan Meier survival curves and Cox proportional hazards models to derive hazard ratios (HR) and 95% confidence intervals (CI) for the association between race (Black vs non-Black patients) and adverse outcomes. Pre-defined covariates were included in a sequential fashion to the unadjusted model to assess the impact of covariate adjustment on the estimate for race. First, we added demographic variables, including age and sex, followed by the addition of SES factors including education, income and employment status, and then psychosocial factors. Because virtually all the psychological factors were related to race, to avoid model overfitting, we constructed a global psychological distress measure integrating the six scales of psychological characteristics (symptoms of depression, anxiety, anger, perceived general stress, PTSD, and hostility) using similar methodology previously followed by us and others (42–44). Individuals were ranked on each of the six psychological measures; then all ranks were averaged for each participant to obtain a global psychological distress score (42). We also ran additional models were psychosocial factors were included as separate variables rather than in the aggregated score. Lastly, we added to the model baseline traditional risk factors and clinical characteristics, including smoking, history of hypertension, dyslipidemia, diabetes mellitus, and heart failure, left ventricular ejection fraction, BMI, and type of MI. This sequence was selected because SES and psychological factors were considered more "proximal" to race than traditional/clinical factors in the relationship to outcome, and potentially in the causal pathway between race and traditional risk factors. However, because we were interested in comparing the effect of SES vs. traditional/clinical risk factors on the estimate for race, we also inverted the order of the adjustment factors in the sequential models. Finally, we tested for interactions between race and

SES variables, including race-by-income, race-by-education or race-by-employment status interactions. The assumption of proportionality for the Cox proportional hazards regression model was assessed graphically and formally tested with the Schoenfeld residuals test.

The proportion of missing covariate data ranged between 0% and 11%. To avoid loss of information and possible bias due to these missing covariate values, multiple imputation was performed for the primary analysis with 50 imputations using Markov Chain Monte Carlo equations with SAS PROC MI. Imputed regression estimates were then combined using SAS PROC MIANALYZE.

Lastly, we performed a mediation analysis with bootstrapping (1,000 bootstrap samples and a 95 percent confidence interval (CI) to test the statistical effects of SES on the association of race with MACE (SPSS PROCESS macro version 2.16.3). This method uses an ordinary least squares or logistic regression-based path framework to estimate direct and indirect effects and produces CIs from bias-corrected bootstrap samples. Out of our three SES indicators, we chose income as the primary marker of SES in our mediation analysis because it may be a more sensitive indicator relative to educational attainment and employment, especially among Black individuals. Compared to Whites, Blacks receive less income and are less likely to be employed at the same education levels (31). All other statistical analyses were conducted using SAS 9.4 [SAS Institute Inc., Cary, NC] and significance level was set at $\alpha = 0.05$, two tailed.

RESULTS

Baseline Characteristics

A total of 205 Black and 108 non-Black participants were enrolled in the study. Table 1 shows descriptive characteristics of the analytic sample by race. There were no differences in age, but Black patients were more often female, less likely to be married, and had a more adverse socioeconomic profile, including lower income, lower education and lower likelihood to be employed. Black participants had more traditional cardiovascular risk factors than non-Black participants, including a higher BMI and a more frequent history of hypertension and diabetes mellitus. Black patients were also more likely to have a history of heart failure, but there were no differences by race in type of MI and left ventricular ejection fraction; Black patients were actually less likely than their non-Black counterparts to have obstructive CAD. Differences were also noted for use of preventive cardiac medications, with Black patients being less likely to be taking aspirin and statins. When psychological factors were compared by race, Black patients had a worse psychological risk profile for virtually all measures, and especially for depression, PTSD, and hostility scores, compared with non-Black patients.

Race and Adverse Cardiovascular Outcomes

During a median follow up of 5 years, 71 of 205 (35%) Black and 20 of 108 (19%) non-Black patients developed a composite study endpoint. In addition to the primary endpoint, Black patients had a higher rate of events for each individual component than non-Black patients (Figure 1). The cumulative incidence of adverse cardiovascular events was significantly higher in Black compared to non-Black patients, with an unadjusted hazard ratio of 2.1, 95% CI, 1.3–3.6 (Figure 2).

