Depressed autonomic nervous system function in African Americans and individuals of lower social class: A potential mechanism of race- and class-related disparities in health outcomes

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Background Both race and social class influence cardiovascular outcomes, through mechanisms not yet fully understood. Minority race and lower social class are sources of chronic stress, which can alter autonomic nervous system function. Heart rate variability (HRV), a measure of autonomic function, is also an important predictor of cardiovascular outcomes.

Methods To determine whether minority race/ethnicity and lower social class are associated with depressed HRV, we prospectively collected data on sociodemographic, clinical, psychological, and behavioral factors by survey in 360 outpatients undergoing ambulatory electrocardiographic monitoring. Heart rate variability (24-hour) was measured by frequency domain analysis.

Results In unadjusted analysis, African Americans had lower HRV than whites, and individuals of lower social class as measured by education, occupation, and income had lower HRV than those of higher class. In multivariable analysis, both race and social class were independent predictors of ultralow frequency power after controlling for clinical and psychological factors. African Americans were 3.45 (95% CI 1.74-6.98, $P = .0004$) times as likely as whites to have depressed HRV (ultralow frequency power, lowest tertile), and non–college graduates 2.94 (95% CI, 1.71-5.14, $P = .0001$) times as likely as college graduates to have depressed HRV.

Conclusions Heart rate variability is lower in African Americans and individuals of lower social class, independent of the effects of measured clinical, psychological, or behavioral factors. This suggests that the adverse effects of minority race and lower social class on cardiovascular outcomes may be mediated by dysregulation of autonomic function. (Am Heart J 2005;150:153-60.)

Both race and social class influence cardiovascular outcomes. African Americans suffer greater cardiovascular morbidity and mortality at every age.[1,2] Sudden cardiac death rates are higher, survival from arrest lower,[3] and prognosis after myocardial infarction worse than for whites.[4] Similarly, lower social class confers worse survival, most dramatically in the United States among Western countries.[5] Decreasing education,[6-8] occupational status,[8,9] and income[6,10] are each related to mortality, morbidity, and poor functional status. Differences in access to health care, comorbidities, psychological factors, such as depression, and health-related behaviors, such as smoking, contribute to the increased mortality in individuals of minority race or lower social class.[11] However, these factors are not sufficient to explain the impact of race and social class on cardiovascular outcomes.[2,8,9]

How race and lower social class "get under the skin" to alter cardiovascular outcomes remains unknown. Many environmental factors associated with minority race and lower social class produce a chronic state of stress.[12] We hypothesized that, similar to the effects of other psychological stressors,[13-16] the chronic stress imposed by minority group status and lower social class adversely alters autonomic nervous

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Table IA. Demographic and clinical characteristics: Race

