Insurance Status, not Race, is Associated with Mortality After an Acute Cardiovascular Event in Maryland

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BACKGROUND: It is unclear how lack of health insurance or otherwise being underinsured contributes to observed racial disparities in health outcomes related to cardiovascular disease.

OBJECTIVE: To determine the relative risk of death associated with insurance and race after hospital admission for an acute cardiovascular event.

DESIGN: Prospective cohort study in three hospitals in Maryland representing different demographics between 1993 and 2007.

PATIENTS: Patients with an incident admission who were either white or black, and had either private insurance, state-based insurance or were uninsured. 4,908 patients were diagnosed with acute myocardial infarction, 6,759 with coronary atherosclerosis, and 1,293 with stroke.

MAIN MEASURES: Demographic and clinical patient-level data were collected from an administrative billing database and neighborhood household income was collected from the 2000 US Census. The outcome of all-cause mortality was collected from the Social Security Death Master File.

KEY RESULTS: In an analysis adjusted for race, disease severity, location, neighborhood household income among other confounders, being underinsured was associated with an increased risk of death after myocardial infarction (relative hazard, 1.31 [95 % CI: 1.09, 1.59]), coronary atherosclerosis (relative hazard, 1.50 [95 % CI: 1.26, 1.80]) or stroke (relative hazard, 1.25 [95 % CI: 0.91, 1.72]). Black race was not associated with an increased risk of death after myocardial infarction (relative hazard, 1.03 [95 % CI: 0.85, 1.24]), or after stroke (relative hazard, 1.18 [95 % CI: 0.86, 1.61]) and was associated with a decreased risk of death after coronary atherosclerosis (relative hazard, 0.82 [95 % CI: 0.69, 0.98]).

CONCLUSIONS: Race was not associated with an increased risk of death, before or after adjustment. Being underinsured was strongly associated with death among those admitted with myocardial infarction, or a coronary atherosclerosis event. Our results support growing evidence implicating insurance status and socioeconomic factors as important drivers of health disparities, and potentially racial disparities.

KEY WORDS: health disparities; insurance coverage; socioeconomic status; race; cardiovascular disease.
J Gen Intern Med
DOI: 10.1007/s11606-012-2147-9

INTRODUCTION

African Americans living in poor, urban neighborhoods bear a high burden of illnesses and early death. By one estimate, life expectancy in these communities is 14 to 18 years less than that of other US groups living in healthier neighborhoods.[1] A major component of this mortality disparity is due to cardiovascular disease (CVD).[2–5] Since effective management of hypertension and other modifiable risk factors can delay and prevent deaths due to CVD, poor or incomplete management of these conditions may be at least partly responsible for the reported mortality disparities.[6–8] Lack of health insurance, or otherwise being underinsured (that is, having less than typical private insurance), may be a major cause of insufficient treatment and subsequent mortality, but it is unclear the extent that observed racial disparities are actually due to being underinsured.

Insurance coverage in the US is expected to expand in the coming years. However, it remains to be seen whether excess mortality due to CVD among those who are underinsured will decrease with this expansion in coverage. Being underinsured is highly associated with race and socioeconomic status (SES), and therefore it is unclear whether health effects associated with health insurance are due to race, factors that correlate with SES (like neighborhood psychosocial hazards, access to healthful foods, or lifestyle habits), or something else entirely.[9–11] Thus, accounting for race and SES is essential to disentangling the complex relationship between insurance coverage and excess mortality.

In order to better understand the impact of being underinsured on morality due to CVD, we sought to quantify the association between insurance status and race on all-cause

Electronic supplementary material *The online version of this article (doi:10.1007/s11606-012-2147-9) contains supplementary material, which is available to authorized users.*

Received April 2, 2012
Revised June 2, 2012
Accepted June 12, 2012

Published online: 21 July 2012

mortality independent of SES and other important confounders (like disease severity). To accomplish this, we determined the relative risk of mortality associated with insurance (underinsured versus privately insured) and race (black versus white), while accounting for neighborhood SES and disease severity in a sample of patients admitted to one of three Maryland hospitals for a CVD event: acute myocardial infarction (MI), coronary atherosclerosis or stroke.

METHODS

Dataset

The dataset consisted of all patients admitted to one of three Maryland hospitals with a primary ICD-9 diagnosis code of acute MI (code 410), coronary atherosclerosis (code 414), or stroke (code 434). Clinical definitions of all codes are provided in the online appendix. The definition of coronary atherosclerosis included all variants, whether native vessels or grafted vessels. All diagnoses were, at least in theory, relatively specific for legitimate anatomic coronary artery disease with risk of complications over time.

Each hospital served demographically distinct populations: a low SES urban/inner city population (1993-2007), a middle SES urban population (1995-2007) and a high SES suburban population (1997-2007). The period of retrospective observation began with the patient's first admission (i.e., incident admission), and ended at the time of death or censor on December 31, 2007. Subjects were limited to patients self-reporting as white or black, at least 18 years of age, and with either private insurance or state-based insurance (excluding Medicare; see online appendix for the flow of subject exclusions). We excluded patients who did not consider themselves black or white, since they comprised a small percentage of patients and we sought a clear and dichotomous comparison. Subjects whose insurance was Medicare were excluded since no data were available about their coverage prior to age 65, and the primary comparison of interest was between the privately insured and the underinsured (rather than private versus public insurance). We did not censor patients at the age of 65, because we suspected that insurance coverage prior to this age would affect subsequent health in later years even though Medicare is an alternate form of coverage. Those who were close to 65 years at the time of study entry were assumed to have different long-term health risks, depending on their insurance status prior to being eligible for Medicare. This study was approved by the Johns Hopkins Institutional Review Board.

