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CLINICAL INVESTIGATIONS

Socioeconomic status and the development of atrial fibrillation in Hispanics, African Americans and non-Hispanic whites

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Background: Atrial fibrillation (AF) is the most common arrhythmia and is associated with significant morbidity and mortality. Despite having a higher burden of traditional AF risk factors, African American and Hispanic minorities have a lower incidence of AF when compared to non-Hispanic whites, referred to as the "racial paradox."

Hypothesis: Lower SES among Hispanics and African Americans may help to explain the lower incidence rates of AF compared to non-Hispanic whites.

Methods: An electrocardiogram/electronic medical records database in New York State was interrogated for individuals free of AF for development of subsequent AF from 2000 to 2013. SES was assessed per zip code via a composite of 6 measures *Z*-scored to the New York State average. SES was reclassified into decile groups. Cox regression analysis controlling for all baseline differences was used to estimate the independent predictive ability of SES for AF.

Results: We identified 48 631 persons (43% Hispanic, 37% African Americans, and 20% non-Hispanic white; mean age 59 years; mean follow-up of 3.2 years) of which 4556 AF cases occurred. Hispanics and African Americans had lower AF risk than whites in all SES deciles ($P < 0.001$ by log-rank test). Higher SES was borderline associated with lower AF risk (hazard ratio: 0.990, 95% confidence interval: 0.980-1.001, $P = 0.061$). *P* trend analysis was not significant by any race/ethnic group by SES deciles for AF.

Conclusions: Our study suggests that non-Hispanic whites were at higher risk for AF compared to nonwhites, and this was independent of SES.

KEYWORDS

Arrhythmias, Atrial Fibrillation, Risk Factors, Race and Ethnicity

1 | INTRODUCTION

Atrial fibrillation (AF) is the most common cardiac arrhythmia,[1] whose prevalence is increasing.[1,2] It represents a major public health problem and is closely related to stroke, mortality, decreased quality of life, and a high healthcare cost burden.[3] Previous research has identified AF risk factors to gather a better understanding of its causation; however, 44% of identified risk factors for AF remain unexplained.[4]

Studies examining the association between socioeconomic status (SES) and incident AF in the US population are limited, especially those examining racial/ethnic differences. It is known that both the incidence and risk burden of AF vary by racial/ethnic groups and SES status.[5] For example, despite having a higher burden of traditional AF risk factors, African Americans and Hispanics have a lower incidence of AF when compared to non-Hispanic whites.[6–9] This has been referred to as the "racial paradox."[10] Underdiagnosis of AF in minorities due to lower individual SES and poorer access to healthcare could explain this racial paradox.[11]

Exploration of the interaction between race/ethnicity and SES for the development of AF could further the understanding of this observed differences in risk of AF. Furthermore, there exists no literature investigating the relationship between SES and Hispanic ethnicity with incidence of AF, including the Atherosclerosis Risk in Communities (ARIC) study.[5] Therefore, we examined the relationship

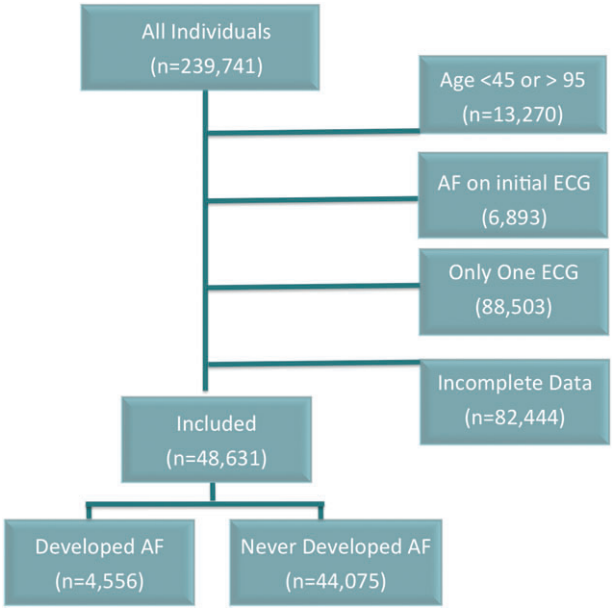

FIGURE 1 Cohort selection for included patients. Abbreviations: AF, atrial fibrillation; ECG, electrocardiogram.

between the development of AF in Hispanics, African Americans and non-Hispanic whites by SES. We hypothesized that lower SES among Hispanics and African Americans may help to explain the lower incidence rates of AF compared to non-Hispanic whites.