As shown in Figure 3, in sequential, nested multivariable models, addition of demographic variables did not affect the estimate by race (HR, 2.2, 95% CI, 1.3 – 3.6). Addition of SES variables induced a substantial attenuation of the differences in outcome by race (HR, 1.3, 95% CI, 0.8-2.4). Addition of the composite psychological distress index to the model did not further attenuate the difference in outcome by race (HR 1.4, 95% CI, 0.8– 2.5). Including psychosocial factors as separate variables in the model provided fairly similar results (data not shown); the HR for race was 1.5 (95% CI, 0.8-2.8). Lastly, addition of clinical risk factors including smoking history, BMI, history of hypertension, history of diabetes mellitus, history of heart failure, left ventricular ejection fraction and type of MI, contributed further to explain the residual risk, bringing the estimate for race close to the null (HR 1.1, 95% CI, 0.6– 1.9).

Next, we compared the impact the order of the adjustment factors in the sequential models after the demographics model (Table 2). Addition of SES variables first, heavily attenuated differences in outcome by race (HR, 1.3, 95% CI, 0.8-2.4), with a percent effect explained of 82%. In comparison, addition of clinical risk factors first, attenuated the effect to a lesser extent (HR 1.6, 95% CI, 0.9-2.7), with a percent effect explained of 55%. Lastly, addition of both SES and clinical risk factors together, brought the estimate for race close to the null (HR 1.1, 95% CI, 0.6– 1.9), with a percent effect explained of 92%. There were no significant interactions between race and SES variables, including race-by-income, race-by-education or race-by-employment status interactions.

Mediation Analysis

To quantify the effect of SES in the pathway linking Black race to MACE, we performed formal mediation analysis using income as a representative measure of SES. As shown in Figure 4, lower income significantly mediated the association of Black race with MACE by 45.7% (indirect effect/total effect).

DISCUSSION

In this sample of young and middle-aged men and women with recent MI, Black MI survivors had a more than a two-fold increased risk of adverse cardiovascular outcomes over 5 years of follow-up, and the excess risk was driven more by SES than by clinical risk factors. Psychological factors did not contribute to the disparity once SES factors were accounted for. The combination of SES and clinical risk factors explained most of the excess risk for Black patients with a much greater contribution of SES than clinical risk factors. A lower SES represented the dominant explanation for race-related differences in outcome in this study; a lower income explained almost 50% of the disparity. These results highlight the importance of SES as a determinant of health among young and middle-aged survivors of a MI and advance our understanding of the high risk for adverse outcomes faced by Black patients.

In the United States, race and SES are highly connected. However, no previous study has examined whether SES explains race-related outcome differences after an early-onset MI in younger individuals. Two previous studies found that SES explained a worse outcome after MI among Black than non-Black patients in older populations (21,32). A third study evaluated the relationship between race, area-level SES (measured by zip code–level median household income from Census data), and life expectancy among Medicare beneficiaries who were hospitalized with MI (6), and found that both Black race and low area-level SES were independent predictors of shorter life expectancy after acute MI. The authors found that post MI life expectancy was shorter for Black patients than White patients across all SES levels only in patients between 65 and 75 years of age. After multivariable adjustment, only younger Black patients (<68 years) had shorter life expectancies than their White counterparts, whereas older

Black patients had longer life expectancies than Whites. Thus, even though this sample was limited to patients aged ≥65 years, it highlights the fact that younger Black patients are at a disproportionately higher risk after an MI. This study also found that the largest White-Black gap in life expectancy occurred in younger patients living in high and medium-SES areas. In our study we found no evidence of interaction between race and SES, but we used individual-level SES rather than area-level SES. Consistent with our results, in another study of older patients, socioeconomic and social factors were the most important characteristics differentiating White and Black patients after an MI, and characteristics associated with Black race, including SES and social factors, but not race itself, were associated with mortality risk after MI (32).