Death Information

Patients were matched to the May 31, 2008 Social Security Administration's Death Master File (SSA DMF, accessed August, 2008), an all cause death registry. The matching algorithm was adapted from a previously published algorithm [12] using social security number, date of birth and last name. Subjects who did not match were assumed to be alive on December 31, 2007.

The SSA DMF death information was validated by a random sample matched to the National Death Index, a standard for obtaining death information. The sensitivity of the SSA DMF for identifying patients who had died was 86 % (95 % CI: 76 %, 94 %) and the specificity was estimated as 97 % (95% CI: 96 %, 98 %), indicating good agreement. Sensitivity and specificity did not vary by insurance status or race (for details on the algorithm and validation, see the online appendix).

Insurance Status

Insurance status at the time of admission was obtained from the hospital billing database. Patients with Medicaid and uninsured patients were classified as being underinsured. During the study period (1993-2007), many adults in Maryland cycled between Medicaid and uninsured status, due to the requirement that Medicaid beneficiaries demonstrate on-going disability or need through an annual reapplication process. Lower physician reimbursement by Medicaid may also limit access to primary care, placing enrollees at higher risk than those who are privately insured.[13] Furthermore, if a patient was uninsured at the time of hospital admission and subsequently awarded Medicaid coverage for that visit, the patient was retroactively classified as having Medicaid. Medicaid was rarely awarded prior to the first month after discharge, and therefore may have had little impact on immediate management of cardiovascular risk factors. For these reasons, the distinction between Medicaid status and uninsured status during the time frame of this analysis was imprecise and warranted inclusion of Medicaid in the underinsured category.

Race

Race was self-reported on patient information forms. A small percentage of subjects (7 % for acute MI, 8 % for coronary atherosclerosis and 6 % for stroke) did not classify themselves in either category and were excluded. More detailed information was not available regarding ethnicities and other races (such as Hispanic or mixed race).

Median Neighborhood Income

Subject addresses were geocoded to census block groups using ArcGIS 9.10 (ESRI, Redlands, California). Each subject was then linked to 2000 Census estimates of median

household income as a proxy for neighborhood SES. Block group estimates of median household income were not intended to measure subject-level income, but rather reflect neighborhood characteristics in a similar approach to previous literature.[14–19] The successful match rate of addresses to Census block group was approximately 80 %. To reduce missing data bias, multiple imputation methods were conducted using the *mi impute* function of Stata 11.[20–22] Specifically, complete median household income was log-transformed and treated as a continuous outcome in a multivariate linear regression under the assumption that the missing data were missing at random.[23] The covariates on which the imputation datasets were based included age, sex, race, marital status, hospital location, disease severity, length of stay, insurance status and death. For each 30 repetitions, the imputed variable was categorized by low (<$30,000), medium ($30,000 to $60,000) and high (>$60,000) neighborhood income for the final models.

Risk Adjustment

Risk adjustment is of particular importance to ensure that individuals of similar disease severity are compared appropriately. [24] Disease severity was measured by 3 M's All Patient Refined Diagnosis Related Groups (APR–DRG) version 12, 15, 20 and 25 for fiscal years 1993-1998, 1999-2003, 2004-2006, and 2007, respectively. The APR–DRG defines mortality risk in a four-point scale (1 to 4), 1 indicating low risk and 4 referring to high risk of mortality, and also a separate four-point scale for disease severity; both were included as covariates in the analysis. This method has recently been used successfully in epidemiologic risk adjustment.[25–27] Length of stay, another proxy of disease severity, was categorized by every two days, and treated as a continuous variable.

Confounders

Age, marital status (defined as married, single or formerly married) and sex were included in the survival models. Since some in-hospital treatments and elective/semi-elective procedures listed as secondary diagnoses were likely related to access to care, these variables may have mediated the effect of insurance status on death. Therefore, in-hospital procedures and variables related to treatment beyond the primary diagnosis code were not included as confounders. Calendar year of admission was also investigated as a potential confounder, since treatments and hospital protocols may have changed from 1993 to 2006. Based on model fit statistics, calendar year was included for acute MI (restricted cubic spline with 5 knots) and coronary atherosclerosis (linear spline with 4 knots), but not stroke.

Statistical Analysis

Baseline demographics and clinical characteristics were described by medians (and interquartile ranges) and percent frequencies. For each clinical condition, Cox proportional hazard models were used to estimate relative hazard (RH) of risk of death between the exposures (underinsured vs. privately insured; black vs. white) and reported with 95 % confidence intervals.

The proportional hazards assumption was tested by assessing the interactions between all main effects (insurance and race) and confounders with time as well as graphical approaches (assessment of Schoenfeld residuals, plotting the log(-log(S(t))) against time, and testing whether the log hazard-ratio function is constant over time (using *estat phtest* in Stata).[28] If the interaction with time was significant (according to Bonferroni-corrected p-values at the p=0.05/15 level, since 15 interactions were assessed), then the covariate was included as stratum within the model. Hospital location was also included in the model as a stratification variable: we were interested in the average effect of exposure across hospitals, and we sought to avoid potential confounding related to hospital effects, particularly since hospital populations differed by insurance status and race. The final models quantified the differential risk of death between private insurance and being underinsured, as well as the effect of race. The final models adjusted for age, sex, marital status, APR–DRG disease severity risk score, length of stay and median household income (as a proxy for neighborhood effects), with stratification for hospital location and APR–DRG mortality risk score. The interaction between health insurance and race was also assessed for each condition.

A sensitivity analysis was performed to investigate the potential influence of unmeasured confounding factors associated with death and the two exposures of interest (insurance status and race) on our results.[29–31] This analysis was based on a set of Monte Carlo simulations for each condition (acute myocardial infarction, coronary atherosclerosis and stroke) and each exposure (insurance status and race). Please refer to the online appendix for details.