2 | METHODS

2.1 | Study design

This study is a retrospective epidemiological study of AF in both inpatient and outpatients (n = 239 741), with 1 239 593 cumulative electrocardiograms (ECGs) obtained at Montefiore Medical Center between January 1, 2000 and September 8, 2013. Patients were included if their age was >45 and <95 years. Age >45 years was chosen because AF is more common with advanced age. Patients were excluded if they had AF on their initial ECG, 1 ECG, or incomplete covariate data. Patients were followed for a maximum of 10-year incidence risk.

Race/ethnicity was self-reported, and all race/ethnic categories were mutually exclusive. We used the term race/ethnicity as Hispanics are generally considered to be a multiracial group, composed largely of white, but also African American and other races.[9]

All authors report no conflict of interest, including final interests, activities, relationships or affiliations. Investigations were in accordance with the Declaration of Helsinki.

2.2 | Cohort population

Our cohort population consisted largely of an inner-city population in Bronx County, New York. Bronx County consists of about 1.4 million

TABLE 1 Baseline characteristics by race/ethnicity

	Non-Hispanic White, n = 9504, Mean, % (SD)	African American, n = 18 167, Mean, % (SD)	Hispanic, n = 20 960, Mean, % (SD)	Total, n = 48 631	*P* Value
Demographics					
Age, y	68.6 (16.6)	58.1 (17.0)	56.6 (16.8)	59.5 (17.5)	<0.01
Male	48.50%	35.50%	37.50%	38.90%	<0.01
Height, cm	164.7 (21.5)	166.1 (20.3)	161.2 (18.3)	163.7 (19.8)	<0.01
Weight, kg	77.3 (66.2)	83.8 (27.0)	77.8 (24.1)	80.0 (37.2)	<0.01
Systolic BP, mm Hg	132.0 (24.1)	135.9 (24.8)	130.8 (23.7)	133.0 (24.3)	<0.01
Diastolic BP, mm Hg	71.9 (13.9)	77.0 (15.2)	73.6 (13.9)	74.3 (14.6)	<0.01
Mean survival years	2.83 (3.04)	3.31 (3.16)	3.36 (3.21)	3.23 (3.16)	<0.01
Social economic status	−1.5 (2.9)	−3.4 (2.8)	−4.3 (2.7)	−3.4 (2.9)	<0.01
Comorbidities					
Development of atrial fibrillation	16.80%	8.70%	7.70%	9.80%	<0.01
Cardiac murmur	0.50%	1.10%	0.90%	0.90%	<0.01
Diabetes mellitus	14.50%	25.00%	26.80%	23.70%	<0.01
Heart failure	7.40%	8.90%	7.40%	8.00%	<0.01
Myocardial infarction	0.10%	0.20%	0.20%	0.20%	0.036
Smoking	0.30%	0.70%	0.90%	0.70%	<0.01
Treatment for hypertension	44.00%	59.60%	52.70%	53.60%	<0.01
ECG characteristics					
LVH	30.10%	41.90%	29.90%	34.40%	<0.01
PR interval, ms	164.52 (37.5)	162.58 (31.8)	154.89 (27.6)	159.7 (31.6)	<0.01
Medication					
β-Blockers	67.3%	61.2%	56.4%	60.3%	<0.01
Calcium channel blockers	44.1%	52.4%	40.6%	45.7%	<0.01
Digoxin	15.8%	8.8%	7.7%	9.7%	<0.01

Abbreviations: BP, blood pressure; ECG, electrocardiogram; LVH, left ventricular hypertrophy; SD, standard deviation.