	White, n = 215	Hispanic, n = 26	P*	African American, n = 41	P*	P†
Age	50 ± 1	40 ± 3	.003	45 ± 3	.12	.08
Male	102 (48%)	9 (36%)	.25	10 (25%)	.006	.32
Social class						
Non–college graduate	99 (47%)	22 (85%)	.0002	33 (81%)	<.0001	.66
Manual/clerical occupation	35 (21%)	7 (54%)	.016	18 (54%)	.0002	.96
Income ≤ $70000/y	111 (63%)	26 (100%)	.013	24 (88%)	.009	.25
Charlson comorbidity score	0.58 ± .07	0.73 ± .21	.49	0.93 ± .17	.065	.56
Hypertension	49 (23%)	4 (15%)	.38	17 (41%)	.016	.025
Cancer	20 (9%)	1 (4%)	0.38	5 (12%)	.57	.25
Diabetes	17 (8%)	4 (15%)	.24	6 (15%)	.19	.93
Angina	18 (8%)	3 (11%)	.60	5 (12%)	.45	.94
Heart attack	17 (8%)	1 (4%)	.46	3 (7%)	.89	.55
Congestive heart failure	11 (5%)	2 (8%)	.60	3 (7%)	.57	.95
Renal disease	8 (4%)	2 (8%)	.38	3 (7%)	.33	.95
Vascular disease	6 (3%)	0	.24	1 (2%)	.89	.42
Stroke	7 (3%)	1 (4%)	.87	6 (14%)	.009	.14
Neurologic disease	5 (2%)	3 (11%)	.042	3 (7%)	.13	.56
Valvular disease	5 (2%)	1 (4%)	.66	1 (2%)	.96	.74
Liver disease	4 (2%)	1 (4%)	.54	0	.66	.20
HIV	1 (0.5%)	1 (4%)	.16	0	.66	.20
Medication use						
β-Blockers	20 (14%)	2 (13%)	.82	4 (21%)	.47	.50
ACE-I/ARB	18 (13%)	1 (6%)	.39	3 (16%)	.74	.37
Holter variables						
Referring physician			.17		.55	.35
Internist/other	127 (62%)	21 (80%)		24 (68%)		
Cardiologist	76 (37%)	5 (19%)		11 (31%)		
Indication			.63		.07	.012
Palpitations	87 (43%)	13 (50%)		20 (57%)		
Syncope/dizziness	39 (19%)	3 (12%)		6 (17%)		
Arrhythmia history	39 (19%)	7 (27%)		1 (2%)		
Heart disease	25 (12%)	2 (8%)		3 (9%)		
Other	14 (7%)	1 (4%)		5 (14%)		
Findings						
PVCs/ 24 h	9 (2-197)	2 (0-41)	.036	8 (0-32)	.13	.47
PACs/ 24 h	16 (5-70)	4 (1-16)	.008	11 (1-63)	.19	.13
Psychological/behavioral factors						
Depression score	11.46 ± .29	14 ± .85	.018	12.78 ± .66	.065	.24
Current smoker	14 (7%)	5 (19%)	.023	13 (31%)	<.0001	.25
Exercise ≥3 d/wk	109 (54%)	10 (40%)	.17	18 (48%)	.51	.50

system (ANS) function and thereby increases vulnerability to cardiac events. Autonomic nervous system function can be noninvasively measured by methods of heart rate variability (HRV) analysis,[17,18] which quantifies the beat-to-beat changes in heart rate caused by changes in sympathetic and vagal activity.[17,18] Depressed HRV is an important predictor of cardiovascular outcomes, correlating with increased mortality in the general population[19,20] as well as poor prognosis after myocardial infarction.[21,22] Autonomic dysregulation may thus provide a physiologic pathway through which race and social class influence cardiovascular outcomes.

In this study of outpatients undergoing ambulatory electrocardiogram (Holter), we prospectively collected clinical and demographic data and analyzed HRV to (1) determine whether race/ethnicity and social class are associated with an impairment in ANS function as measured by HRV; (2) determine whether these factors influence HRV independent of clinical, psychological, and behavioral factors; and (3) quantify the risk of depressed HRV associated with minority race and low social class.

Methods

Study participants

Outpatients referred to the Yale New Haven Hospital Holter laboratory for clinically indicated Holter monitoring between October 1998 and March 2000 were screened (N = 419).