In an effort to understand racial disparities in outcomes after MI, our study integrated robust psychological measures as these can be important mediators in the pathway connecting SES and cardiovascular outcomes (45–47). Although psychological stress is a known risk factor for incident cardiovascular disease, including MI (48–50), much of the previous work related to the role of psychological stress in health disparities by race has been limited to single domains of stress, such as discrimination, or to general perceived stress. Using comprehensive measures of psychological distress, we found that psychological disturbances did not contribute to disparities in outcome by race once socioeconomic factors were accounted for.

An important implication of our findings is that understanding the importance of social determinants of health in relation to traditional clinical risk factors is needed if we are to overcome existing disparities in outcomes (51). Although clinical interventions that address traditional risk factors may decrease the risk for both Black and non-Black patients after an MI, they are unlikely to eliminate racial disparities in CVD without concomitant interventions that

address upstream SES disadvantage. Our study suggests that this may be especially true among younger patients with MI, a group in which disparities in outcome by race after an MI are largest. Addressing SES inequalities is therefore urgently needed to improve the outcomes of younger Black patients with coronary heart disease. Policy changes or interventions targeted at upstream social determinants should be prioritized, along with risk factor control, in order to ameliorate health disparities.

The present findings should be interpreted in the context of potential limitations. First, the MIMS2 study included study participants from a single institution, therefore the results may not be generalizable throughout the country. However, the location of our study within the Atlanta metropolitan area allowed us to enroll an urban patient population with large representation of young Black patients. Second, because this was an observational study, race may be a proxy of unmeasured characteristics that differ by race. However, our study collected variables in multiple domains, including SES, psychological distress, and clinical risk factors, and the combination of these factors explained outcome differences by race almost completely. Third, this study relied on self-identified racial categories; thus, contributions of genetically-determined components of race/ethnicity to outcomes could not be determined. Nonetheless, in the context of racial disparities in health outcomes and social determinants of health, self-identified race is more relevant to consider than genetic ancestry (52). Indeed, our results support the notion that genetic factors do not play a large role in mortality difference by race, given that the latter was largely explained by socioeconomic characteristics, which are potentially modifiable. Lastly, we did not have information on health insurance and insurance coverage for medications.

There are also important strengths to this study. To our knowledge, this is one of a few studies of race-based differences in the outcome of MI among younger patients, and the first study to examine a complex set of patient characteristics, including individual-level SES indicators, a comprehensive psychological assessment, and detailed clinical data in explaining inequalities in outcome by race. The large number of young Black patients, the nearly equal numbers of men and women, and the broad portfolio of SES and psychological assessments make this study unique and well-suited to explore this question.

CONCLUSION

In a cohort of young and middle-aged post MI patients, we demonstrate that Black patients have more than a two-fold risk of developing adverse cardiovascular events compared to non-Black patients. While a multitude of factors contribute to these disparities, SES indicators are major drivers of these differences. Our results underscore the importance of social determinants of health for this at-risk population, and highlight the need to intervene in this area in order to mitigate racial disparities in the outcome of early-onset MI.