Values in table denoted as mean or number (%).

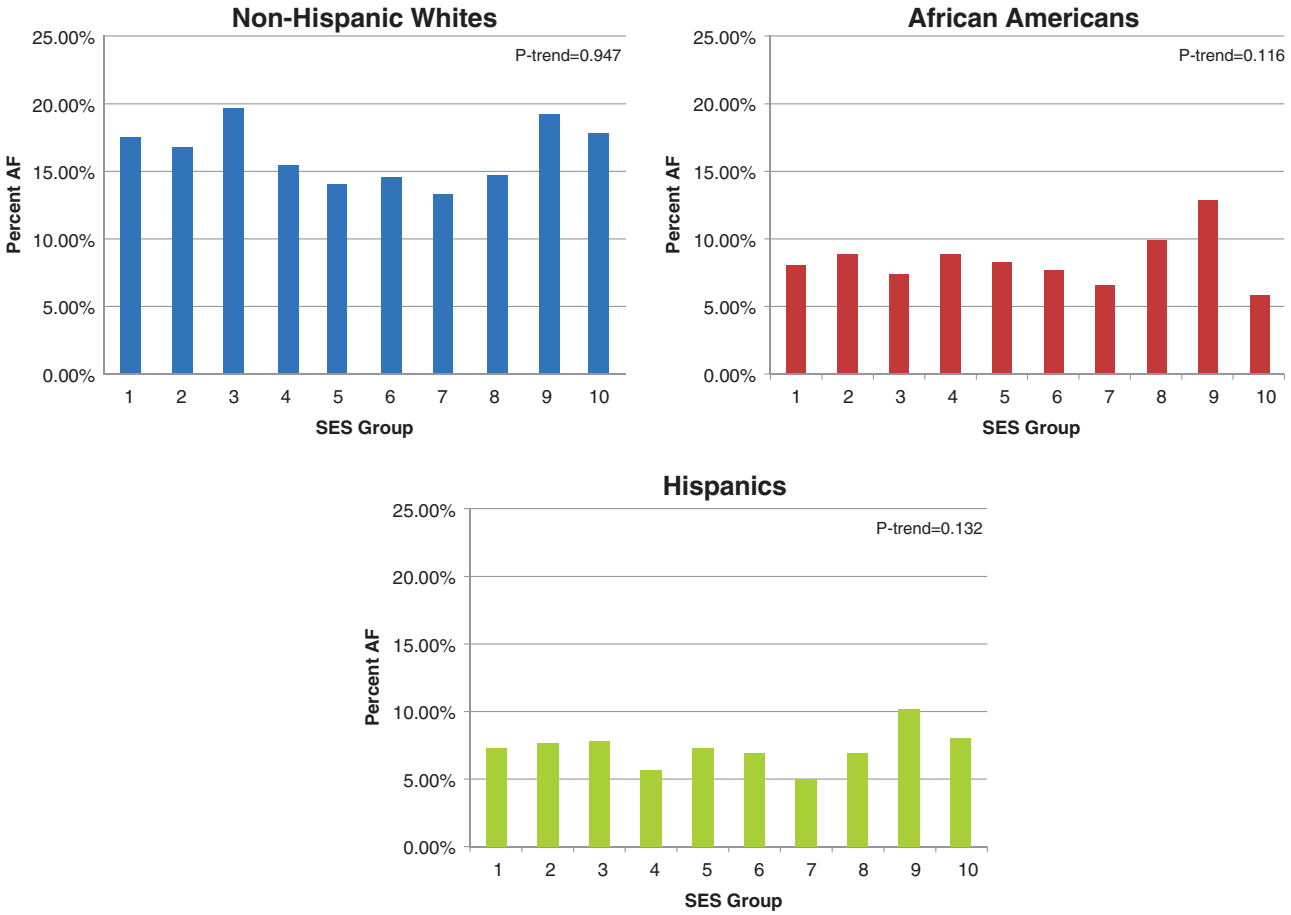

FIGURE 2 Percent of AF by SES per race/ethnic group. On the scale, 1 is lowest and 10 is highest SES. *P* trend >0.05 indicates no significant trend noted after adjusting for all baseline differences in Cox regression model. Abbreviations: AF, atrial fibrillation; SES, socioeconomic status.