Table IB. Demographic and clinical characteristics: Social class

	College graduate, n = 127	Non–college graduate, n = 154	*P*
Age	48 ± 1	49 ± 1	.68
Male (%)	66 (52%)	55 (36%)	.006
Race			<.0001
White	115 (90%)	99 (64%)	
Hispanic	4 (3%)	22 (14%)	
American	8 (6%)	33 (21%)	
Charlson comorbidity score	.53 ± .10	.73 ± .09	.13
Hypertension	26 (20%)	43 (27%)	.15
Cancer	12 (9%)	14 (9%)	.92
Diabetes	7 (6%)	20 (13%)	.03
Angina	10 (8%)	15 (10%)	.58
Heart attack	6 (5%)	15 (9%)	.11
Congestive heart failure	5 (4%)	11 (7%)	.24
Renal disease	6 (5%)	7 (5%)	.94
PVD	4 (3%)	3 (2%)	.52
Stroke	3 (2%)	11 (7%)	.067
Neurologic disease	3 (2%)	5 (8%)	.21
Valvular disease	3 (2%)	4 (3%)	.90
Liver disease	2 (2%)	2 (1%)	.85
HIV	1 (1%)	1 (1%)	.89
Medication use			
β-Blockers	13 (15%)	13 (15%)	.93
ACE-I/ARB	8 (9%)	14 (16%)	.14
Holter variables			
Referring physician			.88
Internist/other	75 (64%)	96 (65%)	
Cardiologist	42 (36%)	50 (34%)	
Indication			.061
Palpitations	57 (45%)	63 (41%)	
Syncope/dizziness	17 (13%)	30 (19%)	
Arrhythmia history	27 (21%)	20 (13%)	
Heart disease	8 (6%)	22 (14%)	
Other	18 (14%)	19 (12%)	
Findings			
PVCs/ 24 h	8 (1-194)	8 (1-95)	.75
PACs/ 24 h	17 (4-68)	3 (0-64)	.36
Psychological/behavioral factors			
Depression	11 ± 0.4	13 ± 0.3	.0005
Current smoker	3%	18%	<.0001
Exercise ≥ 3 d/wk	59%	46%	.03

Values represent mean ± SE, N (percentage), or median (interquartile range). *ACE I*, Angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; *PVC*, premature ventricular contraction; *PAC*, premature atrial contraction.
**P* as compared with whites.
†*P* value for comparision between Hispanics and African Americans.

Forty-four patients (10%) were excluded because of atrial fibrillation (n = 17), paced rhythms (n = 18), or language other than English or Spanish (n = 9). The remaining 375 were invited to participate, of whom 15 (4%) refused. Of these participating 360 patients, 316 had Holter studies suitable for analysis. Patients with inadequate Holter recordings did not differ from included patients by race or social class. The 282 non-Hispanic whites, non-Hispanic African Americans, and Hispanics with adequate Holter recordings were included in the final analysis (13 Asians and 21 patients of other or unreported race were excluded).

Study design

Participants completed the study survey before undergoing 24-hour Holter recordings, which were later analyzed for HRV. This study was approved by the Yale Human Investigation Committee and all patients gave informed consent.

Survey

Race/ethnicity and social class. Patients reported race/ethnicity as Caucasian/White, African American/Black, Asian/Pacific Islander, Latino/Hispanic, or Other. Stratified measures of social class included education, occupation, and income. Education was dichotomized for analysis based on graduation from college or not. A 6-level revised Hollingshead categorization of occupation[23] was used and then dichotomized into manual/clerical or professional/managerial for analysis. Income was defined as the total household income after taxes and later dichotomized at the highest tertile, $70 000.

Clinical factors. Chronic illness was measured using the Charlson comorbidity index.[24] Patients reported having "been told by a doctor" of each of 14 diagnoses and listed current medications.

Psychological and behavioral factors. Depressive symptoms were measured using a 7-item questionnaire derived from the Center for Epidemiology Studies Depression scale,[25] which asks about frequency of symptoms in the last week. Other questions included tobacco use and exercise (days per week). Scores for depression and exercise were stratified at the median.

Processing of Holter recordings and HRV analysis

Holter recordings were digitally sampled and analyzed using a Zymed system (Oxnard, Calif). Each tape was manually processed and edited. A list of R-R intervals with annotations denoting normal beats, types of ectopics, and noise was processed and analyzed with customized software. The R-R interval data file was edited to remove ectopics and noise, and gaps filled in by interpolated linear splines. Those Holter recordings with >20% interpolated segments or <18 total recorded hours were excluded from further analysis (n = 73). The power spectrum was computed using a fast Fourier transform with a Parzen window and corrected for attenuation due to windowing and sampling. The power spectrum was integrated over 5 discrete frequency bands as defined by Bigger et al[22]: ultralow frequency (ULF) <0.0033 Hz; very low frequency (VLF) 0.0033 to <0.04 Hz; low frequency (LF) 0.04 to <0.15 Hz; and high frequency (HF) 0.15 to 0.40 Hz. The standard deviation of N-N intervals (SDNN) was computed.