Statistical analyses and graphics were conducted using Stata 11 (StataCorp, College Station, Texas, 2009), SAS (SAS Institute, Cary, North Carolina), and R (version 2.14.0). Statistical significance was defined by $P<0.05$.

Funding

This study was not funded by external sources.

RESULTS

Baseline demographic and clinical characteristics by insurance status are presented for acute MI (Table 1a), coronary

Table 1. Baseline characteristics by condition and insurance status. Median [IQR] or % (n).

	Acute myocardial infarction			Coronary atherosclerosis			Stroke		
	n=4908			n=6759			n=1293		
	Privately Insured	Underinsured	P-value	Privately Insured	Underinsured	P-value	Privately Insured	Underinsured	P-value
	n=4146 (84 %)	n=762 (16 %)		n=6026 (89 %)	n=733 (11 %)		n=931 (72 %)	n=362 (28 %)	
Age	56 [49, 62]	53 [46, 59]	< 0.001	58 [52, 63]	54 [47, 60]	< 0.001	57 [48, 64]	53 [44, 61]	< 0.001
Male	73 % (2968)	58 % (428)	< 0.001	75 % (4427)	53 % (374)	< 0.001	56 % (509)	45 % (191)	0.001
Black	13 % (531)	36 % (271)	< 0.001	11 % (682)	37 % (273)	< 0.001	35 % (322)	71 % (256)	< 0.001
Marital status			< 0.001			< 0.001			< 0.001
Married	69 % (2877)	31 % (233)		73 % (4400)	29 % (216)		57 % (531)	22 % (79)	
Single	20 % (814)	49 % (373)		17 % (1037)	46 % (337)		26 % (244)	62 % (226)	
Divorced/ Separated	6 % (256)	14 % (108)		5 % (329)	18 % (130)		7 % (69)	10 % (36)	
Widowed	5 % (199)	6 % (48)		4 % (260)	7 % (50)		9 % (87)	6 % (21)	
Neighborhood Median Household Income			< 0.001			< 0.001			< 0.001
< $30 000	10 % (355)	40 % (241)		9 % (426)	42 % (248)		17 % (130)	52 % (156)	
$30 000 - $60 000	53 % (1805)	51 % (313)		51 % (2500)	48 % (283)		47 % (373)	43 % (129)	
>$60 000	37 % (1266)	9 % (56)		40 % (1949)	10 % (60)		36 % (287)	5 % (15)	
Hospital Location			< 0.001			< 0.001			< 0.001
Urban, low SES	66 % (2730)	76 % (576)		87 % (5250)	86 % (633)		43 % (397)	68 % (247)	
Urban, mid SES	21 % (882)	23 % (173)		10 % (579)	13 % (96)		31 % (290)	29 % (103)	
Suburban, high SES	13 % (534)	2 % (13)		3 % (197)	1 % (4)		26 % (244)	3 % (12)	
Severity of Illness*			< 0.001			< 0.001			0.001
1 (Mild)	25 % (974)	17 % (129)		28 % (1699)	17 % (125)		15 % (131)	10 % (35)	
2 (Moderate)	48 % (1892)	46 % (346)		50 % (2956)	54 % (391)		53 % (474)	46 % (166)	
3 (Major)	17 % (667)	21 % (155)		17 % (1022)	22 % (162)		21 % (183)	27 % (96)	
4 (Extreme)	11 % (429)	17 % (126)		5 % (301)	7 % (53)		11 % (101)	17 % (60)	
Mortality Risk*			< 0.001			< 0.001			0.08
1 (Mild)	51 % (1949)	44 % (317)		58 % (3446)	49 % (356)		58 % (457)	52 % (173)	
2 (Moderate)	25 % (945)	25 % (184)		24 % (1421)	28 % (201)		23 % (177)	24 % (79)	
3 (Major)	15 % (587)	18 % (130)		15 % (879)	19 % (136)		10 % (81)	15 % (51)	
4 (Extreme)	9 % (343)	13 % (94)		3 % (179)	4 % (28)		9 % (70)	9 % (28)	
Length of Stay (days)	3 [2, 6]	4 [2, 6]	< 0.001	2 [1,6]	3 [1, 7]	< 0.001	4 [2, 7]	5 [3, 9]	< 0.001
Died	20 % (825)	24 % (180)	0.019	18 % (1108)	26 % (190)	< 0.001	24 %	25 %	0.72
Death rate per 100 person years	2.9	4.0	0.001	2.6	3.9	< 0.001	4.7	5.1	0.56
Follow up time in years†	7.0 [3.5, 10.1]	5.1 [2.5, 9.3]	0.001	7.2 [3.7, 10.4]	6.1 [3.4, 9.6]	< 0.001	4.4 [1.7, 7.8]	3.8 [1.5, 7.5]	0.40

** All patient refined diagnosis-related group categories. † Death or censoring. P-values calculated using Wilcoxon rank sum test for continuous variables, Fisher's exact test for categorical variables and Poisson regression for incidence rate comparisons.*

atherosclerosis (Table 1b) and stroke (Table 1c). In all three conditions, the proportion of subjects who were underinsured was much lower than those with private insurance, ranging from 11 % to 28 %. Underinsured status was also associated with younger age at time of admission, female sex, black race, unmarried status and lower neighborhood median household income. Clinically, the underinsured had a higher disease severity and a longer length of stay than those with private insurance. Within CVD conditions, the incidence rate of death per 100 person years was also higher among the underinsured.

ASSOCIATION OF INSURANCE STATUS AND RACE WITH DEATH

Figure 1 presents unadjusted Kaplan–Meier graphs by condition comparing insurance status (top row) and race (bottom row). These graphs demonstrate that underinsured subjects died sooner than subjects with private insurance, whereas there was similar survival between whites and blacks.