individuals, with a large minority population consisting of a majority of Hispanics, followed by African Americans and then non-Hispanic whites. Bronx County is considered an underserved area, with SES variables such as high school graduation rates, higher education, and per capita income well below the national average.[9]

2.3 | SES Variable

An SES variable was calculated for each individual in the cohort. Six SES variables for each neighborhood by zip code (log of median household income; log of median value of housing units; the percentage of households receiving interest, dividend, or net rental income; education; the percentage of adults who completed college, and the percentage of employed individuals in executive, managerial, or professional positions) were normalized (*Z* scored) to the New York State average. A combined *Z* score of the 6 was calculated for each patient and reported by racial/ethnic cohort. A *Z* score of 0 is the 50th percentile of the New York State average. *Z* scores were reported as opposed to percentages to maintain a continuous distribution relative to the New York State average and for easier visual discernment, as most individuals were below the 1%. This methodology is consistent with previous literature that has used *Z*-scoring techniques to facilitate comparisons between variables, such as the Multi-Ethnic Study of Atherosclerosis (MESA) cohort.[12]

2.4 | Follow-up analysis

Follow-up started from the initial ECG. For those who developed AF, days were counted from initial normal ECG without AF to the first ECG that demonstrated AF. For those who did not develop AF, survival days were counted from initial normal ECG without AF until the last ECG without AF.

2.5 | Outcome ascertainment

Diagnosis of AF was determined by ECG. Montefiore Medical Center uses a computerized ECG system (GE Healthcare, Wauwatosa, WI) to collect, store, and analyze ECGs. This system is widely used and has been validated by the Food and Drug Administration, and meets all applicable standards for resting computerized ECG analysis.[13] The computerized system includes the 12SL program for automated ECG interpretation, which was used in this study. To determine the presence of atrial fibrillation, the 12SL algorithm looks for an irregular rhythm or fibrillatory waves without the presence of particular concurrent abnormal rhythms. The 12SL algorithm to detect AF has been validated in multiple studies,[14,15] with a reported sensitivity of 90.8% and a specificity of 98.9%.[16] All ECGs were reviewed, and diagnosis of AF confirmed by board-certified cardiologists.

The authors acknowledge that diagnosis of incident AF is via ECG, as opposed to the usual formal for incident AF diagnosis, which includes 1 diagnosis in the inpatient setting and 2 diagnoses in the

TABLE 2 Hazard ratios for different risk factors to develop atrial fibrillation

Risk Factors	HR (95% CI)	*P* Value
SES, Z-scored to NYS average	0.99 (0.98,1.00)	0.061
Race/ethnicity		
Non-Hispanic white	1.00 (reference)	
African American	0.72 (0.67-0.78)	<0.001
Hispanic	0.69 (0.64-0.75)	<0.001
Prevalent heart failure, yes	1.77 (1.64-1.91)	<0.001
Male sex, yes	1.43 (1.34-1.52)	<0.001
Age, 5 years	1.25 (1.24-1.27)	<0.001
LVH by electrocardiogram, yes	1.14 (1.08-1.21)	<0.001
Diabetes, yes	1.06 (0.99-1.13)	0.100
Height, 10 cm	1.02 (1.00-1.03)	0.010
Weight, 15 kg	1.01 (1.00-1.01)	<0.001
PR interval on first ECG, ms	1.00 (1.00-1.01)	<0.001
Diastolic BP, 10 mm Hg	0.99 (0.96-1.02)	0.410
Systolic BP, 20 mm Hg	0.99 (0.97-1.00)	0.130
Antihypertensive medication use, yes	0.74 (0.69-0.78)	<0.001
Presence of murmur, yes	0.72 (0.51-1.01)	0.060

Abbreviations: BP, blood pressure; CI, confidence interval; ECG, electrocardiogram; HR, hazard ratio; LVH, left ventricular hypertrophy; NYS, New York State; SES, socioeconomic status.