Statistical analyses

All HRV parameters except SDNN were highly skewed and therefore natural log transformed. Bivariate associations of all variables with HRV parameters were performed and adjusted for age with standard least squares linear regression.

To evaluate the independence of associations of race/ethnicity and social class with HRV, multivariable models including all Table I variables (except Charlson score, a derived variable), race, and education were created and linear regression performed (stepwise regression with forward selection) for each HRV variable. To quantify the relative likelihood of depressed HRV associated with minority race and low social class while controlling for other risk factors, multivariable logistic regression was performed using a series of models including race, education, age, sex, and those clinical, psychological, and behavioral variables with significant bivariate associations with each HRV parameter. Odds ratios were calculated for depressed ULF and SDNN, the HRV variables most closely correlated with adverse cardiovascular disease outcomes.[20,22] Depressed ULF and SDNN were defined as the lowest tertile for each.

For multivariable analysis, only African American or white patients were included, as the Hispanic subgroup (26) was too small. We selected educational level as the indicator of social class because it had the highest survey response rate and has the strongest correlation with health outcomes.[7]

All analyses were performed using JMP 5.0 software (SAS Institute, Cary, NC).

Results

Participants

The study population included the 282 individuals meeting all study criteria. Mean age was 48 ± 17 years, 43% were men. Most participants were healthy; mean Charlson score was 0.64 ± 1.13. The population was 76% white (N = 215), 15% African American (N = 41), and 9% Hispanic (N = 26). Fifty-five percent (N = 154) had not graduated from college, 29% (N = 60) worked in manual or clerical occupations, and 69% (N = 146) had an income ≤ $70,001 per year.

Comparisons of sociodemographic, clinical, psychological, and behavioral characteristics between whites, Hispanics, and African Americans are shown in Table IA, and between college graduates and nongraduates in Table IB. Whites reported lower rates of most diseases and of smoking and had marginally lower Charlson score and use of β-blockers than African Americans. Whites scored higher on all measures of social class than the other groups. College graduates were more likely to be white and male, reported marginally lower rates of diseases and had lower Charlson score, and were significantly less likely to report depression, smoking, or low exercise level than nongraduates. There were no differences by race or education in type of referring physician (predominantly internists) or in indication for Holter monitoring (most for palpitations or syncope/dizziness).

Association of social class, race/ethnicity, clinical, psychological, and behavioral factors with HRV parameters

Age-adjusted bivariate associations of all variables with HRV are shown in Table II. Highest HRV for each parameter was seen in whites, followed by Hispanics, then African Americans (Table II and Figure 1.) Significant differences were seen between whites and African Americans for each parameter, and between whites and Hispanics for ULF and SDNN. Heart rate variability parameters did not differ significantly between African Americans and Hispanics.

Higher social class as measured by education, occupation, and income was associated with higher values of all HRV parameters, with all but HF reaching statistical significance (Table II). Effect of education (Figure 2) and income was graded: as these measures of social class increased so too did age-adjusted HRV.

Among other variables, current smoking and depression were significantly correlated with all of the HRV parameters, and exercise correlated with ULF and SDNN (Table II). Age was inversely associated with VLF, LF, and HF ($P < .01$ for each), and Charlson score was inversely associated with all parameters ($P < .001$ for each). Heart rate variability did not differ between men and women (data not shown).

Table II. Bivariate associations for all HRV parameters (age-adjusted)