In developing the Cox proportional hazards models, the proportionality assumption for mortality risk was not upheld and this variable (categories 1 through 4) was included as strata [32]. The interaction between race and insurance status was not significant when included in the model for each condition. The P-values for the race and insurance status interaction terms were 0.19 for acute MI, 0.20 for coronary atherosclerosis and 0.96 for stroke. Thus, the final models did not include this interaction.

For acute MI, the effect of race was non-significant (RH: 1.03, 95 % CI: 0.85, 1.24), but the risk of death for the underinsured was 31 % higher than those privately insured (95 % CI: 1.09, 1.59), in the fully adjusted models (Table 2). A similar effect was observed for coronary atherosclerosis: the effect of race was significant (RH: 0.82, 95 % CI: 0.69, 0.98), but the confidence interval approached 1. The relative hazard of death for those underinsured was highly significant compared to those with private insurance (RH: 1.50 95 % CI: 1.26, 1.80). For stroke, those who were underinsured had a higher risk of

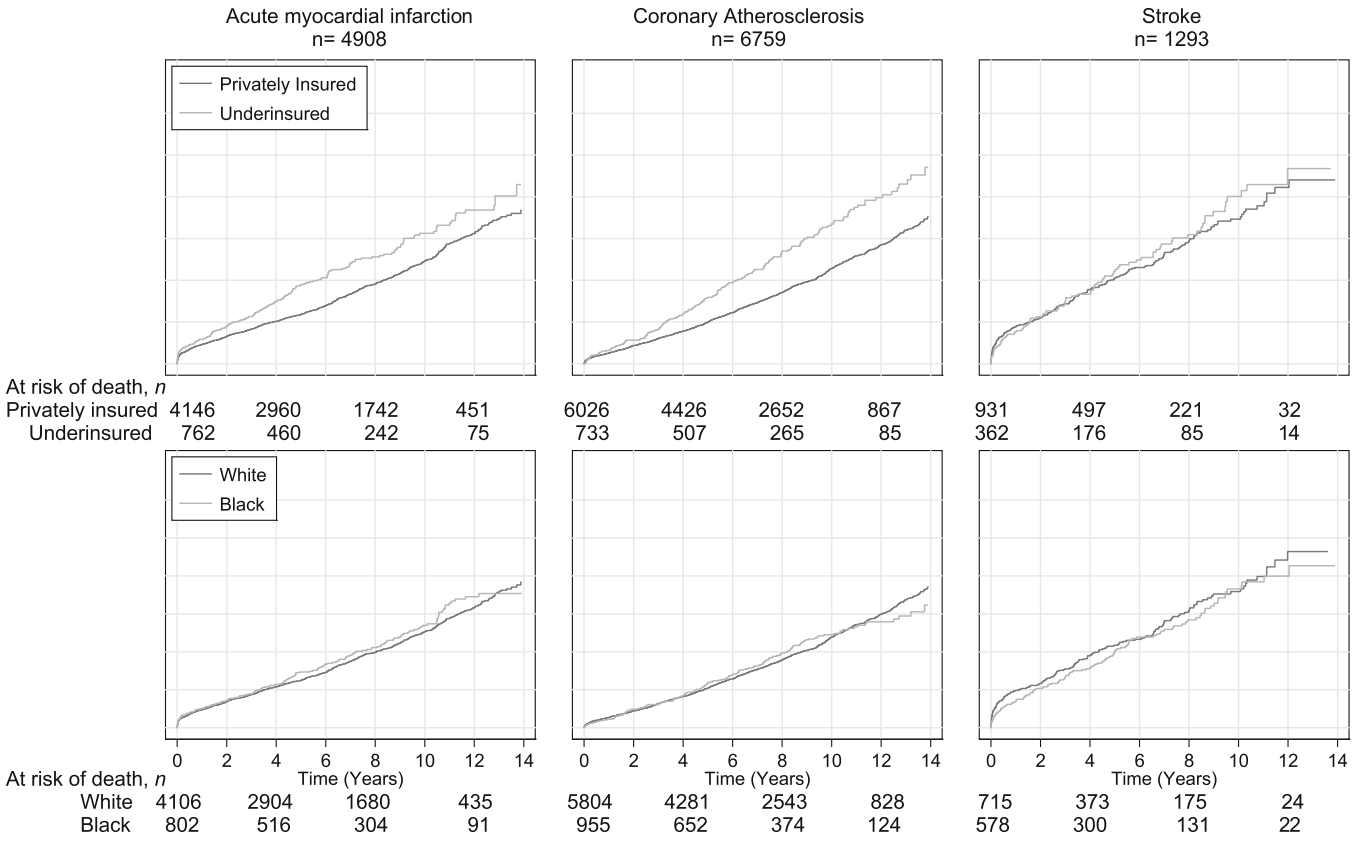

Figure 1. Kaplan–Meier plots, stratified by index admission condition comparing insurance status and race. The grey lines represent the expected high-risk groups (underinsured and black race).

death (RH: 1.25 95 % CI: 0.91, 1.72), but this effect was not significant. The effect of race was also non-significant (RH: 1.18 95 % CI: 0.86, 1.61).