Hazard ratios for various risk factors in multivariable Cox Regression model controlling for SES, age, gender, height, weight, systolic BP, diastolic BP, presence of murmur, diagnosis of diabetes mellitus, diagnosis of heart failure, treatment for hypertension, LVH by electrocardiogram, PR interval on initial ECG, and race/ethnicity.

TABLE 3 *P* interaction values

Variable	*P* Interaction With SES
Race/ethnicity	0.43
Non-Hispanic white	Reference
African American	0.27
Hispanic	0.27
Prevalent heart failure, yes	<0.01
Male sex, yes	0.38
Age, 5 years	0.60
LVH by electrocardiogram, yes	0.17
Diabetes, yes	0.12
Height, 10 cm	0.59
Weight, 15 kg	<0.01
First PR Interval, ms	0.53
Diastolic BP, 10 mm Hg	0.01
Systolic BP, 20 mm Hg	<0.01
Antihypertensive medication use, yes	0.19
Presence of murmur, yes	0.91

Abbreviations: BP, blood pressure; DM, diabetes mellitus; ECG, electrocardiogram; HF, heart failure; LVH, left ventricular hypertrophy; SES, socioeconomic status.

P interaction values of SES in Cox regression model controlling for race/ethnicity, HF, gender, age, LVH by ECG, DM, height, weight, first PR interval, diastolic BP, systolic BP, antihypertensive treatment and presence of murmur.

outpatient setting. The authors believe that diagnosis of AF via ECG provides a higher level of certainty and timing of diagnosis of AF as opposed to inpatient claim data.

All other clinical variables were extracted by searching the electronic medical records (EMR) system. All variables were obtained via EMR query during the study dates. These variables were closer to the initial, as opposed to subsequent ECGs.

2.6 | Statistical analysis

Descriptive statistics were produced for each cohort. Unpaired 2-sided *t* tests were used for the comparisons of continuous variables, and χ^2 tests were used to compare dichotomous variables between patients. Analysis of variance (ANOVA) was performed between continuous variables. Statistical significance was defined by $P < 0.05$.

Cox regression analysis controlled for all baseline differences, excluding current medication use, as nodal blocking agents may alter ECG characteristics utilized in regression analysis and medication use may not have been at initial baseline, which estimated the independent predictive ability of SES as a continuous variable for AF. Baseline differences included age, gender, height, weight, systolic blood pressure (BP), diastolic BP, presence of murmur, diagnosis of diabetes mellitus, diagnosis of heart failure, treatment for hypertension, left ventricular hypertrophy (LVH) by ECG, PR interval on initial ECG, and race/ethnicity. *P* interaction between SES and all covariates were iteratively calculated to determine if there were significant interactions. Cox regression controlled for above-mentioned covariates was then rerun by race/ethnicity to determine race/ethnicity specific hazard ratio (HR) for each covariate for the determination of AF.

SES was then reclassified into decile groups (1 lowest and 10 highest). The log-rank test was used to determine differences in survival times to develop AF by race/ethnicity stratified by SES decile. Cox regression was then run again that controlled for all above-mentioned covariates using SES as a categorical variable. *P* trend was calculated by race/ethnicity to determine if there was a statistical trend, after adjusting for all above-mentioned covariate differences by SES decile, stratified by race/ethnicity to develop AF. This was performed by calculating the median *Z* score of each above-mentioned decile group by race/ethnicity. Each patient was assigned the respective median *Z*-score value per race/ethnic groups' SES decile. A Cox regression analysis was then performed utilizing this additional covariate. The authors believe that this methodology, which compares equivalent socioeconomic classes to each other among racial/ethnic groups, would limit potential bias in healthcare access.

SPSS version 22.0 (IBM, Armonk, NY) and R Studio version 0.98.507 (The R Foundation for Statistical Computing, Vienna, Austria) was used for all statistical analysis. Proportional hazard assumptions were met as verified by plotting the Schoenfeld residuals. Likelihood ratio test was then computed, which compared the original regression model to a regression model that included the interaction term between SES and race/ethnicity.