	ln ULF	*P*	ln VLF	*P*	ln LF	*P*	ln HF	*P*	SDNN	*P*
Race/ethnicity*										
White	9.13 ± .11		6.99 ± .08		6.13 ± .21		5.25 ± .09		115 ± 2.7	
Hispanic	8.65 ± .15	.003	6.83 ± .23	.51	5.83 ± .26	.29	4.90 ± .25	.20	90 ± 7.8	.001
African American	8.54 ± .05	<.0001	6.37 ± .18	.003	5.43 ± .09	.003	4.71 ± .20	.014	86 ± 6.2	<.0001
Social class factors										
Education										
Non–college graduate	8.78 ± .06	<.0001	6.64 ± .09	<.0001	5.73 ± .11	.0002	4.97 ± .10	.02	98 ± 3.2	<.0001
College graduate	9.26 ± .17		7.20 ± .10		6.33 ± .12		5.33 ± .11		122 ± 3.5	
Occupation										
Manual/clerical	8.86 ± .09	.029	6.61 ± .14	.004	5.63 ± .15	.002	4.90 ± .16	.15	100 ± 5.1	.001
Professional/ managerial	9.11 ± .06		7.08 ± .09		6.22 ± .10		5.17 ± .10		114 ± 3.2	
Income										
<$70 000/y	8.94 ± .06	.003	6.77 ± .09	.003	5.90 ± .11	.009	5.04 ± .11	.18	105 ± 3.2	.001
≥$70 000/y	9.27 ± .09		7.28 ± .14		6.42 ± .16		5.31 ± .16		123 ± 4.7	
Psychological/behavioral factors										
Depression										
Yes	8.81 ± .07	.0004	6.56 ± .09	<.0001	5.64 ± .10	<.0001	4.86 ± .11	.002	98 ± 3.6	<.0001
No	9.15 ± .06		7.14 ± .10		6.28 ± .11		5.35 ± .10		117 ± 3.21	
Smoking										
Yes	8.63 ± .14	.005	6.41 ± .20	.02	5.52 ± .24	.04	4.53 ± .23	.006	86 ± 7.2	.001
No	9.05 ± .05		6.95 ± .07		6.06 ± .08		5.21 ± .08		112 ± 2.3	
Exercise (>3 d/wk)										
No	8.86 ± .07	.007	6.84 ± .11	.79	5.99 ± .12	.76	5.03 ± .12	.19	101 ± 3.7	.001
Yes	9.13 ± .07		6.94 ± .10		6.04 ± .12		5.24 ± .11		117 ± 3.6	

All values represent mean ± SE.
**P* values compared to whites. Heart rate variability variables did not differ between African Americans and Hispanics.

Multivariable analyses: Association of race and social class with HRV variables, controlling for clinical, psychological, and behavioral factors

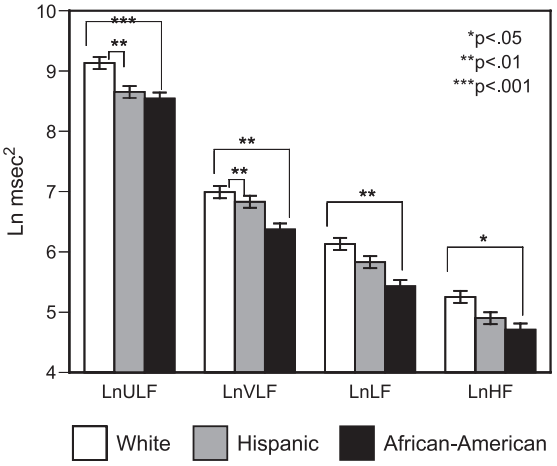
Multivariable models were created including all variables listed in Table I, and linear regression performed, for each HRV variable. Race and education were independently associated with ULF and SDNN. Race was also independently associated with LF. Among the other variables, congestive heart failure and use of angiotensin-converting enzyme inhibitors were independently associated with all HRV parameters. There was no interaction between race and social class in this analysis.

To quantify likelihood of depressed HRV, defined as the lowest tertiles of ULF and of SDNN, associated with minority race and low social class, odds ratios were computed using multivariable logistic regression in a series of models: race or education alone (model 1), both race and education (model 2), and further controlling incrementally for clinical, psychological, and behavioral factors (models 3-5, respectively). African Americans had 3.45 times the likelihood of depressed ULF as whites (95% CI 1.74-6.98, $P = .0004$, Table IIIa). Although the addition of education to the model attenuated the association of race and HRV, this remained highly significant, with an odds ratio of 2.50 (95% CI 1.20-5.20, $P = .011$). Addition of clinical, psychological, and behavioral factors to the model had minimal effect on the strong relationship of race and ULF. Non–college graduates had 2.94 times the likelihood of depressed HRV (95% CI 1.71-5.14, $P < .001$, Table IIIb). Race, comorbidity, depression, and behavioral factors (smoking and exercise) explained part but not all of the association between education and HRV (OR for education in the final model 2.10, 95% CI 1.13-3.95, $P < .05$.) Findings for SDNN were similar. In the final model, African Americans were 2.48 times as likely as whites (95% CI 1.11-5.63, $P = .03$) and non–college graduates 2.03 times as likely as college graduates (95% CI 1.10-3.80, $P = .03$) to have depressed SDNN.