Neighborhood median household income was included in each of the models as a confounder, but was not the primary exposure of interest, and was defined as a three-category variable: >$60,000 (reference), $30,000 to $60,000 and< $30,000. Lower neighborhood income was independently associated with a higher risk of death, and the effect was similar for acute MI and coronary atherosclerosis. The relative hazard of death comparing the middle to highest income group was 1.35 (95 % CI: 1.12, 1.62) for acute MI,

Table 2. Adjusted Relative Hazard Estimates (95 % CI) for All-Cause Mortality According to Insurance Status (Underinsured Vs. Privately Insured) and Race (Black Vs. White), Based on Cox Proportional Hazard Models

	Admission diagnosis		
Variable	**Acute myocardial infarction** *	**Coronary atherosclerosis** †	**Stroke** ‡
Privately insured	1	1	1
Underinsured	**1.31 (1.09, 1.59)**	**1.50 (1.26, 1.80)**	1.25 (0.91, 1.72)
White	1	1	1
Black	1.03 (0.85, 1.24)	**0.82 (0.69, 0.98)**	1.18 (0.86, 1.61)

**Model adjusted for age (continuous), sex, APR–DRG disease severity score (categorical score 1 to 4), marital status (married, single, formerly married), length of stay (continuous), and median household income (<$30,000, $30,000 to $60,000, >$60,000) and a restricted cubic spline for calendar year of admission, with stratification for three hospital locations and APR–DRG mortality risk (categorical score 1 to 4)*

† Model adjusted for age (continuous), sex, APR–DRG disease severity score (categorical score 1 to 4), marital status (married, single, formerly married), length of stay (continuous), and median household income (<$30,000, $30,000 to $60,000, >$60,000) and a linear spline for calendar year of admission, with stratification for three hospital locations and APR–DRG mortality risk (categorical score 1 to 4)

‡ Model adjusted for age (continuous), sex, APR–DRG disease severity score (categorical score 1 to 4), marital status (married, single, formerly married), length of stay (continuous), and median household income (<$30,000, $30,000 to $60,000, >$60,000), with stratification for three hospital locations and APR–DRG mortality risk (categorical score 1 to 4)

Missing median household income data were accounted for in 30 imputations.

***Bold** indicates P< 0.05. APR–DRG= All patients refined diagnosis-related group*

and 1.34 (95 % CI: 1.15, 1.59) for coronary atherosclerosis. The effect was stronger for the lowest income group: 1.42 (95 % CI: 1.10, 1.83) for acute MI and 1.45 (95 % CI: 1.16, 1.82) for coronary atherosclerosis. The RH estimates for stroke also indicated that living in lower income neighborhoods was a risk factor for death, but these effects were not significant. The risk of death was 37 % higher for the middle income group (RH: 1.37, 95 % CI: 0.90, 2.09) and 29 % higher for the lowest income group (RH: 1.29, 95 % CI: 0.77, 2.14).

CONFOUNDING BY UNMEASURED COVARIATES

Lastly, a sensitivity analysis determined the potential impact of unmeasured confounders on our results. Figure 2 presents the results from the final models for each condition, and how a potential unmeasured confounder might have influenced the confidence interval estimates of the relative hazards in contour plots. The analysis assessed the magnitude of association with both dependent and independent variables that a confounder would be required to have, in order to eliminate the significant adjusted association of being underinsured with acute MI and coronary atherosclerosis (that is, when the 95 % lower confidence limit became less than 1), and the effect of race for coronary atherosclerosis (when the 95 % upper confidence limit exceeded 1). Similarly, we assessed how a potential unmeasured confounder might induce a significant effect among the relationships not found to be significant (the effect of race for acute MI, and the effect of race and insurance status for stroke). The plots show that a confounding factor with odds ratio (OR) associations of at least two for death and insurance status would eliminate the significant effect of insurance in the acute MI model; a confounder of similar strength would be required to induce a significant effect of insurance in the stroke model. An unusually strong confounder would be required to reduce the effect of insurance for coronary atherosclerosis (ORs>4). In contrast, a relatively weak confounder would eliminate the significant effect