3 | RESULTS

In total, 48 631 patients met inclusion criteria, of whom 4556 developed AF (Figure 1). Baseline characteristics are shown in Table 1. Our

cohort totaled 161 454 person-years of follow-up (mean = 3.32 years). In general, non-Hispanic whites tended to be older, had a higher percent of males, and had a higher incidence of AF compared to other races/ethnicities. Medication use differed with non-Hispanic whites with higher use of β-blockers and digoxin. Also, in general, African Americans and Hispanics had almost twice the rate of diabetes compared to non-Hispanic whites, and African Americans had the highest rates of heart failure and treatment for hypertension compared to all races. Incidence of AF was 9.8%.

SES differences were statistically different between all racial/ethnic cohorts and appeared to have a bimodal distribution in all race/ethnicities (Table 1, Figure 2). Non-Hispanic whites had the highest and Hispanics had the lowest SES. SES ranged from −13.5 to 3.8, with a non-Hispanic white range of −9.5 to 3.9, African American range of −10.0 to 3.5, and Hispanic range from −13.6 to 3.5. Hispanics and African Americans had lower AF risk than whites in all SES deciles (log-rank test *P* value <0.001).

Table 2 demonstrates hazard ratios for the independent predictive ability of each covariate for AF. Higher SES was borderline associated with lower AF risk (HR = 0.99, 95% confidence interval [CI]: 0.980-1.00, *P* = 0.061). Risk factors for AF included the presence of heart failure, male gender, then age. Hispanics and African Americans maintained statistically lower HRs compared to non-Hispanic Caucasians for the development of AF. *P* interactions are available in Table 3.

The SES variable, stratified by race/ethnicity, was not predictive for AF in non-Hispanic whites and African Americans. However, there appeared to be a borderline association for a higher SES to be less predictive of AF in Hispanics (HR: 0.98, 95% CI: 0.97-1.00, *P* = 0.078).

In comparison to the lowest SES group (group 1), groups 4, 5, and 7 had a statistically significant lower HR to develop AF: decile 4 (HR: 0.85, 95% CI: 0.75-0.95, *P* = 0.005); decile 5 (HR: 0.89, 95% CI: 0.79-0.999, *P* = 0.036), and decile 7 (HR: 0.83, 95% CI: 0.72-0.96, *P* = 0.011). Figure 2 demonstrates the percent of AF by SES decile, stratified by race/ethnicity. *P* trend was not significant by any race/ethnic group for a trend among SES deciles and AF (*P* trend for all overall cohort = 0.199; *P* trend for non-Hispanic whites = 0.947; *P* trend for African Americans = 0.116; and *P* trend for Hispanics = 0.132).

Likelihood ratio test comparing the original Cox regression model utilizing SES as a continuous variable to one with an interaction term between SES and race/ethnicity, resulted in $\chi^2 = 4$ (2 degrees of freedom) and *P* value of 0.135.

4 | DISCUSSION

This is a large-scale retrospective study of the interaction between race/ethnicity and SES for the development of AF in a racially and socioeconomically diverse inner-city population. We demonstrated that there is a trend for significance for individuals with a higher SES score to have a lower risk of AF, and that non-Hispanic whites had higher rates of AF compared to African Americans and Hispanics. Our study suggests that non-Hispanic whites are at higher risk for AF compared to nonwhites, and this is independent of SES.