Discussion

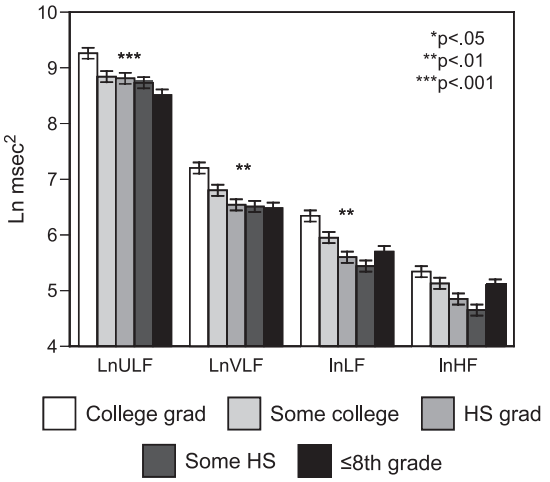
In this study, all HRV parameters were lower in African Americans than whites, with African Americans >3 times as likely to have depressed ULF, a strong predictor of cardiovascular mortality. The HRV of Hispanic individuals fell between that of African Americans and whites. Individuals of lower social class, as

Figure 1



Effect of race/ethnicity on HRV. Whites had significantly higher ULF, VLF, LF, and HF power than did African Americans and significantly higher ULF and VLF than Hispanics. Heart rate variability did not differ between African Americans and Hispanics.

Figure 2



Effect of educational level on HRV. Increasing educational level was significantly associated with increased ULF, VLF, and LF (χ^2 for linear trend).

measured by education, occupational status, or income, demonstrated lower HRV. These effects of race and social class were independent of other known health-modifying risk factors measured in this study.

Physiologic studies have demonstrated that HRV reflects modulations of parasympathetic and sympathetic tone.[17,18] The consistent depression of indices of HRV shown here suggests that a state of ANS dysfunction exists in individuals of minority race/ethnicity or lower social class.

How race and class might affect autonomic function is not known. Many factors known to lower HRV were more prevalent in African Americans and individuals of lower social class, including greater comorbidities, depression, and health-damaging behaviors. However, race and social class remained independently correlated with depressed HRV after controlling for these factors. There are several plausible biologic and environmental explanations for these findings. First, the possibility that racial/ethnic minorities and individuals of lower social class have preclinical or undiagnosed disease, which would not be captured by self-report, cannot be excluded. However, the consistent and graded HRV differences across different stratifications of social class suggest a second hypothesis, that the explanation may lie in environmental stresses created by subordinate social status. Although the measured indicators of social class did not explain the differences in HRV between African Americans and whites, the unique daily challenges facing many minority communities, such as population density, crime rate, exposure to environmental hazards, or social injustices, have been described as sources of chronic stress.[1,12] Other chronic stressors such as work stress, anxiety, and depression[13-16] lower HRV. Thus, it is possible that heightened sympathetic arousal in response to the daily societal stresses of minority race may over time lead to chronic dysregulation of autonomic function.[26]

The ability of physiologic systems to adapt to stressors, or "achieve stability through change," has been described as "allostasis."[27] According to the allostasis paradigm, as discussed by McEwen et al, "health functioning requires ongoing adjustments of internal physiologic milieu, with physiologic systems exhibiting fluctuating levels of activity as they respond and adapt to environmental demands."[27] Excessive stresses to the system, or increased allostatic load, result in "wear and tear of the regulatory systems," which has been shown to correlate with increased mortality.[27] It has been suggested that HRV may provide an integrated measure of allostasis.[28] Thus, the lower HRV seen in individuals of minority race and lower social class may reflect increased allostatic load, resulting in this "wear and tear," thereby dampening neurohormonal regulatory functions and increasing vulnerability to disease.