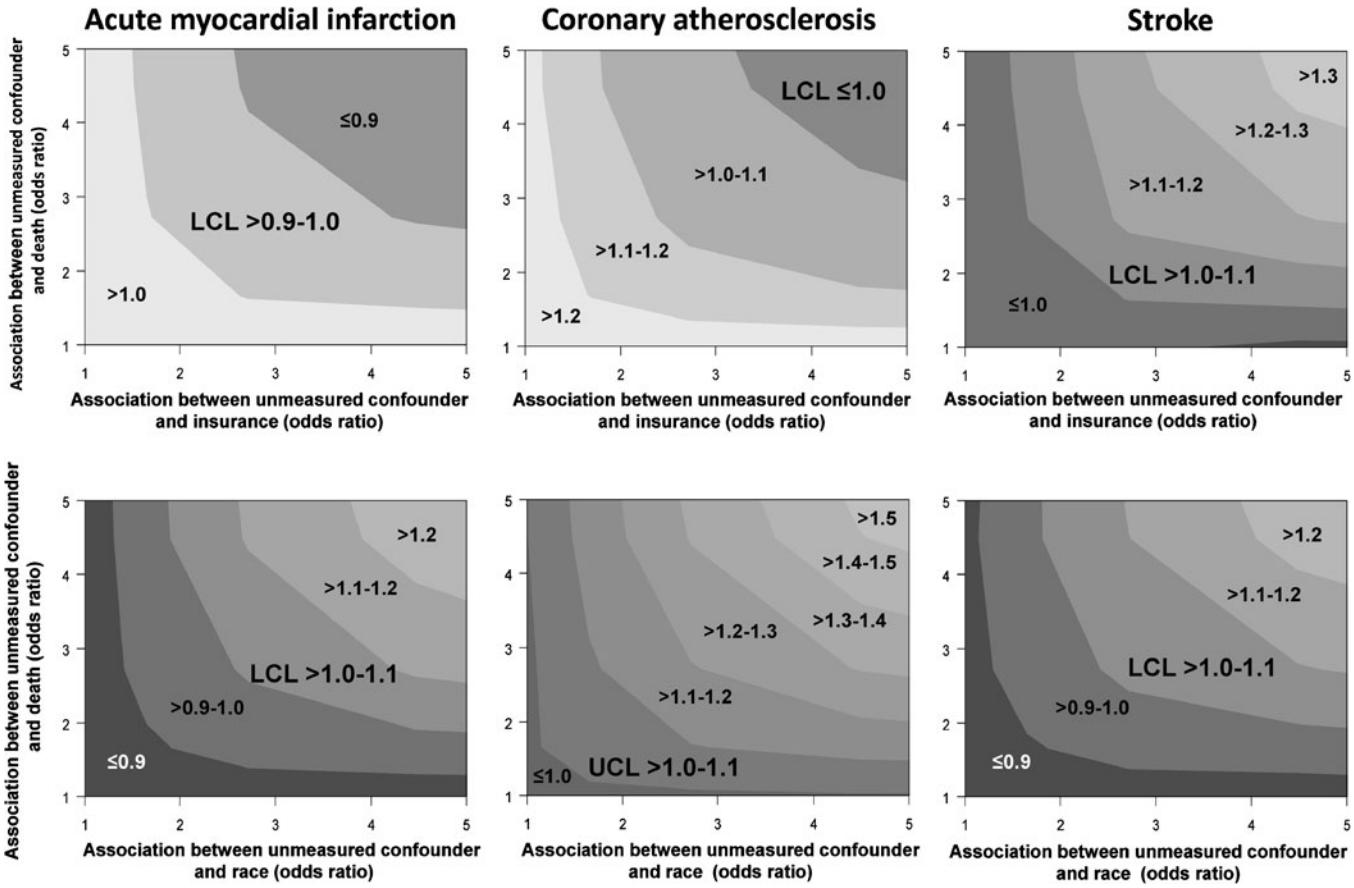

Figure 2. Sensitivity analyses investigating the effect of potential unmeasured confounding covariates on the 95 % significance level of association between insurance status and death, and race and death. These contour plots display how a potential confounding factor of varying degrees of association with the exposure of interest (insurance status or race, *x*-axis) and death (*y*-axis) would influence (increase or reduce) the labeled 95 % confidence limit of the relative hazard of death for being underinsured (compared to privately insured) or being black (compared to being white) in order to nullify or induce a significant effect.

of race for coronary atherosclerosis (ORs of about 1.5). A strong confounder (ORs>3.5–4) would be necessary to induce a significant effect of race in the acute MI and stroke models. These sensitivity analyses indicate that our results are fairly robust to unmeasured confounding at the 95 % level, except for the effect of race in coronary atherosclerosis.

DISCUSSION

This analysis showed a significantly increased risk of death associated with being underinsured (relative to those with private insurance) among those admitted with acute MI (31 % higher risk of death) and coronary atherosclerosis (50 % higher). Among those admitted with stroke, the risk of death was higher among the underinsured (25 % higher), but this effect was not significant. This association was independent of race, neighborhood SES and disease severity. Importantly, there was no significant association observed between race and risk of death for acute MI and stroke. Among those with coronary atherosclerosis, black race was protective (18 % lower risk of death, p=0.033), but a sensitivity analysis showed that this significance was not particularly robust to a potential unmeasured confounder.

The relative hazard of death was highest for coronary atherosclerosis, followed by acute MI, and stroke. This gradient in risk may be due to the fact that patients admitting with coronary atherosclerosis have less advanced disease, and therefore may have greater potential benefit from treatment than patients presenting with acute MI or stroke. Furthermore, stroke often occurs without warning (whereas many patients with coronary events have preceding symptoms), which may limit the opportunity to prevent future events, regardless of insurance status (a possible form of selection bias).

Our results are congruent with recent findings that general health disparities by race are minimized or eliminated when accounting for patient characteristics, geographic areas, social environments and class.[33,34] This consistency across two disparate study designs and health outcomes supports the validity of our analysis. In the context of CVD and mortality, our results support growing evidence implicating insurance [35] and socioeconomic factors as important drivers of racial disparities.[36] Indeed, lower neighborhood household income was also strongly associated with increased mortality. At the clinical level, attention should be given to individuals who are underinsured, as they constitute a particularly high-risk group. Furthermore, our results support the expansion of state-based insurance coverage with services comparable to private insurance as a potentially effective approach to closing mortality disparities due to CVD.

In this analysis, insurance status is likely a proxy for access to care and subsequent poor or incomplete management of cardiovascular risk factors among those with CVD. The phenomena associated with being underinsured, including insurance instability, problems with clinics accepting payments, and inability to afford medications, may be some of the factors that define this high-risk group and contribute to poor disease management. It is important to note, however, that the expansion of insurance coverage may be a necessary but not sufficient remedy for health disparities. For example, other factors related to SES and insurance status, such as health beliefs, will have an important impact on treatment adherence and subsequent outcomes, but will not be addressed through the expansion of health insurance alone.

Since all study subjects had been admitted to a hospital with a CVD event, we were unable to explore the potential role of insurance status and race on incident CVD events or the probability of surviving the initial event. Therefore, the impact of race on CVD prevalence and subsequent mortality in a community-based sample was not assessed. Once an event had occurred, however, our results indicate that race was not a primary driver of mortality, either before or after adjustment for known confounders including insurance status and neighborhood level SES. Additionally, the models that included the interaction suggested that the effect of insurance status was not modified by race when other confounders were taken into account. These results, while encouraging, require additional corroboration that race-based disparities in mortality are small after accounting for economic disparities, among those who have presented with a CVD event.

There were many strengths of this study. Firstly, we performed a prospective analysis (exposure defined prior to outcome) using retrospectively collected data. Secondly, the sample sizes for acute MI and coronary atherosclerosis were large, with substantial proportions of both exposures of interest. In a univariate analysis with 80 % power, the estimated detectable effect sizes (relative hazards) were about 1.11 and 1.12, for insurance status and race for these two conditions. The estimated effect size with 80 % power for stroke was 1.19 for insurance status and 1.17 for race. Interestingly, the results from the multivariate model were close to these minimum detectable differences (1.25 and 1.18, respectively).