Our results extend prior investigations that there exists racial/ethnic differences in the risk factors for and incidence of AF.[1,17–21] In our inner-city population, non-Hispanic whites continued to have the highest incidence of AF, compared to African Americans and Hispanics. Moreover, the distribution of risk factors for AF varied by race/ethnicity. For instance, African Americans had a higher incidence of heart failure and treatment of hypertension compared to non-African American cohorts, confirmed by earlier reports.[20,22] Of note, heart failure, closely followed by treatment for hypertension, also remained the strongest risk factor to predict AF. Moreover, our study continued to confirm the racial paradox by demonstrating that though non-Hispanic whites are at a higher SES, they continued to have significantly elevated rates of AF compared to African Americans and Hispanics. Interestingly, the MESA study concluded that a larger proportion of AF events appear to be attributable to hypertension among nonwhite populations compared with non-Hispanic whites,[23] which was confirmed in the Reasons for Geographic and Racial Differences in Stroke (REGARDS) study.[24] However, these articles, in addition to others, further conclude that these differences in risk factors do not explain the racial paradox in AF as well.

It is interesting that neither the elevation of BP nor the presence of diabetes were associated with incident AF in this study. This is inconsistent with previous literature. The authors believe that the effects of BP (systolic or diastolic) may have been tempered by the use of antihypertensive agents, which showed a beneficial effect against developing AF. The presence of diabetes is also a considered a risk factor for AF, but also may have been tempered by the effects of diabetic medication and subsequent control of patients' blood sugars. We also note that particular SES deciles (4, 5, and 7) were protective of incident AF. This is interesting, and is likely due to the bimodal distribution of incident AF plotted against SES. Deciles 4, 5, and 7 likely fall in the nadir of incident AF. In addition, racial differences in AF incidence by levels of SES were assessed. Previous studies have shown that the incidence of AF is lower in African Americans than whites.[20,23–25] However, lower SES among minorities and subsequent reduced access to healthcare[26] may only partly explain this association. This assumes that underdiagnosis of AF occurs more frequently among those with low SES.[11] In our analysis, African Americans and Hispanics had lower rates of AF than non-Hispanics whites in each SES decile group, and there was a similar trend in the association between lower SES and higher AF risk for our entire cohort and in each racial/ethnic group, though these were not statistically significant. These results suggested that lower SES in African Americans and Hispanics compared with non-Hispanic whites may not have been a major determinant of the demonstrated racial/ethnic differences in AF incidence. Our findings that SES does not influence the relation between ethnicity and AF is further supported with no significant difference between either our original regression model or model with an interaction term between SES and race/ethnicity. Further validating our conclusions includes an analysis of the ARIC study data investigating risk factors of atherosclerosis and cardiovascular disease, which also found that differences in SES did not

explain the lower risk of AF in African Americans compared with whites.[5]

Given the concurrently higher incidence of AF and paradoxically lower risk burden for AF, we hypothesized that there exists an unexplained risk factor in whites compared to nonwhites for the development of AF. Marcus et al extended the possibility that there exists genetic effects by showing that an increased percent of European ancestry within African Americans significantly increased the risk of AF.[17]

4.1 | Strengths/limitations

We acknowledge several limitations to our study. First, it is possible that patients were enrolled in our study with a previous diagnosis of AF, but did not demonstrate an irregular rhythm on ECG and were misclassified. We acknowledged that we did not exclude initial diagnosis of AF via patients' personal medical history nor query medical claims data to ascertain AF. Second, a large segment of our population was also excluded due to missing complete patient data (mostly consisting of height/weight), which could bias our results and may be biased by healthcare access. Third, several of our risks factors (including systolic BP and diabetes) were not predictive of incident AF, which is contrary to the literature. Only patients who declared race/ethnicity as determined by their medical record were enrolled, which provided a potential for further bias. We may have also missed patients whose diagnosis of AF was via other forms of cardiac monitoring (ie, Holter or telemetry monitoring) other than ECG. Our data may not be generalizable to many populations, and may be less valid for higher SES categories.

5 | CONCLUSION

To our knowledge, this is the first study to investigate the interaction between SES and race/ethnicity for the development of AF in a non-Hispanic white, African American, and Hispanic cohort. We demonstrated that there is a tendency for individuals with a higher SES score to have a lower risk of AF, and that non-Hispanic whites had higher rates of AF compared to African Americans and Hispanics. However, our study suggested that non-Hispanic whites are at higher risk for AF compared to nonwhites, and that this is independent of SES.

Conflicts of interest

The authors declare no potential conflict of interests.

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