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TABLES/FIGURES

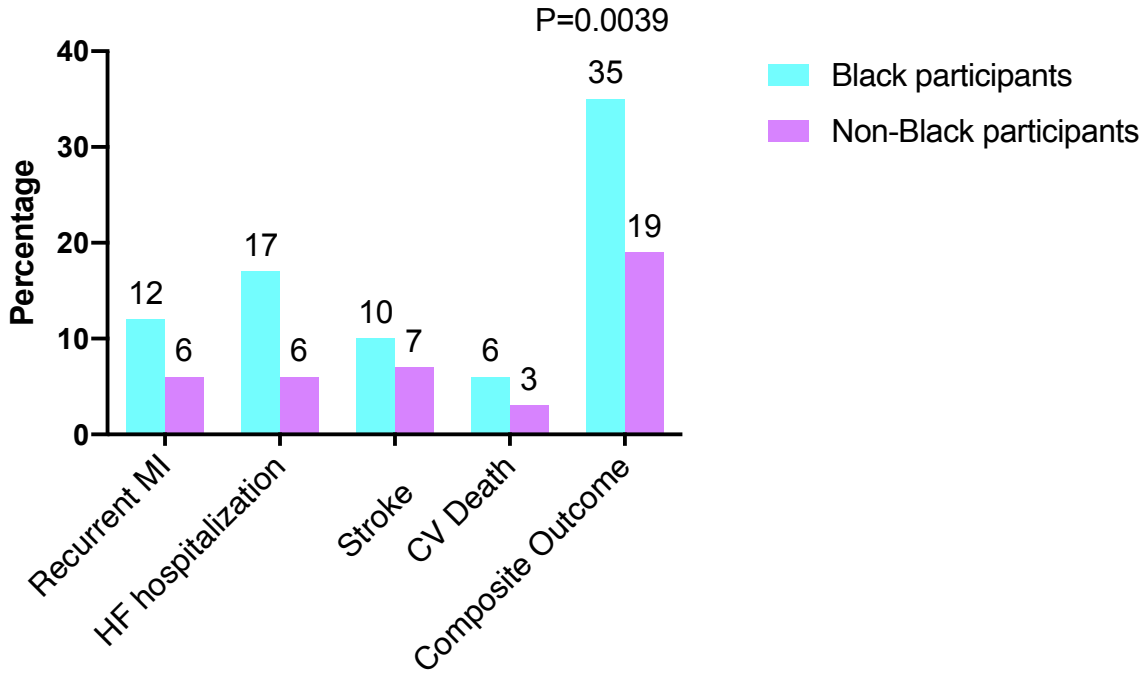

Figure 1. Composite Outcome and Individual Outcomes by Race
Abbreviations: CV=cardiovascular, MI=myocardial infarction

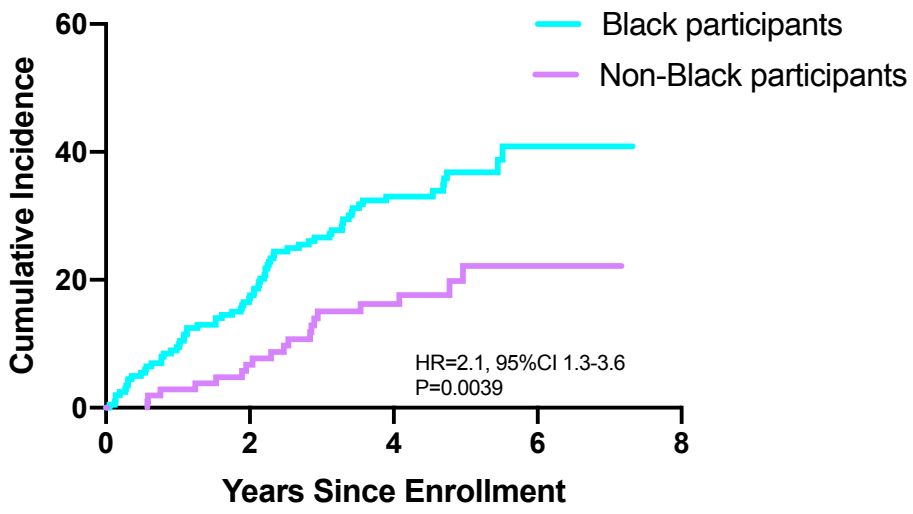

Figure 2. Cumulative Incidence for Association Between Race and Adverse Cardiovascular Outcomes (Composite Endpoint of Recurrent MI, Heart Failure Hospitalization, Stroke and Cardiovascular Death)

