











ORIGINAL RESEARCH

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

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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 role played by socioeconomic, psychosocial, and clinical differences.

METHODS AND RESULTS: 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 Cox proportional-hazard models to estimate the association of race with a composite end point of recurrent MI, stroke, heart failure, or cardiovascular death after adjusting for demographic, socioeconomic status, psychological, and clinical risk factors. The mean age was 50 years, and 50% were women. Compared with non-Black patients, Black patients had lower socioeconomic status 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%); hazard ratio (HR) 2.1, 95% CI, 1.3 to 3.6. Adjustment for socioeconomic status weakened the association (HR 1.3, 95% CI, 0.8–2.4) more than adjustment for clinical and psychological risk factors. A lower income explained 46% of the race-related disparity in outcome.

CONCLUSIONS: Among young and middle-aged adult survivors of an MI, Black patients have a 2-fold higher risk of adverse outcomes, which is largely driven by upstream socioeconomic factors rather than downstream psychological and clinical risk factors.

Key Words: cardiovascular disease ■ prognosis ■ risk factors ■ socioeconomic position

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 to 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

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CLINICAL PERSPECTIVE

What Is New?

- In young and middle-aged survivors of myocardial infarction, we demonstrate that Black patients have more than a 2-fold risk of developing adverse cardiovascular events compared with non-black patients.
- While a multitude of factors contribute to these disparities, socioeconomic status indicators are major drivers of these differences.

What Are the Clinical Implications?

- These 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 myocardial infarction.

Nonstandard Abbreviations and Acronyms

MIMS2	Myocardial Infarction and Mental Stress Study 2
SES	socioeconomic status

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 White individuals.⁹

This issue is especially unresolved among individuals with coronary heart disease. 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).^{10–15} In a large study of hospitalized patients with MI, the excess mortality in Black patients compared with White patients was observed only among patients younger than 65 years of age, and differences in mortality by race diminished as age increased.¹⁶ 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.^{17–19} This earlier study, however, lacked information on SES. To date, few studies have focused on the young and middle-aged post-MI population to understand the reasons behind the high risk of Black patients and 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.²⁰ 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 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.

METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Study Design

Between August 2012 and March 2016, a total of 313 adult men and women were enrolled from the MIMS2 (Myocardial Infarction and Mental Stress Study 2), a prospective cohort study of patients 18 to 60 years of age with a documented history of MI in the previous 8 months at Emory-affiliated hospitals in Atlanta, Georgia.²¹ 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 ECG or other evidence of myocardial necrosis.²² 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 antidepressants. 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 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/m²). 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] versus 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.²³

We obtained 6 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.²⁴ PTSD symptoms were assessed using the civilian version of the PTSD Symptom Checklist, a 17-item scale.²⁵ Trait anxiety was measured with the State-Trait Anxiety Inventory.²⁶ To measure trait anger symptoms, we used the Spielberger’s State-Trait Anger Expression Inventory²⁷; to measure hostility, we administered the Cook-Medley Hostility Scale,²⁸ and to assess general perceived stress, we used the Perceived Stress Scale.²⁹

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 ~3- 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 (A.J.S., A.A.Q., V.V.), 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 end point 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. Next, we used cumulative incidence plots and Cox proportional hazards models to derive hazard ratios (HR) and 95% CI for the association between race (Black versus 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.³⁰ 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.^{31–33} Individuals were ranked on each of the 6 psychological measures; then all ranks were averaged for each participant to obtain a global psychological distress score.³¹ We also ran additional models where 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 versus 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, and 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

Table 1. Descriptive Characteristics of Participants Stratified by Race (N=313) in the Myocardial Infarction and Mental Stress 2 Study (MIMS2) at Baseline