Finally, it is possible that genetic differences underlie the racial differences in HRV seen in this study. Further investigation is warranted to evaluate the mechanisms through which race and social class may alter autonomic function.

Depressed indices of HRV predict morbidity[19] and mortality[19,20] in normal individuals, as well as patients

Table III.

a. Risk of depressed HRV: race

	African American, compared to white	
	Odds ratio (95% CI)	***P***
Model 1: race	3.45 (1.74-6.98)	.0004
Model 2: model 1 + education (sociodemographic)	2.50 (1.21-5.20)	.011
Model 3: model 2 + Charlson (clinical)	2.22 (1.06-4.68)	.034
Model 4: model 3 + depression (psychological)	2.16 (1.03-4.38)	.041
Model 5: model 4 + smoking, exercise (behaviors)	2.44 (1.09-5.56)	.030

b. Risk of Depressed HRV: social class

	Non–college graduates, compared with college graduates	
	Odds ratio (95% confidence interval)	***P***
Model 1: education	2.94 (1.71-5.14)	<.001
Model 2: model 1 + race (sociodemographic)	2.38 (1.35-4.27)	.003
Model 3: model 2 + Charlson (clinical)	2.37 (1.34-4.27)	.003
Model 4: model 3 + depression (psychological)	2.24 (1.24-4.07)	.007
Model 5: model 4 + smoking, exercise (behaviors)	2.10 (1.13-3.95)	.02

after myocardial infarction.[21,22] Altered ANS function may directly contribute to cardiac mortality, as sympathetic hyperactivity and decreased vagal tone increase cardiac workload, predispose to arrhythmia,[26,29] and alter endothelial function,[30] enhancing atherosclerosis. Or, HRV may be a more general indicator of neurohormonal function, impacting broadly on health status and outcomes. Depressed ULF and SDNN, the indices most strongly correlated with race and social class in this analysis, are the indices most predictive of mortality.[20,21] Changes in autonomic function may thus be physiologic mediators of the effects of race and social class on cardiovascular outcomes.

Few prior studies have evaluated the effects of race and social class on HRV and none have evaluated the interaction. In the ARIC study,[31] LF was lower in African Americans than whites, but HF was higher. Some,[32,33] but not all,[13] small studies have found social class to correlate with short-term measures of HRV. These studies however have not consistently controlled for psychological factors, nor evaluated the lower HRV frequencies, which showed the strongest associations with race and class in this study. Heart rate variability has been shown independent of race in patients with congestive heart failure[34] and of education in patients post-MI.[35] In these patients, the effects of psychosocial factors may have been overshadowed by the considerable effects of MI and congestive heart failure on autonomic function.[36] The current study shows that HRV is directly influenced by race and by social class independent of measured clinical, psychological, or behavioral factors.

Limitations

Although the racial composition of this study population mirrored that of New Haven County (and of the United States) with 13% African Americans, the total number of African Americans was small, and few were of higher social class. However, statistically significant, independent associations of race as well as social class with HRV were seen despite the size and uneven distribution of the groups. In addition, the patient population was drawn from patients referred for Holter monitoring. It is possible that patients of minority race or lower social class were referred for Holter monitoring at later stages of underlying disease. Although multivariable analysis showed effects of race on HRV to be independent of measured comorbidities, confounding by unmeasured factors cannot be excluded. However, several factors argue against referral bias as an explanation of the findings. First, the race-specific prevalences of cardiovascular diseases in the study population were similar to those in the population overall.[37] In addition, the majority of each racial group was referred for Holter monitoring by internists, for generally benign symptoms. Larger, population-based studies are needed to confirm these preliminary findings and to further evaluate potential race-class interactions.

Conclusion

In this preliminary study, HRV was lower in African Americans and individuals of lower social class, independent of the effects of clinical, psychological, or behavioral factors. It is possible that the chronic societal

stresses of minority race and lower social class may contribute to disparities in cardiovascular outcomes through autonomic dysregulation.

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