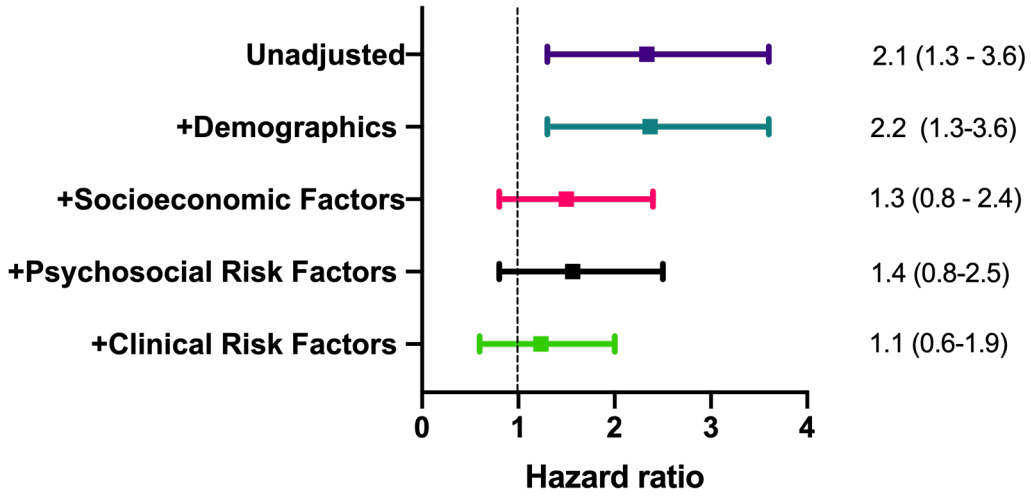

Figure 3 (Central Illustration). Forest Plot for Nested, Sequential Models for the Association of Race with Adverse Cardiovascular Events (Composite Endpoint of Recurrent MI, Heart Failure Hospitalization, Stroke and Cardiovascular Death)
***Hazard Ratio analysis of Black vs non-Black patients**
Demographic factors: age and sex
Socioeconomic factors: education, income and employment
Psychosocial factors: composite distress score
Clinical risk factors: smoking, BMI, History of hypertension, history of diabetes mellitus, history of dyslipidemia, history of heart failure, left ventricular ejection fraction and type of MI

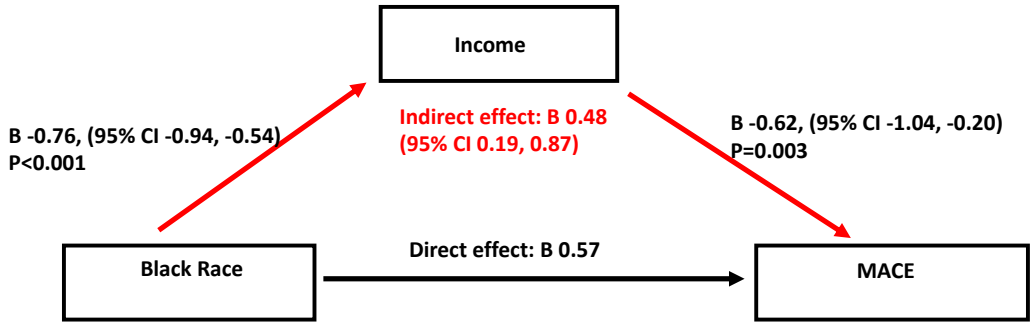

Figure 4. Mediation analysis
***Mediation analysis linking Black race and MACE through income (as marker of SES)**
Indirect effect =-0.76*-0.62. This pathway accounted for 45.7% of the total effect (indirect effect/(indirect effect +direct effect) x100).