Variable	Black Participants (n=205)	Non-Blacks Participants (n=108)
Demographics		
Age, y, mean (SD)	50 (7)	51 (6)
Age <50 y, %	42	31
Female, %	56	36
Married/living with partner, %	30	65
Income, %		
<\$35 000/y	64	27
\$35 000–\$75 000/y	28	25
>\$75 000/y	8	48
Education >12 y, %	52	73
Employed, %	38	64
Cardiovascular risk factors		
BMI (kg/m ²), mean (SD)	32 (8)	30 (7)
Ever smoker, %	58	49
History of hypertension, %	88	69
History of dyslipidemia, %	81	79
History of diabetes mellitus, %	37	21
Prior MI to index MI, %	24	16
History of stroke, %	6	3
History of CABG, %	18	25
History of PTCA, %	69	70
Comorbidities		
Congestive heart failure, %	14	3
Peripheral artery disease %	2	3
Chronic obstructive pulmonary disease, %	7	7
Chronic kidney disease, %	5	3
Coronary angiography and electrocardiography results		
Gensini severity score, mean (SD)	37 (43)	49 (46)
Obstructive CAD (stenosis ≥70%), %	81	91
3-Vessel disease (at ≥70%), %	11	18
LV ejection fraction, mean (SD)	51 (12)	51 (12)
LV ejection fraction ≤35%, %	15	14
ST-segment elevation MI, %	26	36
Medication use		
Beta-blocker, %	86	83
Statin, %	81	92
Aspirin, %	77	91
P2Y12 inhibitors, %	65	79
ACE inhibitors, %	50	42
Anti-diabetics, %	32	20

(Continued)

Table 1. Continued

Variable	Black Participants (n=205)	Non-Blacks Participants (n=108)
Antidepressants, %	16	20
Laboratory values during index MI		
Maximum troponin (ng/L), mean (SD)	35 (60)	23 (45)
Hemoglobin A1c (%), mean (SD)	7 (2)	6 (2)
Total cholesterol (mg/dL), mean (SD)	175 (50)	176 (50)
HDL (mg/dL), mean (SD)	43 (13)	42 (16)
Triglycerides (mg/dL), mean (SD)	143 (121)	169 (118)
Psychosocial risk factors		
Beck Depression inventory, mean (SD)	14 (11)	10 (9)
PTSD Symptom Checklist, mean (SD)	34 (15)	28 (13)
Anger Expression Inventory, mean (SD)	31 (12)	29 (14)
Anxiety State Inventory, mean (SD)	37 (13)	35 (13)
Perceived Stress Scale, mean (SD)	17 (9)	15 (9)
Hostility Scale, mean (SD)	0.2 (1)	−0.3 (1)
Composite distress score, mean (SD)	151 (63)	125 (65)

ACE indicates 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; PTSD, post-traumatic stress disorder; and SD, standard deviation.

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 (1000 bootstrap samples and a 95 % CI to test the statistical effects of SES on the association of race with major adverse cardiac events [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 3 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 with White individuals, Black individuals receive less income and are less likely to be employed at the same education levels.²⁰ 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.