Thirdly, there was a relatively long duration of follow-up (up to 14 years) that covered three distinct demographic areas, including traditionally underserved low-resource urban communities. These results cannot be generalized to rural areas or even other states, where state-based insurance benefits differ. Thus, these results are most applicable to Maryland, and more importantly, to groups with ambulatory-sensitive conditions such as CVD. Indeed, this analysis only included those with particular CVD events and it is

unclear how the effect of insurance on mortality would manifest in other chronic conditions.

Fourthly, we validated our use of the Social Security Administration Death Master File to identify those who had died by the National Death Index. There was minimal misclassification and most measurement error was found in misclassifying a subject who died as alive, which would bias towards (slightly) lower reported death rates (see online appendix for more information).

Lastly, this analysis is unique, since few studies have used a hospital billing database matched to census data for long-term outcomes in a cohort with CVD. Neighborhood-level SES correlates with social and community hazards that may be associated with higher mortality.[37,38] For example, high crime, availability of healthy food and otherwise stressful aspects within neighborhoods are exposures correlated with median household income that would adversely affect health. Accounting for these risk factors, at least partially, through median household income by census block group was important in demonstrating the putative independent effects of insurance status.

There were several important limitations to this study. Firstly, there was inherent measurement error in the administrative database that was not quantifiable in the analysis. Most uninsured patients applied for state-based insurance upon hospital admission, and therefore we were unable to distinguish uninsured subjects from those with state-based insurance prior to admission. While state-based insurance is not the same as having no insurance, we suspect that both profiles are at higher risk of adverse outcomes than those with private insurance. Nevertheless, we do not know the extent to which the associations we observed between being underinsured and mortality were driven by particularly bad outcomes among patients who lacked insurance altogether, and conversely, the extent to which including those with state-based insurance in our underinsured group may have attenuated the mortality risks we observed. Future work should examine the relative impact of no insurance compared with state-based insurance and, subsequently, the features of state-based insurance that contribute to poor outcomes.

Secondly, we were unable to account for insurance status after hospital discharge as a time-varying covariate. It is possible that those who were underinsured obtained private insurance later and vice versa and it is unclear the extent to which this might have attenuated the association between being underinsured and mortality. Nevertheless, it is important to note that being underinsured at the time of admission is reflective of chronic treatment and care prior to the event.

Thirdly, we acknowledge that median household income by census block group is an imperfect measure of SES. Neighborhood characteristics likely reflect important aspects of living conditions as well as shared norms that impact health, and has been used in previously published studies [16,18,19] as a strategy to adjust for SES.

Lastly, after adjustment for known and measured confounders, residual confounding due to unmeasured or imprecisely measured factors may have impacted the estimates of risk, as in any observation study. Our sensitivity analyses indicate, however, that the effect of any single unmeasured confounder must have an unusually strong effect to change the inferences of our results for insurance status. Similarly, for acute MI and stroke, an unmeasured confounder would need to be uncommonly large to make the effect of race statistically significant.

In summary, among a clinical population admitted to the hospital with an acute cardiovascular event, there was an increased risk of mortality among subjects who were underinsured compared to those who had private insurance. This effect was independent of race and other confounders. A sensitivity analysis suggested that an unmeasured confounder would need to be unusually strong to nullify the effect of insurance, indicating our results are at least moderately robust. Given the recent changes in health insurance and healthcare reform, this study underscores the need to closely investigate the factors relating to health insurance that may explain these disparities. Indeed, targeting these factors may relieve the burden of mortality disproportionally affecting those who are underinsured.

Acknowledgments: *The authors thank Chris Cobbs and the Johns Hopkins Casemix department for use of the data and anonymous reviewers in an earlier manuscript draft.*

Conflict of Interest: *The authors declare that they do not have a conflict of interest.*

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REFERENCES

1. **Murray CCJL, Kulkarni SC, Michaud C, Tomijima N, Bulzacchelli MT, Iandiorio TJ, et al.** Eight Americas: investigating mortality disparities across races, counties, and race-counties in the United States. PLoS Medicine. 2006;3:e260.
2. **Egan BM, Zhao Y, Axon RN.** US trends in prevalence, awareness, treatment and control of hypertension, 1988-2008. JAMA. 2010;303:2043–50.
3. **Gu Q, Burt VL, Paulose-Ram R, Yoon S, Gillum RF.** High blood pressure and cardiovascular disease mortality risk among US adults: the third National Health and Nutrition Examination Survey and mortality follow-up study. Ann Epidemiol. 2008;18:302–9.
4. **Barnato AE, Lucas FL, Staiger D, Wennberg DE, Chandra A.** Hospital-level racial disparities in acute myocardial infarction treatment and outcomes. Med Care. 2005;43:308–319.
5. **Budoff MJ, Nasir K, Mao S, et al.** Ethnic differences of the presence and severity of coronary atherosclerosis. Atherosclerosis. 2006;187:343–350.
6. **Kivimäki M, Shipley MJ, Ferrie JE, Singh-Manoux A, Batty GD, Chandola T, Marmot MG, Smith GD.** Best-practice interventions to reduce socioeconomic inequalities of coronary heart disease mortality in UK: a prospective occupational cohort study. Lancet. 2008;372:1648–54.