Variable	Black races (n=205)	Non-Blacks races (n=108)	*P-value**
Demographics			
Age, y, mean (SD)	50 (7)	51 (6)	0.13
Age <50 y, %	42	31	0.04
Female, %	56	36	0.0008
Married/living with partner, %	30	65	<0.001
Income, %			<0.001
<$35,000/yr	64	27	
$35,000-$75,000/yr	28	25	
>$75,000/yr	8	48	
Education >12 y, %	52	73	0.0004
Employed, %	38	64	<0.0001
Cardiovascular risk factors			
BMI (kg/m2), mean (SD)	32 (8)	30 (7)	0.03
Ever smoker, %	58	49	0.15
History of hypertension, %	88	69	<0.001
History of dyslipidemia, %	81	79	0.63
History of diabetes mellitus, %	37	21	0.004
Prior MI to index MI, %	24	16	0.11
History of stroke, %	6	3	0.17
History of CABG, %	18	25	0.12
History of PTCA, %	69	70	0.77
Comorbidities			
Congestive heart failure, %	14	3	0.002
Peripheral artery disease %	2	3	0.56
Chronic obstructive pulmonary disease, %	7	7	0.9
Chronic kidney disease, %	5	3	0.3
Coronary angiography and electrocardiography results			
Gensini severity score, mean (SD)	37 (43)	49 (46)	0.03
Obstructive CAD (stenosis ≥ 70%), %	81	91	0.04
3-Vessel disease (at ≥ 70%), %	11	18	0.10
LV ejection fraction, mean (SD)	51 (12)	51 (12)	0.99
LV ejection fraction ≤ 35%, %	15	14	0.9
ST-segment elevation MI, %	26	36	0.07
Medication use			
Beta-blocker, %	86	83	0.4
Statin, %	81	92	0.01
Aspirin, %	77	91	0.003
P2Y12 inhibitors, %	65	79	0.008
ACE inhibitors, %	50	42	0.2
Anti-diabetics, %	32	20	0.03
Antidepressants, %	16	20	0.4
Laboratory values during index MI			

Maximum troponin (ng/L), mean (SD)	35 (60)	23 (45)	0.07
Hemoglobin A1c (%), mean (SD)	7 (2)	6 (2)	0.03
Total cholesterol (mg/dL), mean (SD)	175 (50)	176 (50)	0.8
HDL (mg/dL), mean (SD)	43 (13)	42 (16)	0.5
Triglycerides (mg/dL), mean (SD)	143 (121)	169 (118)	0.1
Psychosocial risk factors			
Beck Depression inventory, mean (SD)	14 (11)	10 (9)	0.004
PTSD Symptom Checklist, mean (SD)	34 (15)	28 (13)	0.0007
Anger Expression Inventory, mean (SD)	31 (12)	29 (14)	0.09
Anxiety State Inventory, mean (SD)	37 (13)	35 (13)	0.4
Perceived Stress Scale, mean (SD)	17 (9)	15 (9)	0.07
Hostility Scale, mean (SD)	0.2 (1)	-0.3 (1)	<0.0001
Composite distress score, mean (SD)	151(63)	125(65)	0.0012

Table 1. Descriptive characteristics of participants stratified by race (N=313) in the Myocardial Infarction and Mental Stress 2 Study (MIMS2) at baseline

Abbreviations: ACE=angiotensin converting enzyme, BMI=body mass index, CABG= coronary artery bypass graft, CAD=coronary artery disease, HDL= high density lipoprotein, LV=left ventricular, MI=myocardial infarction, PTCA= percutaneous transluminal coronary angioplasty, PTDS=post-traumatic stress disorder, SD=standard deviation, y=years
*Continuous variables compared using *t* tests, and categorical variables compared using χ^2 tests.

	HR (95% CI), Black vs. Non-Black Participants	Percent Effect Explained
Model 1: Adjusted for demographic variables (age and sex)	2.2 (1.3-3.6)	--
Model 2: **SES first:** Adjusted for demographic variables + socioeconomic factors (education, income & employment)	1.3 (0.8- 2.4)	82%*
Model 3: **Clinical factors first:** Adjusted for demographic variables + clinical risk factors (smoking history, BMI, history of hypertension, history of diabetes mellitus, history of heart failure, history of dyslipidemia, left ventricular ejection fraction and type of MI)	1.6 (0.9-2.7)	55%†
Model 4: **Both SES and clinical factors:** Adjusted for demographic variables and both socioeconomic and clinical factors (all variables in Models 2 and 3)	1.1 (0.6-1.9)	92%†

Table 2. Comparative Models for the Association of Race with Cardiovascular Events (Composite Endpoint of Recurrent MI, Heart Failure Hospitalization, Stroke and Cardiovascular Death)

Abbreviations: BMI= body mass index, CI=confidence interval, HR= hazard ratio, MI= myocardial infarction. The percent effect explained was derived by calculating percent change in the hazard ratio.

* Compared to Model 1.
† Compared to Model 2.