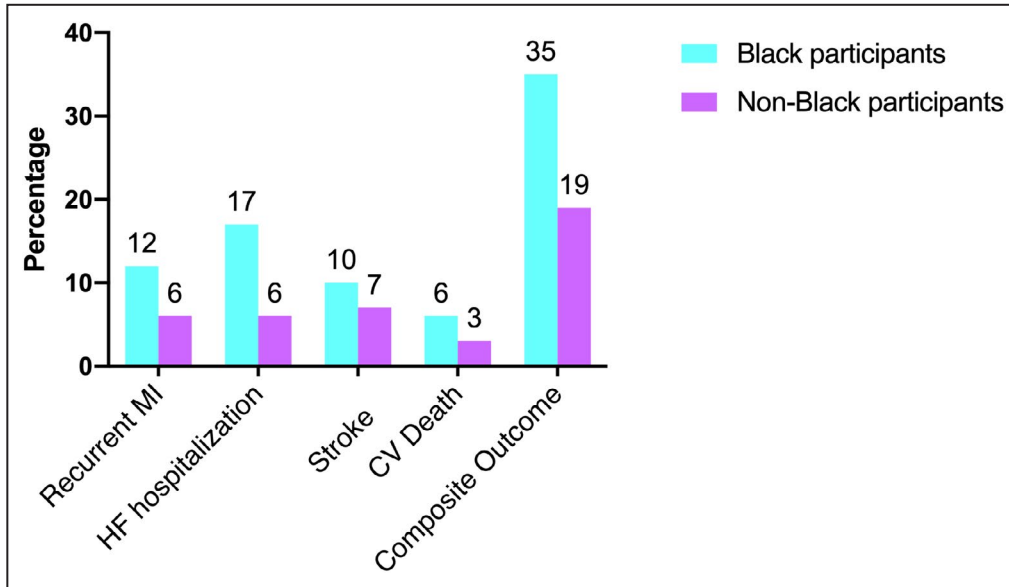


Figure 1. Composite outcome and individual outcomes by race. CV indicates cardiovascular; HF, heart failure; and MI, myocardial infarction.

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 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

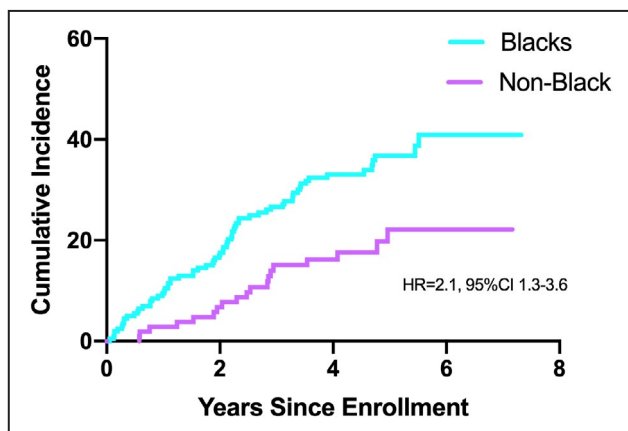


Figure 2. Cumulative incidence for the association between race and adverse cardiovascular outcomes (composite endpoint of recurrent MI, heart failure hospitalization, stroke, and cardiovascular death). MI indicates myocardial infarction.

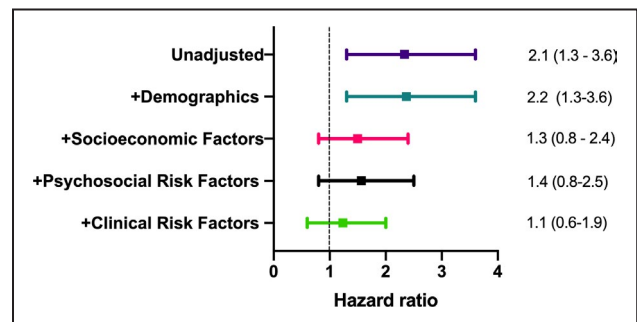


Figure 3. Forest plot for nested, sequential models for the association of race with adverse cardiovascular events (composite end point of recurrent MI, heart failure hospitalization, stroke, and cardiovascular death). Hazard ratio analysis of Black vs non-Black patients. Demographic factors: age and 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. BMI indicates body mass index; and MI, myocardial infarction.

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

	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: <i>SES first</i> : Adjusted for demographic variables+socioeconomic factors (education, income & employment)	1.3 (0.8–2.4)	82%
Model 3: <i>Clinical factors first</i> : 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: <i>Both SES and clinical factors</i> : Adjusted for demographic variables and both socioeconomic and clinical factors (all variables in Models 2 and 3)	1.1 (0.6–1.9)	92%

The percent effect explained was derived by calculating percent change in the hazard ratio. BMI indicates body mass index; HR, hazard ratio; and MI, myocardial infarction.

*Compared to Model 1.

scores, compared with non-Black patients. Descriptive characteristics of clinical risk factors stratified by income category appear in Table S1.