7. **Thomas AJ, Eberly LE, Davey Smith G, Neaton JD, Stamler J.** Race/ethnicity, income, major risk factors, and cardiovascular disease mortality. Am J Public Health. 2005;95:1417–23.
8. **Davey Smith G, Neaton JD, Wentworth D, Stamler R, Stamler J.** Mortality differences between black and white men in the USA: contribution of income and other risk factors among men screened for the MRFIT. MRFIT Research Group. Multiple Risk Factor Intervention Tria. Lancet. 1998;351:934–9.
9. **LaVeist TA.** Disentangling race and socioeconomic status: a key to understanding health inequalities. J Urban Health. 2005;82(2 Suppl 3):26–34.
10. **Navarro V.** Race or class versus race and class: mortality differentials in the United States. Lancet. 1990;336:1238–40.
11. **Groman R, Ginsburg J.** American College of Physicians. Racial and ethnic disparities in health care: a position paper of the American College of Physicians. Ann Intern Med. 2004;141:225–32.
12. **Hauser TH, Ho KK.** Accuracy of on-line databases in determining vital status. J Clin Epidemiol. 2001;54:1267–1270.
13. **Fakhraei SH.** Payments for physician services: an analysis of Maryland Medicaid reimbursement rates. Int J Healthc Tech Manag. 2006;7:129–42.
14. **Petersen KL, Marsland AL, Flory J, Votruba-Drzal E, Muldoon MF, Manuck SB.** Community socioeconomic status is associated with circulating interleukin-6 and C-reactive protein. Psychosom Med. 2008;70:646–53.
15. **Scheffler RM, Brown TT, Syme L, Kawachi I, Tolstykh I, Iribarren C.** Community-level social capital and recurrence of acute coronary syndrome. Soc Sci Med. 2008;66:1603–13.
16. **Rabi DM, Edwards AL, Svenson LW, et al.** Association of median household income with burden of coronary artery disease among individuals with diabetes. Circ Cardiovasc Qual Outcomes. 2010;3(1):48–53.
17. **Reinier K, Thomas E, Andrusiek DL, Aufderheide TP, Brooks SC, Callaway CW, et al.** Socioeconomic status and incidence of sudden cardiac arrest. CMAJ. 2011;183:1705–12.
18. **Guilley E, Herrmann F, Rapin C, et al.** Socioeconomic and living conditions are determinants of hip fracture incidence and age occurrence among community-dwelling elderly. Osteoporos Int. 2011;22:647–53.
19. **Jolly M, Mikolaitis RA, Shakoor N, Fogg LF, Block JA.** Education, zip code-based annualized household income, and health outcomes in patients with systemic lupus erythematosus. J Rheumatol. 2010;37:1150–1157.
20. **Zeng Z.** Multiple imputation for missing income data in population-based health surveillance. J Public Health Manag Pract. 2009;15:E12–21.
21. **Nur U, Shack LG, Rachet B, Carpenter JR, Coleman MP.** Modelling relative survival in the presence of incomplete data: a tutorial. Int J Epidemiol. 2010;39:118–128.
22. **Shrier I, Kahn SR, Steele RJ.** Effect of early physical activity on long-term outcome after venous thrombosis. Clin J Sport Med. 2009;19:487–493.
23. **Rubin DB.** Multiple imputation for nonresponse in surveys. New York: John Wiley & Sons; 1987.
24. **Iezzoni L, ed.** Risk adjustment for measuring healthcare outcomes. 3rd ed. Chicago: Health administration press; 2003.
25. **Nante N, Messina G, Cecchini M, et al.** Sex differences in use of interventional cardiology persist after risk adjustment. J Epidemiol Community Health. 2009;63:203–208.
26. **Sedman AB, Bahl V, Bunting E, et al.** Clinical redesign using all patient refined diagnosis related groups. Pediatrics. 2004;114:965–969.
27. **Romano PS, Chan BK.** Risk-adjusting acute myocardial infarction mortality: are APR–DRGs the right tool? Health Serv Res. 2000;34:1469–1489.
28. **Cleves M, Gould W, Gutierrez R, Marchenko Y.** An introduction to survival analysis using Stata, 2nd Edition. Stata Press College Station, Texas: 2008.
29. **Lash TL, Fink AK.** Semi-Automated Sensitivity Analysis to Assess Systematic Errors in Observational Data. Epidemiology. 2003;14:451–458.
30. **Phillips CV.** Quantifying and Reporting Uncertainty from Systematic Errors. Epidemiology. 2003;14:459–466.
31. **Kitahata MM, Gange SJ, Abraham AG, Merriman B, Saag MS, Justice AC, et al.** Effect of early versus deferred antiretroviral therapy for HIV on survival. N Engl J Med. 2009;360:1815–26.
32. **Klein JP, Moeschberger ML.** Survival Analysis: Techniques for Censored and Truncated Data 2nd edition. New York, NY: Springer; 2003.
33. **Spertus JA, Jones PG, Masoudi FA, Rumsfeld JS, Krumholz HM.** Factors associated with racial differences in myocardial infarction outcomes. Ann Intern Med. 2009;150:314–24.
34. **Laveist T, Pollack K, Thorpe R Jr, Fesahazion R, Gaskin D.** Place, not race: disparities dissipate in southwest Baltimore when blacks and whites live under similar conditions. Health Aff. 2011;30:1880–7.
35. **Fowler-Brown A, Corbie-Smith G, Garrett J, Lurie N.** Risk of cardiovascular events and death–does insurance matter? J Gen Intern Med. 2007;22:502–507.
36. **Bleich SN, Thorpe RJ, Sharif-Harris H, Fesahazion R, Laveist TA.** Social context explains race disparities in obesity among women. J Epidemiol Community Health. 2010;64:465–469.
37. **Borell LN.** Diez Roux AV, Rose K, Catellier D, Clark BL, Atherosclerosis risk in communities study: neighborhood characteristics and mortality in the atherosclerosis risk in communities study. Int J Epidemiol. 2004;33:398–407.
38. **Diez Roux AV, Merkin SS, Arnett D, Chambless L, Massing M, Nieto FJ, Sorlie P, Szklo M, Tyroler HA, Watson RL.** Community of residence and incidence of coronary heart disease. N Engl J Med. 2001.