Race and Adverse Cardiovascular Outcomes

During a median follow up of 5 years, 71 of 205 (35%) Black and 20 of 108 (18%) non-Black patients developed a composite study end point. In addition to the primary end point, 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 with non-Black patients, with an unadjusted HR of 2.1, 95% CI, 1.3–3.6, $P=0.004$ (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 of 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

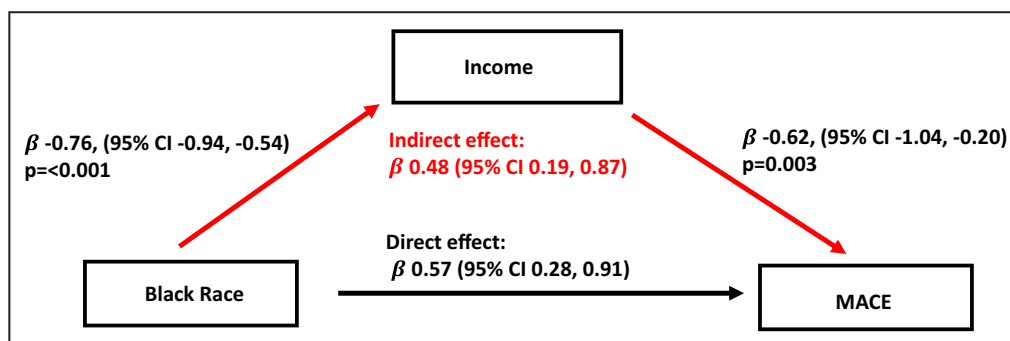


Figure 4. Mediation analysis.

Mediation analysis linking Black race and major adverse cardiac events 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) $\times 100$).

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 major adverse cardiac events, 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 major adverse cardiac events 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 2-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.^{10,34} 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.³⁴

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.^{35–37} Although psychological stress is a known risk factor for incident cardiovascular disease, including MI,^{38–40} 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 after accounting for socioeconomic factors.

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.⁴¹ 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 cardiovascular disease 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.⁴² 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.

CONCLUSIONS

In a cohort of young and middle-aged post-MI patients, we demonstrate that Black patients have more than a 2-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.

ARTICLE INFORMATION

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Disclosures

None.

Supplementary Material

Table S1

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SUPPLEMENTAL MATERIAL

Table S1. Descriptive characteristics of clinical risk factors stratified by income category (N=278) in the Myocardial Infarction and Mental Stress 2 Study (MIMS2) at baseline.

Variable	<35K (n=141)	35K-75K (n=74)	>75K (n=63)
Baseline demographics			
Age, y, mean (SD)	51 (7)	51 (7)	52 (5)
Female, %	57	49	40
Married/living with partner, %	26	46	84
Education >12 y, %	46	66	92
Employed, %	24	58	86
Clinical Risk Factors			
BMI (kg/m ²), mean (SD)	32 (7)	32 (7)	30 (7)
Ever smoker, %	66	41	41
History of hypertension, %	90	78	64
History of dyslipidemia, %	82	79	79
History of diabetes mellitus, %	43	28	13
History of congestive heart failure, %	12	9	0
History of stroke, %	8	1	2
History of CABG, %	22	23	16
History of PTCA, %	69	74	70
Coronary angiography and electrocardiography results			
Gensini severity score, mean (SD)	42 (47)	39 (37)	44 (50)
Obstructive CAD (stenosis \geq 70%), %	83	89	85
3-Vessel disease (at \geq 70%), %	16	11	13
LV ejection fraction, mean (SD)	52 (12)	50 (13)	52 (11)
ST-segment elevation MI, %	26	32	35
Medication use			
Beta-blocker, %	87	89	76
Statin, %	84	77	95
Aspirin, %	83	70	92
P2Y12 inhibitors, %	67	72	74
ACE inhibitors, %	52	38	9
Anti-diabetics, %	36	26	15
Antidepressants, %	18	22	13
Psychosocial risk factors			
Beck Depression inventory, mean (SD)	16 (11)	10 (8)	7 (8)
PTSD Symptom Checklist, mean (SD)	37 (16)	29 (11)	25 (10)
Anger Expression Inventory, mean (SD)	33 (14)	30 (12)	25 (12)
Anxiety State Inventory, mean (SD)	40 (14)	34 (11)	30 (11)
Perceived Stress Scale, mean (SD)	19 (8)	15 (8)	11 (8)
Hostility Scale, mean (SD)	21 (9)	18 (8)	13 (7)
Composite distress score, mean (SD)	166 (64)	135 (57)	96 (51)

BMI=body mass index, SD=standard deviation, MI= myocardial infarction